

Bovine Medicine

Diseases and Husbandry of Cattle

Second edition

Edited by

A.H. Andrews

with

R.W. Blowey

H. Boyd

R.G. Eddy

Blackwell

Science

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Preface

It is now about ten years since the first edition of *Bovine* vertent omissions will be rectified in any future reprint

Medicine was published. While it was originally antici-or edition of this work.

pated that it would be used mainly in Britain and

I hope that everyone reading the new edition will find

Europe, it is pleasing to note that a good proportion of

it to be an interesting source of information.

the sales have been in other parts of the world. In

recognition of this, more emphasis has been placed on

Readers' note

conditions and their treatment in areas other than

temperate regions. Additionally, a new section gives an

Inevitably, when it comes to therapies, medicines and

insight to the differences in bovine medicine as prac-

vaccines, each country has different needs and require-

tised in various parts of the world.

ments. The laws concerning the use of particular thera-

Almost all parts of the book have been updated or

peutic and preventative agents may vary and the reader

completely rewritten. There are some new chapters, is reminded that it is his or her duty to ensure that any including one which integrates the various problems preparation prescribed conforms with all relevant which occur in cattle, another on basic surgical techniques and others on artificial insemination and embryo national legislation where the preparation is to be used. It is also essential to ensure that dosages and routes transfer. An effort has been made to encompass all the of administration are determined according to any main subjects which occur in the husbandry and dis-national or local directions and other product information of cattle.

tion which has been provided with the medicine. While I wish to thank all the authors and co-editors for their every effort has been made to ensure that the uses sugar hard work. Roger Blowey, Hugh Boyd and Roger Eddy gested and doses recommended are correct they should have provided advice and assistance despite their many always be checked with currently available information. commitments. I would also like to thank John Sproat

It must also be remembered that any meat withdrawal for supplying the photographs of cattle used on the end time or milk withholding time for drugs should follow the papers.

guidelines of the country in which a drug is used.

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A.H. Andrews

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Preface to the First Edition

Bovine Medicine aims to provide, within the covers of would be pleased to receive comments from readers on

one book, much of the practical information available any deficiencies or difficulties encountered in presentation cattle disease and production. Such an objective is tion or content.

admirable in sentiment but very difficult to achieve in

It has taken approximately 2 years to complete a practice. It involves the concentration of effort by a work of this magnitude. The continual expansion in large number of different, and often very busy, experts

veterinary knowledge and expertise may well mean that into one volume. For the present part it is hoped that in certain areas some recent developments have been what we have produced will not only be a source of omitted. Again, we would be pleased for any such deficiencies to be pointed out to us.

educational read. It is hoped that it will be used as a

There has been considerable recent interest in alternative working guide rather than a reference book and that it native medicine for animals. Mindful of this, a section will be of particular help to those at the 'sharp end' of is included on the subject to help readers make up their the veterinary profession, i.e. in practice. Bearing this own minds on its relevance to cattle therapy.

in mind, this work does not contain every detail con-

I must thank Blackwell Science, and particularly cerning each disease, organism or clinical entity.

Peter Saugman for his patience during the production

Inevitably there are some areas of subject overlap of this book. Much work has also fallen on my coedi-

as might be expected with skin conditions and ecto-
tors Hugh Boyd, Roger Blowey and Roger Eddy.
parasites and 'downer cow', etc. Where possible each
However, the book would not have been completed but
author has provided his or her own perspective on the
for the dedicated secretarial and managerial help of
subject.

Mrs Rosemary Forster.

In addition, references have been kept to a minimum
to ensure a less disjointed read. In consequence, we

A.H. Andrews

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Part 1

MANAGEMENT

Chapter 1

Calf Rearing

D.M. Allen

Introduction

3

calves with signs of lice should be treated with an

Calf reception

3

approved ectoparasite product. A multivitamin injec-

Rearing systems

3

tion containing vitamins A, D and E is good value for

Early weaning: bucket feeding

4

money.

Early weaning: machine feeding

5

The calves should be housed in pens bedded liberally

Follow-on rearing

5

with dry straw and clean, fresh water should be made

Performance targets

5

available. Milk is best withheld for a few hours after

Veal production

5

Calf identification

6

arrival but any calves that appear stressed may be given a warm drink of 1 litre of a proprietary electrolyte solution.

If calves are housed individually, which is permissible

Introduction

up to eight weeks of age, pens should be at least 1.8 × 1.0 metres and should permit visual and physical

Whether calves from the dairy herd are being reared as contact with at least one other calf. Group-housed

dairy herd replacements or for beef production, a good calves up to 150 kg liveweight need a minimum space

start in life is essential. Calves of subnormal weight at allowance of 1.5 m², rising to 2.0 m² at 150–200 kg. Pen

three months of age tend to lag behind throughout the floors should have a slope from back to front of at least growing period.

1 in 20 to permit good drainage.

The starting point for good calf rearing is the con-

Calves do not mind cold weather but need good ventilation without drafts. The modern trend in temperate

bucket feeding within the first six hours of life and a climates is to erect simple monopitch buildings in which further 2 litres within 12 hours. The passive immunity calves can be housed until they are 12 weeks or older. conferred on the calves by the immune lactoglobulin in In the coldest weather straw bales or wooden sheets can colostrum is vital to disease resistance, especially if be placed above the rear half of pens to provide more calves are transferred to another farm for rearing, prob- insulation. Where calves are single-penned, the pens are ably via a collection centre or auction. Mortality among dismantled at weaning and the calves left where they calves deprived of colostrum is high. Therefore, it is are as a rearing group. If buildings and pens are used advisable to keep some colostrum in a freezer in case continuously for calf rearing, after each batch they of emergencies.

should be power washed or steam cleaned, disinfected Colostrum is a rich feed and a good source of the fat and left empty for at least two weeks before restocking. soluble vitamins A, D and E. Therefore it is sensible to

feed all available colostrum even to calves that are beyond the stage when they can gain passive immunity.

Rearing systems

In high yielding dairy herds, calves are weaned off their

Calf reception

dams as soon as possible after they have received colostrum and are reared by the early weaning system

Dairy-bred calves for beef rearing are usually pioneered in the UK but also applicable worldwide.

chased at about two weeks of age, often from a calf

Calves can be weaned off milk replacer after five to group or dealer. When they arrive at the farm, calves seven weeks, which makes the system convenient and should be inspected individually and any showing signs saves money compared to weaning at an older age. The of ill health returned to the supplier or isolated. Navels secret of success is to ensure that individual calves are should be dipped in a concentrated iodine or phenol eating at least 1 kg per day of a palatable early weaning solution to guard against navel ill and joint ill. Any concentrate before they are weaned.

4 • Chapter 1

The commonest rearing system is a twice daily bucket calves are moved at this time. Frequent observation is feed of milk replacer, but some rearers use computer-needed to identify the early symptoms of pneumonia – controlled machines that mix milk replacer and provide listlessness, holding back at feeding time, a runny nose, it to the calves via teats from which they suckle.

rapid breathing or coughing. Veterinary advice should

In the EU, where milk production is subject to milk be sought straight away. Not only is pneumonia a major quotas, milk produced over the quota has no market cause of calf mortality but also infected calves that value so it is sensible to feed it to calves rather than pur-recover often fail to thrive due to lung damage.

chase a milk replacer. However, if milk production is at Dehorning and castration are stressful and so should or under quota, a milk replacer should be used. Feeding not be done together, nor should they coincide with the whole milk needs just the same attention to tempera-

stress of weaning. Healthy calves can be dehorned three weeks into the rearing period using a hot air or hot iron disbudder, with castration at four weeks.

The high cost of dried skimmed milk on which milk replacers used to be based has led to the development of so-called 'zero' milk replacers. These are based on

Early weaning: bucket feeding

dried whey supplemented with fats and lactose to equate as closely as possible to cow's milk. Whey-based replacers are up to 25 per cent cheaper than skimmed

The commonest early weaning rearing method is twice daily bucket feeding with weaning at five to seven

replacers are up to 25 per cent cheaper than skimmed daily bucket feeding with weaning at five to seven milk products but can give equally good results.

weeks. With milk replacer mixed at 125 g/l, the first full

For bucket feeding, replacers are mixed at 125–

feed for a purchased calf is 1 litre. Home-bred calves

150 g/l, according to the manufacturer's recommenda-

are fed this level twice daily until they are about five

tion. The mix is made up with water at 45–50°C so that

days old, then the feeding level per feed is increased by

the temperature at feeding is 42°C. Automatic feeding machines mix replacer at 100–125 g/l but can be set per feed, that is 4 litres per day.

to vary reconstitution rates according to the stage of rearing. Some expert calf rearers have found a useful saving. It is essential to keep all feeding equipment in labour, without detriment to calf performance, by scrupulously clean.

feeding the calves milk replacer once daily after the first seven to ten days. In this case the feeding level is built up gradually to 3 litres per day. However, the saving in labour should not compromise calf inspection, which target in a well-managed calf rearing unit should be to keep mortality below 3 per cent.

From the start calves are fed an early weaning concentrate. Scouring is most likely in the first two weeks follow-

concentrate containing 18 per cent crude protein and with
ing removal from the dam and may be simply because
a good amino acid profile, since the calf needs some of
the calf has been overfed or the milk replacer is at the
its protein intake to escape fermentation and degrada-
wrong temperature. However, it may also be caused by
tion in the forestomachs. The energy value should be at
pathogenic organisms. *E. coli* is the classic cause but a least 12.5 MJ ME/kg
DM.

survey of calf units by practising veterinarians identi-
Even the youngest calves crave some roughage in
fied a number of other pathogens responsible for scour-
their diet and, rather than letting them pick up straw
ing. The immediate reaction to scouring is to take the
from the bedding, it is better to feed hay or straw from
calf off milk and feed a warm electrolyte solution. If the
a rack. Hay is rarely of good enough quality for calves
scouring does not start to clear up within 24 hours vet-
and, even if it is, they eat too much of it at the expense
erinary advice should be sought. Antibiotics should
of concentrates and become pot-bellied. Instead, feed

only be used on veterinary prescription.

bright, dry barley or oat straw.

If salmonella infection is suspected, veterinary advice

As previously mentioned, calves must be eating at

should be sought straight away and rectal swabs taken

least 1 kg concentrate per day before they can be

for laboratory analysis so that the appropriate anti-

weaned. Weaning may be abrupt after about 35 days, or

biotic can be prescribed. Infected calves should be iso-

a gradual weaning procedure may be used in which the

lated. Many *Salmonella* species are transmissible to

level of milk replacer is reduced gradually over an addi-

humans so during an outbreak special attention needs

tional 5–10 days to encourage concentrate consump-

to be paid to personal hygiene. Salmonellosis usually

tion. Gradual weaning avoids the check to growth that

occurs in the first two or three weeks of life but can

accompanies abrupt weaning.

occur later, even beyond the milk feeding period

The early weaning concentrate should be fed *ad*

(Chapter 15).

libitum until it is replaced by a cheaper follow-on

Pneumonia can occur at any time but is most prevalent in still, damp winter weather and is exacerbated if introduced (p. 5).

Calf Rearing • 5

The consumption of milk powder is 15–20 kg and the consumption of milk replacer powder is 25–30 kg target daily gain to weaning is 0.5–0.6 kg/day, depending on breed type and sex.

depending on breed type and sex.

Early weaning: machine feeding

Follow-on rearing

Feeding milk *ad libitum* to group-housed calves can be the preferred choice where the buildings used for calf

When calves are weaned at five to seven weeks the early rearing do not lend themselves to the erection of individual pens or, as in dairy herds, where calves are born *libitum*. In the case of calves going into intensive beef over a long period.

systems, concentrate feeding continues to appetite

The saving in labour is not as great as might be through to 12 weeks, although the finishing diet is introduced because, although mixing is automatic and reduced gradually from eight to ten weeks.

cleaning is quicker, handling the calves is more time

Where calves are designated for forage-based beef consuming, especially teaching them to suckle.

systems, forage is introduced by the tenth week and the

The most sophisticated machines are computer controlled and recognize individual calves fitted with an early weaning concentrate is replaced by a cheaper mix.

When calves reared through the winter are to be grazed

electronic tag. So mixing rates and feeding levels can be varied from calf to calf, including a gradual weaning

0.6–0.8 kg/day until turnout in the spring or the ability

procedure. Some machines can feed whole milk as well as milk replacer and even dispense a small quantity of growth on high quality grazed grass will be inhibited.

concentrates to encourage calves to eat dry feed immediately after suckling. Of course these machines are rationed at about 21/2 kg/day with forage fed to appetite. expensive but the cost is spread over up to 80 calves that can be reared at a time, so the annual depreciation per calf may be reasonable.

Performance targets

A cheaper approach to *ad libitum* feeding held sway for a time but is little used now. This was to feed Performance targets for early weaned bull calves fed cold acidified milk which stays fresh for two to three concentrates *ad libitum* to three months are shown in days stored in a simple plastic bin and is led to a teat Table 1.1.

through a tube fitted with a non-return valve. The equipment is cheap but lacks the sophisticated control of individual calves achieved by computer-controlled

Veal production

feeders.

Calves are trained to suckle about 1 litre of milk

Veal production is a specialized system of calf rearing replacer and then the milk supply is removed until the designed to produce a white meat that is especially next feed. The procedure is repeated twice in the next popular in Italy and Germany. Traditionally, calves were 24 hours and then calves are allowed to suckle *ad libitum*, with not more than six calves per teat. Intake ter at 14–16 weeks, producing a carcass of 100–110 kg.

may be depressed in the coldest winter weather and Subsequently the feeding period was extended to 22 then it is advisable to use an immersion heater to take weeks or more to produce a carcass of 160+ kg.

the chill off the milk replacer. The feeding equipment Consumer revulsion at the unnatural production should be cleaned thoroughly between mixes.

method has brought about considerable changes in the A trough containing early weaning concentrates way veal is produced, sanctioned by EU and national should be placed near the teats, but far enough away to legislation. For example, veal crates have been banned

avoid spoilage by saliva or spilt milk.

in Britain since 1990, although EU legislation does not

Calves fed *ad libitum* consume more milk replacer

ban existing crated housing in continental Europe until

than restricted bucket-fed calves. This manifests itself

2006. Similarly, legislation stipulates a minimum iron

as rather loose faeces that must be differentiated from

content for calf milks and insists that calves over two

scouring. The high replacer intake inhibits early con-

weeks of age have access to digestible solid food. The

centrate consumption and may delay weaning. There-

effect has been to promote the production of veal that

fore, it is important to employ a gradual weaning

is pink in colour rather than white.

programme to allow weaning at five to seven weeks.

A welfare-friendly veal production system has been

Even at these high intakes of milk replacer, it is essen-

demonstrated experimentally in Britain with group-

tial that fresh water should always be available.

housed calves fed milk replacer from a machine and

Table 1.1

Calf rearing targets to three months of age for bull calves fed concentrates *ad libitum*. Source: Meat and Livestock Commission (MLC).

Bucket feeding

Machine feeding

Holstein–Friesian

Charolais ♀ F

Holstein–Friesian

Charolais ♀ F

Hereford ♀ F

Simmental ♀ F

Hereford ♀ F

Simmental ♀ F

Feeds (kg)

Milk powder

15–20

15–20

25–30

25–30

Concentrates

170

185

160

180

Liveweight (kg)

Purchase

50

55

50

55

Weaning

68

76

71

80

3 months

110

120

115

130

Daily gain (kg)

Preweaning

0.5

0.6

0.6

0.7

Post weaning

0.9

1.0

0.9

1.0

F = Holstein–Friesian.

supplemented with barley straw as a source of roughage.

that being able to trace the whereabouts of cattle

Carcasses were acceptable to the veal trade but vari-

throughout their lives is an essential requirement of

ability of performance was an unsolved problem.

food safety for people and disease control in cattle.

In the Netherlands, the main EU veal producer, there

Traceability is subject to EU legislation. The British

are now two approaches to veal production. First there

Cattle Tracing System (CTS) is administered by the

is a white veal system in which group-housed calves are

British Cattle Movement Service (BCMS) that commenced operations in September 1998. This fully computerized system was preceded by a paper passport system that started in July 1996. A one-off survey was undertaken of all cattle in 2000, including older cows employed by some producers in which calves are fed and bulls which did not have passports, so that all cattle could be included in the BCMS database.

Calves must be double tagged within 36 hours of birth unless they are being sent for immediate slaughter. An application for a passport must normally be made within seven days of tagging but calves can be

Calf identification

moved twice during the first 28 days of life, using the

reverse of the cattle passport application form as a temporary passport. A beneficial consequence of the BSE crisis and subsequent foot-and-mouth disease epidemic is a realization replaced the former alpha numeric identity.

Chapter 2

Suckler Herds

D.M. Allen

Introduction

7

ment. Can the herd be integrated with a sheep flock?

Planning the suckler herd

7

Are there arable crop residues that can be used to

Choice of bull

7

cheapen cow feeding? Are buildings and feeds available

Choice of cow breed type

8

to add value to calves by feeding them beyond weaning?

Rearing replacement heifers

9

The answers to these and other relevant questions

Suckler herd management

10

provide the framework on which a profitable enterprise

Suckled calf management

11

can be built.

Grassland management

12

Targets of performance

13

Particularly important is the choice of season of calving. Most herds calve in the spring or at the start of the rainy season because this minimizes cow feeding costs. The cow is working hardest suckling her calf on

Introduction

low cost, high quality grazed forage. On productive grassland autumn calving is an option, the extra cost of The key indicator of profitability and technical efficiency winter feeding cows suckling calves being offset by

ciency in suckler herds is the calf output produced greater calf weaning weight. Autumn calving usually annually from each cow that is bulled. The most profitable achieves the highest financial gross margin per cow, but spring calving rivals its gross margin per hectare. number of live calves per 100 cows bulled, with low calf mortality. The need to reduce production costs has forced many former autumn-calving herds to change to spring calving. per kg for well-reared calves. This is as true under

dry range conditions as it is on productive temperate grassland. The decision on calving season may be forced by the

availability of housing and labour, regardless of feed availability. Suckler herd management is not the simple matter of housing. Housing allows a choice of calving season it may seem at first sight, with a cow suckling a single calf and avoids poaching of land by outwintered stock. The linked components are reproductive efficiency,

However, the provision of housing increases fixed costs milk production and growth. However, in practice the on the farm.

body condition of cows is a simple and sensitive barometer of their nutritional status and potential performance. Controlling body condition through the year is

Choice of bull

the key to high herd output at low cost.

Most beef suckler cows are kept on marginal land in

The bull contributes half the genes of all the calves sired

upland or range areas where winter (or dry season)

by him and so choice of breed and individual bull are

feeds are scarce. So it is common for calves to be sold

both critical to herd performance. Even at weaning,

at weaning, or after a period of further feeding, to fin-

when maternal effects are expressed at their maximum,

ishers on better land. However, increasingly in UK

sire breed has a greater effect on weaning weight than

upland herds where suitable buildings are available,

dam type.

male calves are finished as bull beef on purchased

Heavy breeds such as Charolais and Simmental are concentrates.

generally used as terminal sires, that is to produce the slaughter generation. They sire calves with the highest weaning weights (Table 2.1) and the rapid gains are

Planning the suckler herd

carried through into the post-weaning period. However, the cost of this extra growth performance is a higher

Fitting a suckler enterprise into farm resources is as proportion of assisted calvings and greater neonatal calf important to profitable production as herd management mortality (Table 2.2). Overall, nevertheless, calves sired

7

8 • Chapter 2

by heavier breeds produce the greatest annual output of also be affected if herd replacements are home bred. weaning weight per cow.

Then easy calving breeds with good maternal abilities It is several years since these survey data were collected would be preferred (see p. 9).

lected and there may have been subsequent changes in

The selection of an individual bull within a breed is the relative performance of breeds. Also, breeds such as just as important as breed choice. In recent years the double-muscled Belgian Blue are now available to bull selection has been transformed by the development of a sophisticated statistical method of analysis by commercial producers in the UK. This breed is just below the Charolais in growth performance but has better breeding records from pedigree herds, known by greater dystokia. Belgian Blue crosses have exceptional carcass characteristics with high killing out and meat yield percentages. The analysis of records from all related cattle, whatever herd they are in, effectively disentangles management and genetic effects on performance to calculate estimated breeding values (EBVs) that can be used with confidence to select bulls of above average genetic

lais, Limousin and Simmental. However, some producers prefer local breeds or use Angus bulls to gain quality merit.

In Britain the recording agency Signet combines

premiums that offset poorer growth performance. Easy

EBVs for selected performance characters into selec-

care Angus and Herefords are often selected in pastoral

indices of overall genetic merit. There are two such

and range countries where large numbers of cattle are

indices. The calving value is used where ease of calving

managed by a single stockworker. Breed choice would

is paramount, for example for heifer matings and in the

selection of bulls to breed female replacements. The

beef index is used for bull selection when growth rate

Table 2.1

Effects of sire breed on calf 200-day weights.

and carcass quality are the objectives, for example for

Source: Meat and Livestock Commission (MLC).

terminal sires in suckler herds. Signet publishes EBVs

and selection indices for participating breeds. A

Sire breed

Type of farm

maternal index is under development.

Lowland

Upland

Hill

Choice of cow breed type

Hereford

208

194

184

200-day weight (kg)

Difference from

In range countries the tradition was to keep purebred

Hereford (+ /- kg)

herds of Angus, Hereford, Shorthorn or local breed

Charolais

+32

+33

+21

cattle. To some extent that tradition still exists. For

Simmental

+24

+28

+14

example, French suckler cows are still largely purebred.

South Devon

+23

+27

+16

However, crossbred cows have a considerable advan-

(North) Devon

+17

+21

+7

tage over purebreds due to hybrid vigour (or heterosis),

Lincoln Red

+14

+20

+5

which is most pronounced for improved reproductive

Sussex

+7

+13

+2

efficiency. The outcome is that the weaning weight of

Limousin

+7

+10

+2

calves from crossbred cows is 15–25 per cent above

Aberdeen Angus

-14

-12

-8

the average of the parent breeds. Add to this an

Table 2.2

Effects of sire breed on calving ease and annual productivity. Source: Meat and Livestock Commission (MLC).

Sire breed

Assisted

Calf mortality

Calving interval

Calf weaning weight

calvings (%)

(%)

(days)

per cow per year (kg)

Charolais

9.0

4.8

374

208

Simmental

8.9

4.2

374

203

South Devon

8.7

4.0

375

203

(North) Devon

6.4

2.6

373

200

Limousin

7.4

3.8

375

199

Lincoln Red

6.7

2.0

373

198

Sussex

4.5

1.5

372

196

Hereford

4.0

1.6

372

189

Aberdeen Angus

2.4

1.3

370

179

Suckler Herds • 9

improvement in longevity and the lifetime advantage to score and gestation length) and a high EBV for 200-day crossbreds is considerable.

milk (an estimate of the genetic merit for milk produc-

Recognition of the benefits of crossbreeding in

tion that the sire passes to his female offspring).

Britain is as old as the development of breeds them-

In some parts of the world, notably the US, sophisti-

selves. Traditional crossbreds include the celebrated

cated breeding programmes have been used to create

Blue Grey (White Shorthorn bull ¥ Galloway cow),

composite breeds combining the best features of three

Shorthorn ¥ Highland and Irish-bred Blue Grey

or four foundation breeds. A composite may be bred to (Angus × Dairy Shorthorn). As supplies of these traditional feature maternal qualities, terminal sire qualities, heat tolerance and so on. A four-breed composite is effective, beef breed × dairy cows started to be used as a purpose-bred pure breeding population that suckler cows, notably Hereford × Friesian and Angus × Friesian. It was later still that US ranchers discovered initial first (F1) crosses between the original four breeds. The heterosis advantage of Hereford × Angus and the reciprocal cross over either of the pure breeds.

The creation of composite breeds needs very large numbers of cattle. However, commercial suckled calf as a suckler cow. It produces more milk than the Blue Grey and weans a heavier calf; however, the Blue Grey to composites from international genetics companies

The Hereford × Friesian was, and still is, widely used. However, commercial suckled calf as a suckler cow. It produces more milk than the Blue Grey and weans a heavier calf; however, the Blue Grey to composites from international genetics companies

has better reproductive efficiency and, overall, per-
with large-scale breeding operations involving thou-
formance of the two crosses is similar. Both crosses
sands of cattle. The main demand will be for purpose-
share the advantage of medium body size. Heavy cows
bred suckler herd replacements of good maternal
such as Charolais × Friesian need more feed but are
ability.

unable to translate enough of their greater weight into
heavier calf weaning weight to rival the efficiency of a
lighter breed type. Nonetheless, many British suckled

Rearing replacement heifers

calf producers use these heavier cows and are prepared
to trade a theoretical reduction in efficiency (that may
The replacement policy in a suckler herd should involve
not be apparent at farm level) for the improved con-
culling cows that are persistently barren, calve unac-
formation of calves which commands a premium at the
ceptably late or are on the verge of the emaciation com-
calf sales and later in carcass value.

monly associated with old age. A typical replacement

The penetration of the Friesian breed by extreme rate is 16 per cent which indicates an average herd life dairy Holstein genes from North America has caused of seven years.

such a deterioration in beef breed × Friesian cow

In the UK, culling policies have been disrupted in conformation that it shows through in the calf. This the wake of the BSE crisis by the Over Thirty Month has sparked off a search for alternative suckler herd Scheme (OTMS) in which the carcasses of cattle over replacements that are crossbred with good maternal 30 months old are removed from human consumption. qualities and have acceptable conformation. Moreover, Compensation payments for culls are much lower than in the wake of the BSE crisis many herd owners wish cull cow values before the BSE crisis and this will to breed their own replacements so that they can continue to inhibit planned culling until the scheme is maintain a closed cow herd.

eventually wound down.

Merely saving heifer calves from terminal sire breeds

Replacements may be purchased as calves from dairy such as Charolais increases cow size progressively and herds, as bulling heifers or, less frequently nowadays, as reduces maternal performance – the worst of all worlds. heifers on the point of calving. With calves and bulling The simplest planned approach to breeding replace-heifers it is prudent to purchase a surplus of 15–20 per cents is to breed about half the herd to a sire breed of cent to allow selection. Heifers that prove unsuitable as medium size with good maternal qualities, for example replacements are slaughtered for beef.

Angus or Salers. In smaller herds this is best done by In most situations heifers should be calved for the artificial insemination (AI). In the next generation, first time at two years of age to optimise lifetime performance. However, autumn-born calves from the dairy Angus cross females, for example, might be mated to Salers whose female progeny are in turn mated back to herd would usually be calved at 21/2 years in a spring-Angus. This two-breed rotation is known as criss-calving suckler herd. In either case, good management

crossing. The other half of the herd would usually be is necessary to achieve target mating and post calving mated to terminal sire breeds.

weights (Table 2.3).

Bulls for breeding herd replacements should be

Heifers are best calved at the start of the herd calving selected to have a good calving value index (which period to allow for an almost inevitable slippage in time incorporates EBVs for calf birth weight, ease of calving to the second calving. Also, since dystokia is worst in

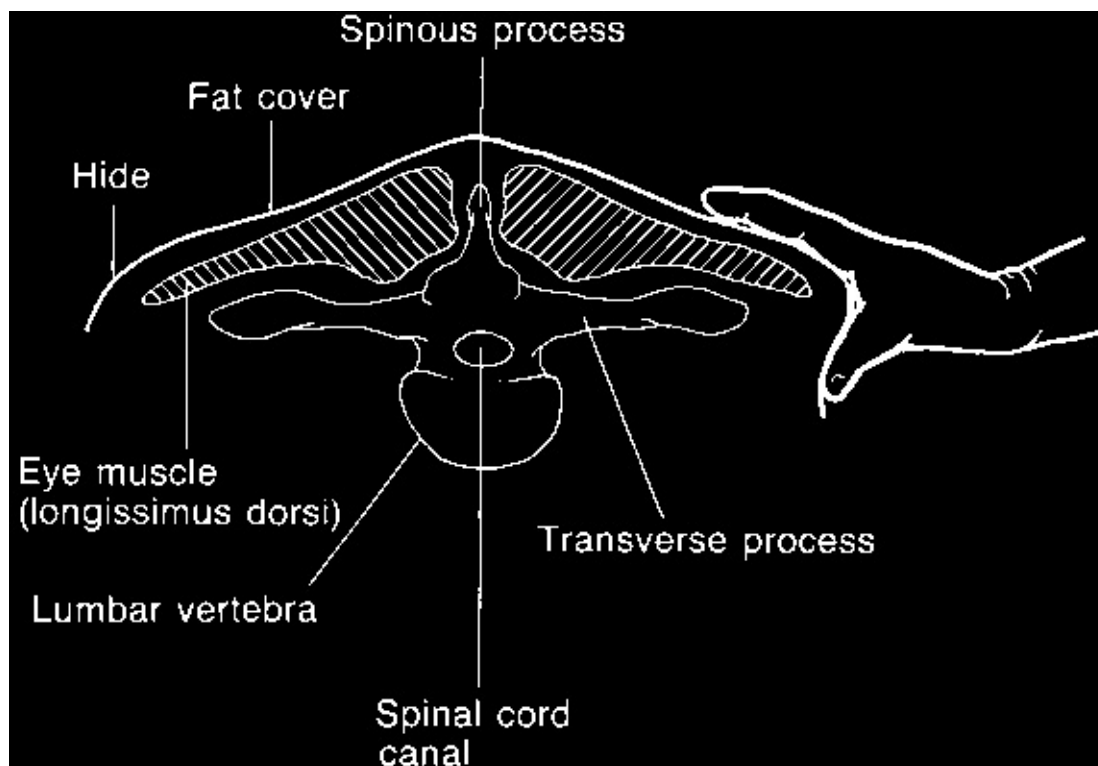


Table 2.3

Rearing targets for replacement heifers.

Mating Post

calving

weight (kg)

weight (kg)

Two-year calving

British breed crosses

325

510

Continental breed crosses

350

550

Calving at 21/2 years

British breed crosses

400

540

Continental breed crosses

435

575

Fig. 2.1

Technique for condition scoring.

heifers, it is desirable to mate them to an easy calving breed such as Angus or Hereford. Difficult calving is a cause of delayed rebreeding.

side of the cow. The thumb curls over the ledge formed by the transverse processes of the spine to feel the overlying fat cover. It is best to handle cows until experi-

Suckler herd management

ence of the technique has been gained but, thereafter, a skilled stockworker can use close visual inspection to Suckler herd management aims for a high proportion obtain a working guide to condition.

of cows producing live calves in a calving period of 12

Descriptions of condition score classes are presented

weeks or less. The advantages of a compact calving

in Table 2.4. One possible confusion is with continental

period are, firstly, that herd rationing matches closely

crosses of good conformation. Their thick muscling may

the nutritional needs of individual cows, saving feed

overhang the transverse processes of the spine and

costs. Secondly, calvings can be supervised closely to

confuse handling or visual assessments. If this is the case, condition should also be assessed by handling the calf. Finally, calf performance is uniform with few of the ribs with the flat of the hand. If the score is above 3 in late-born calves that are so difficult to utilize profitably. Generally, a long drawn out calving period is a sure sign of low herd conception rate. Late calvers have too little time to rebreed by the end of the mating season reduced and feed costs increase.

and get later and later, eventually failing to get in calf Feeding spring-calving cows through the winter that altogether. The only solutions to an over-long calving period are to cull late calvers or switch them between spring- and autumn-calving herds so that they calve on about 100 kg liveweight or 0.5 kg daily. Autumn calvers,

time. The danger of the latter strategy is that cows on the other hand, must be fed through the winter for shuttle from one herd to the other without the under-the additional strain of lactation and must be at condition score 2½ when mated in mid-winter. Therefore, in The main reason why cows fail to conceive is that cows at condition score 3 in the autumn a weight loss they are too thin. Under UK conditions, body condition of only 0.25 kg/day is permissible until they are safely in at mating, scored on a scale from 0 (emaciated) to 5 calf. If there is a time to feed suckler cows generously, (grossly overfat), needs to be 2½ in winter but can be this is it. Thereafter, the rate of weight loss can increase as low as 2 if cows are grazing high quality forage and to 0.5 kg/day. Autumn-calved heifers are still growing as current nutrient intake is high. The key targets that well as milking and should not be allowed to lose weight achieve the necessary body condition at mating are a through the winter. score of 3 in the autumn and 2 at turnout to grazing in

These guidelines are translated into daily metabolizable energy (ME) allowances expressed as megajoules (MJ) for cows fed a typical moderate quality ration in management before calving.

Table 2.5.

The method of condition scoring (Fig. 2.1) is to grip the loin between the thumb and forefinger mid-way between the hip (hook) bone and the last rib on the left side. It is all too easy during the grazing season to concentrate on calf performance and forget that cows at target condition score 2 in the spring need to gain

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Table 2.4

Condition scoring of suckler cows.

Condition score

Description

0

Spine very prominent with no detectable fat cover over the sharp transverse processes of the spine 1

Spine still prominent but transverse processes no longer sharp 2

Transverse processes can still be felt, but now rounded with a thin covering of fat 3

Individual transverse processes can now only be felt with firm pressure by the thumb 4

Transverse processes can hardly be felt, even with firm pressure 5

Transverse processes completely obscured with a thick layer of soft fat and puffy fat deposits around the tail head

Table 2.5

Metabolizable energy (ME) allowances for winter

after itself. Nevertheless, a compact calving means

feeding suckler cows. Reproduced from Allen (2001) with per-

fewer of the late-born calves have subnormal weaning

mission of Chalcombe Publications.

weights. In addition, achieving target body condition

scores in cows is conducive to high milk yield.

Liveweight

Daily weight

Milk yield

ME

In most situations the cow is capable of producing as

(kg)

loss (kg)

(kg)

(MJ/ day)

much milk as the calf can suckle. This can lead to problems early in the suckling period, especially with spring-

Spring calver

calving beef & dairy cows of high milk potential, when

(a) Precalvinga

500

-0.5

0

55

calves consume too much milk and scour. If the cause

600

62

of scouring is viral or bacterial it is worth considering

700

69

an appropriate scour vaccine.

(b) Post calvinga

Milk consumption increases quickly during the first

500

-0.5

10

89

month of suckling and, although calves pick at solid

600

97

food early in life, milk intake dominates calf perform-

700

105

ance in the first three months. With a cow type of mod-

Autumn calver

erate yield potential, milk still accounts for half the calf

(a) Prematingb

gain in the third month and in higher yielding beef

500

-0.25

10

99

breed ¥ Friesians this balance is not reached until the

600

107

fourth month. Nevertheless, by this time the intake of

700

115

(b) Post matingb

solid food by the calf is increasing rapidly.

500

-0.5

7

74

Milk intake is especially important to the spring-born

600

81

calf that has a relatively short suckling period of six to

700

88

seven months. The nutritional requirements of the calf

and seasonal grass growth are well matched, although

a If no weight loss is permissible add 20 MJ ME per day.

supplementary feeding helps to sustain daily gain from

b If no weight loss is permissible add 10 MJ ME per day.

late summer onwards as grass growth and quality decline.

100 kg liveweight by the autumn. If by mid-season there

Management of the autumn-born calf is more com-

is any doubt that the whole herd or individual cows may

plicated because the suckling period is longer and peak

not attain the required total gain, action must be taken

milk yield must be supported on winter rations. The

to rectify the situation. The provision of more selective

most cost-effective approach over the winter is to feed

grazing of fresh pasture may suffice. Alternatively, it

the cow well in the early months until she is safely in

may be necessary to take more drastic action with

calf and then to rely increasingly on creep feeding

autumn calvers and wean calves early to remove the

calves concentrates and the highest quality conserved

strain of lactation.

forage available. It is energetically much more efficient

to feed the calf directly than to increase cow feeding to

stimulate milk yield.

Suckled calf management

When autumn-calved cows are turned out to grass in the spring there is a boost to milk yield, but by late If cow performance is good, calf performance is usually summer calves require creep feeding to sustain daily also good. This is not to say that calf performance looks gains. It is wise to wean the calves sooner rather than

12 • Chapter 2

Table 2.6

Performance targets for lowland and upland suckler herds.

Continental breed sirea

British breed sirea

Autumn

Spring (silage)

Spring (straw)

Autumn

Spring (silage)

Spring (straw)

Calves reared/cow

0.92

0.92

0.92

0.95

0.95

0.95

Calf gain (kg/day)

1.0

1.1

1.1

0.9

1.0

1.0

Weaning (months)

10

7

7

10

7

7

Calf autumn weight (kg)

350

280

280

320

260

260

Cow concentrate (tonnes)

0.25

0.12

0.3

0.25

0.12

0.3

Calf concentrate (tonnes)

0.25

0.08

0.08

0.1

0.05

0.05

Silage (tonnes @ 22% DM)

6.5

5.0

—

6.5

5.0

—

Feeding straw (tonnes)

0.5

1.0

2.5

0.5

1.0

2.5

a Mated to suckler cows of average weight 550–600 kg.

later so that they can be managed separately and the

Grassland management for suckler herds does not

cows left to gain condition before housing. The least

necessarily mean using the levels of nitrogen fertilizer

stressful way of breaking the bond between calf and

employed by dairy farmers, although this may be the

dam at weaning is to house the calves. An alterna-

right policy on some productive lowland farms. In prac-

tive approach is to put the cows and calves in well-

tice, especially on the upland farms where most suckler

fenced adjacent fields within sight and sound of each other. cows are kept in the UK, farmers use only moderate other.

levels of nitrogen fertilizer and rely more on clover to In most ranching and pastoral countries bull calves increase sward productivity. Moreover, extensive are castrated for the convenience of herd management grassland management is encouraged in the EU by and a preference for steer beef over bull beef. However, the payment of extensification premiums for stocking in France, Italy and Spain it has become the custom to lightly.

rear male calves entire because they grow faster and Whatever the type of sward, grazing management leaner than steers and are more profitable. In the UK, should aim to control the average sward height of suckler bull beef is increasing and finds a ready market. grazed and ungrazed areas. Cows gain most weight early The most common approach is to wean spring-born in the grazing season when average sward height is bulls in late summer and transfer them gradually over

maintained at 8–10 cm. However, they graze selectively a period of about three weeks to an all-concentrate diet leaving patches that go to seed and are of poor nutritional quality when they have to be grazed later on. So (Chapter 3).

For the system to be successful it is a considerable until mid season it is necessary to graze swards more advantage to have a compact calving period so that tightly at an average sward height of 6–8 cm to inhibit older bull calves do not pester late-calving cows when seed heading. Thereafter, when the risk of seed heading they are in oestrus. In any case, cows with bull calves is less and there is an inevitable build-up of dead need to be separated from those with heifers by six herbage in the base of the sward, average sward height months of age or there is a risk of premature pregnancy can be relaxed to 8–10 cm to allow selective grazing. in the heifer calves. For this reason the system works In the period of peak grass growth in early summer, best in spring-calving herds because there is no need to high stocking rates of three autumn calving cows per

split the herd.

hectare or four spring calvers are needed to exert the necessary control of sward height. At these relatively high stocking rates it is important to have a reserve

Grassland management

grazing buffer in case of reduced grass growth in dry weather. This can be achieved by setting aside an additional 25 per cent of grassland that is conserved as silage. Grazed grass is the cheapest and one of the most nutritious feeds available on the farm. With financial margins under pressure, the key to profitable production is to utilize grazing to the full. This involves good grazing management and extending the grazing season if possible, but grazed if necessary.

As the season progresses beyond mid season the grazing area needs to increase and this is achieved by grazing aftermaths on fields cut previously for silage or where practicable.

hay.

Suckler Herds • 13

Parasitic worms causing gastroenteritis and bronchi-

targets appropriate to lowland and upland herds in the
tis are less of a problem in suckled calves than in young
UK and other European countries with similar climatic
dairy-bred calves in their first grazing season. Never-
conditions (Table 2.6). For the predominant spring-
theless, it is advisable to treat autumn-born calves at
calving herds targets are shown for herds both on grass
weaning. In any case, both autumn- and spring-born
farms, where silage is the basic winter feed, and on
calves should be treated at yarding to guard against
arable farms, where winter diets are based on straw
winter scour (Chapter 19).

and concentrates. Achieving the physical targets is a
With silage making the priority is a feed with high
prerequisite of profitable production.

intake characteristics rather than the highest possible
ME per kg dry matter (DM). Cutting can be delayed
for a week or so to await dry sunny weather that allows

Reference

an effective 24-hour wilt for clamp silage, longer for big
bales, and produces silage with the best fermentation

Allen, D. (2001) *Rationing Beef Cattle*, 2nd edn. Chalcombe quality.
Publications, Lincoln.

Targets of performance

The objectives of suckler herd management outlined in
this chapter have been compiled into suckler herd

Chapter 3

Beef Finishing Systems

D.M. Allen

Introduction

14

ishers in the autumn may be finished through the

Planning and budgeting

14

ensuing winter for sale at 16–18 months old. Heifers

Finishing suckled calves and stores

15

and lighter steer calves are fed a store ration over the

Winter finishing

15

winter in preparation for grazing the following summer.

Grass finishing

17

Any that fail to finish off grass are yarded for a further

Dairy beef systems

18

winter finishing period.

Cereal (barley) beef

19

Still, all too many cattle are managed aimlessly with

Grass and maize silage beef

19

Eighteen-month beef

20

no particular finishing system in mind and are sold

Grass beef

20

finished or in store condition when prices seem

favourable. This approach leads to a good deal of

trading in suckled calves and stores, some of which may

change ownership three or four times in their lifetime.

Introduction

Farm assurance schemes now limit the number of times

an animal can be traded within the scheme.

A whole range of overlapping beef finishing systems is Under range conditions in the USA, the growth rate applicable to cattle bred in suckler and dairy herds. The of suckled calves is poorer than on European grass higher the lifetime daily gain of the beef system, the farms. However, feed grains are cheap and a special younger is the slaughter age and the lighter the slaught- approach to finishing has been adopted in beef feed ter weight at a stated carcass fat cover. Also, within a lots. Weaned calves from range herds are sold to 'back given beef system, bulls grow faster and are leaner than grounding' farms where they are grown on forage at steers which, in turn, grow faster and are leaner than moderate store rates of gain as feeder cattle. They then heifers.

enter feed lots at 12–18 months of age for rapid finish- These characteristics of beef systems make it possi- ing over a five-month period on a 'hot' high-grain ration. ble to chart average relationships between slaughter In the case of dairy-bred calves, several distinctive

age and slaughter weight, illustrated in Fig. 3.1 for beef systems have been developed. At one extreme Charolais crosses. It is possible to construct similar is cereal (barley) beef in which bulls fed an all- graphs for other breed types and to predict the likely concentrate diet grow rapidly to slaughter at 11–13 slaughter weight of cattle slaughtered at a particular months of age. At the other extreme are forage-based age and vice versa. Note that as long as the Over Thirty systems in which steers and heifers are either winter finished off silage supplemented with concentrates for measure in the UK, beef from cattle over 30 months old slaughter at 16–20 months of age or grass finished in at slaughter is destroyed, although this will change from summer at 20–24 months old.

2004 onwards.

In reality, cattle passport statistics indicate that more Cattle remain in an EU fat class for several weeks. than a third of steers do not follow any of the production The main EU fat class for bull carcasses is 3 on a

tion systems described here and are slaughtered at five-point scale from 1 (ultra lean) to 5 (grossly overfat) 24–30 months of age. The reason is thought to be that and 4L for steers and heifers, which is the average fat farmers delay slaughter until after they have obtained class for all carcasses in Britain.

the second beef subsidy (p. 15), paid from 22 months of Calves from beef suckler herds on marginal land age. It demonstrates how subsidy rules manipulate production and marketing decisions.

farms with more productive land. However, spring-born calves may be overwintered for sale in spring, if buildings and feed are available, and bull calves may

Planning and budgeting

be finished on purchased concentrates as suckler bull beef.

The fall in market prices for beef cattle in the aftermath of the BSE crisis has focused attention on beef system

700

If production is based on borrowed capital, it is important to calculate the interest on working capital

650

that must be paid. The conventional method of calcu-

600

lation is the cost of the calf or store plus half the vari-

550

able costs multiplied by the monthly bank interest rate for the number of months cattle are fed.

500

Sale returns are highly dependent on the quality of

450

cattle produced, especially their conformation. Figure 3.3 illustrates differentials for steer carcasses classified

Slaughter liveweight (kg)

400

by the EU method, in a conformation range from E

350

(excellent), U+, -U, R (average), O+, -O and P (very

12

14

16

18

20

22

24

28

poor). Note the severe discount for poor -O confor-

Slaughter age (months)

mation, especially for carcasses that stray into what is

Fig. 3.1

Average relationships between slaughter age and

regarded by most buyers as overfat class 4H. Hol-

slaughter weight for Charolais cross cattle. (Bulls were slaugh-stein–Friesian carcasses fall into the poorest conforma-

tered at EU fat class 3, steers and heifers at EU fat class 4L.) tion classes -O and P and a high proportion of the beef

糒 = Bulls; 糒 = steers; 糒 = heifers.

is used for manufacturing. Good conformation classes

-U and better contain mainly continental breed crosses.

plans and budgets. Formerly, too many beef farmers

Generally, young bulls sell for the same price per kg were preoccupied by the margin between buying and carcass or slightly less than steers in deadweight sales, selling prices, or even just the sale price achieved but command a premium at selected live auction regardless of cost. Now controlling costs, whilst achieving performance targets, is very important. Also essential is a steady move towards deadweight selling under which maximizing eligibility for EU subsidies, without pressure from supermarket buying specifications, which profitable production is difficult.

The EU beef regime that came into effect in January

Finishing suckled calves and stores

2000 is more complex than previous schemes. As before, bulls may qualify for a single Bull Premium and At the start of a winter feeding period there are two steers for one or both Beef Special Premiums (BSP), options for steers and heifers. Either the cattle are fed the first of which is paid at 9–20 months of age and the to gain at least 0.9 kg/day, often higher, for finishing

second at over 22 months. There is no upper limit on that winter. Or they are fed a store ration to gain 0.6– the number of claims provided the stocking rate does 0.8 kg/day in preparation for grazing the following not exceed 1.8 Livestock Units (LU) per forage hectare, summer.

otherwise claims are scaled back. If a national ceiling It is generally held that the profitability of finishing of claims is exceeded, payments are also scaled back. depends mainly on buying and selling skills. It is per- Cattle must be retained for at least 2 months after the fectly true that the feeder's margin – the difference claim is made. All slaughter cattle, including cull cows, between the purchase and sale price – determines the qualify for a slaughter premium provided they have average gross margin. However, finishers with the best been on the farm for at least two months. Suckler Cow margins owe much of their success to achieving high Premiums are paid up to a farm quota and a minimum standards of cattle performance.

of 5 per cent and maximum of 40 per cent of claims can

be made on heifers over 8 months of age. Complex rules

Winter finishing

apply to Extensification Premiums paid on farms practising extensive production methods. Detailed rules and

Winter finishing has a feeding period of four to eight rates of payment can be found in official publications. months, depending on breed, sex category and individ-

In 2003 discussions were in progress to alter fundamental performance. At the start of the winter the cattle mentally the basis of subsidy payments with headage may be yearling suckled calves or older store cattle. subsidies replaced by area payments.

Commonly, they are fed rationed concentrates with The starting point for planning is a decision on which silage to appetite, but on arable farms arable byproduct beef system fits farm resources best, setting performance targets and drawing up a budget to evaluate

Steers of all breed types are well suited to winter finishing. Early maturing heifers are less suitable because

that can be used to evaluate finishing systems and they finish too quickly at light weights. Nevertheless, shows as an example cereal bull beef from purebred continental cross heifers can be winter finished at Holstein–Friesian bulls.

around 0.9 kg/day on a high forage diet.

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Budget

Example: cereal beefa

(£/head)

(£/head @ 2003 prices)

A Calf or store cost

40

B Calf rearing to 3 months: dairy-bred calf

58

C Forage costs . . . cattle/ha @ . . . £/hab

0

D Concentrate costs 1.9 tonnes @ £ 100/t 190

E Other feeds: straw 0.3 tonnes @ £ 30/t 9

straights . . . tonnes @ . . . /t

by-products . . . tonnes @ . . . /t

F Other variable costs: veterinary

10

bedding

20

marketing

8

Miscellaneous

10

Total

48

G Total calf + variable costs

(A + B + C + D + E + F) 345

H Sale weight (kg liveweight or carcass)

475 kg

I Forecast sale price (£/kg)

0.8 £/kg

J Returns: weight **H** ¥ sale price **I**

380

K Gross margin (excluding premiums)

35

L Premiums: 1st beef special premium

0

2nd beef special premium

0

bull premium

130

slaughter premium

50

extensification premium

0

Total

180

Overall gross margin (including premiums) **K + L**

215

a Holstein–Friesian bull beef.

b Guidelines on forage costs at 2003 prices:

intensive (350 kg N fertilizer/ha) £210/ha

semi-intensive (250 kg N/ha) £150/ha

extensive (75 kg N or less/ha) £70/ha.

Fig. 3.2

Budget format for beef finishing systems.

Standards for winter finishing suckled calves and

stores are shown in Table 3.1. Continental cross steers

185

slaughtered at two years old or more may be too heavy

180

for some buyers.

175

Suckler bull beef is still relatively new to the UK, but

has been commonplace in France for many years. Pro-

170

duction is expanding in the UK but demand is limited

165

and it is important to secure a market before com-

160

mencing production. The system is simplest for spring-

Carcass p/kg

155

born calves which are weaned in the autumn and

–U

150

R

transferred gradually over about three weeks to an all-

145

concentrate diet containing 16 per cent crude protein.

O+

140

However, autumn-born calves weaned in the spring can

3

–O

4L

also be used. The British safety code for bull beef pro-

4H

Better conformation

duction recommends no more than 20 bulls to a pen but

Fatter

much larger groups are housed in continental Europe.

Maize silage is short of protein but has good feed

Fig. 3.3

Price differentials for steer carcasses of different EU

conformation and fat classes. Source: Meat and Livestock

intake characteristics and is an excellent source of

Commission price report 1999.

energy. In continental Europe maize silage is widely

Table 3.1

Standards for winter finishing.

Yearling suckled calf

18-month store

British

Continental

British

Continental

Steer

Steer

Heifer

Steer

Steer

Heifer

Feeding period (months)

5

6

5

5

5

5

Start weight (kg)

320

350

310

420

475

400

Daily gain (kg)

0.9

1.0

0.8

0.8

0.9

0.8

Silage (tonnes, 22% DM)

3.6

3.6

4

5.7

5.9

5.5

Concentrates (kg)

300

450

220

135

265

200

Slaughter weight (kg)

455

525

430

540

610

525

Carcass (kg)

250

295

235

300

340

Table 3.2

Standards for suckler bull beef using continental crosses.

Spring-born**Autumn-born****Concentrates****Maize silage****Concentrates****Maize silage**

Start age (months)

6

6

6

6

Feeding period (months)

6

7

6

7

Start weight (kg)

245

245

230

230

Daily gain (kg)

1.4

1.3

1.4

1.3

Concentrates (tonnes)

1.4

1.0

1.4

1.0

Maize silage (tonnes)a

—

3.3

—

3.1

Slaughter weight (kg)

500

525

485

510

Carcass (kg)

280

295

270

285

a Maize silage at 30% DM.

used as the forage base for intensive bull beef production. The value of daily gain and carcasses being too heavy for the medium-sized feed lots of the Po valley in buyers.

northern Italy with a few hundred to a few thousand cattle are a good example. In these maize silage com-

Grass finishing

prises 30–50 per cent of ration DM.

Maize silage has not been used much for bull beef

Grazed grass is the cheapest and potentially the highest production in Britain but there is potential for systems quality home grown feed. There is a growing realization in which maize silage forms up to 30 per cent of the dry

of the need to exploit grass better by managing swards matter of the diet, producing daily gains not far short to maximize daily gains and, where practicable, to of all-concentrate diets. Where suckled calf producers extend the grazing season.

on marginal land have suitable buildings available, bull In grass finishing systems, achieving high daily gains beef fed purchased concentrates can be a useful way of is especially important so that marketing begins in mid-adding value to herd output.

season and reduces cattle numbers in step with decline- Standards for suckler bull beef are shown in Table ing grass production and quality.

3.2. Achieving the target daily gains is a prerequisite Two aspects of management are crucial. Firstly, in the of profitable production. In practice many producers winter preceding grass finishing, gains are held down at extend the feeding period and take bulls to slaughter a store level so that cattle exhibit rapid compensatory weights in excess of 600 kg. At these high slaughter growth when they are turned out onto spring grass.

weights there is a danger of feed cost exceeding the
Secondly, grazing should be managed to get the best

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Table 3.3

Specimen winter store rations.

Weight

British cross steer/heifer @ 0.6 kg/day

Continental cross steer @ 0.8 kg/day

(kg)

Silage

Straw + concentrate

Silage + concentrate

Straw + concentrate

(kg/day)

(kg/day)

(kg/day)

(kg/day)

200

18

3.5 + 2.0

16 + 1

$$3.5 + 3.0$$

$$250$$

$$21$$

$$4.0 + 2.5$$

$$19 + 1$$

$$4.5 + 3.0$$

$$300$$

$$23$$

$$4.5 + 3.0$$

$$22 + 1$$

$$4.5 + 3.5$$

$$350$$

$$26$$

$$5.0 + 3.5$$

$$20 + 2$$

$$5.0 + 4.0$$

$$400$$

$$28$$

$$5.0 + 4.0$$

$$23 + 2$$

$$5.0 + 4.5$$

combination of daily gains in cattle and regrowth of

Table 3.4

Standards for overwintering and grass finishing

high quality grass.

suckler-bred steers and heifers.

Grass finishing may commence with stores purchased

in the spring or with suckled calves or stores purchased

Continental

British cross

cross

in the autumn that are overwintered before grass fin-

ishing. Specimen low cost rations for overwintering

Steer

Heifer

Steer

Heifer

cattle are shown in Table 3.3. The lower gain of 0.6 kg/

day is optimum for British cross steers and heifers of all

Winter store period

breed types. The higher gain of 0.8 kg/day is suitable for

Feeding period (months)

6

6

6

6

continental cross steers.

Start weight (kg)

270

235

300

260

During the grazing season on set stocked pastures,

Daily gain (kg)

0.6

0.6

0.8

0.6

the provision of a continuous supply of nutritious grass

Concentrates (kg)

90

90

500

90

is achieved by controlling sward height (the average of

Silage (tonnes)

4.5

4.3

5.2

4.5

grazed and ungrazed grass). In the first half of the

Grass finishing

season sward height is controlled at 6–8 cm to inhibit

Turnout weight (kg)

375

345

445

370

seed heading. Then in the second half of the season,

Feeding period (months)

5

5

6

5

when there is less risk of seed heading and dead

Daily gain (kg)

0.8

0.7

0.9

0.8

herbage begins to accumulate in the base of the sward,

Concentrates (kg)

50

0

150

50

grazing height is relaxed to 8–10 cm to allow more selec-

Slaughter weight (kg)

500

450

600

490

tive grazing.

Carcass (kg)

275

245

335

270

The confidence to manage cattle at the high early summer stocking rates needed to achieve this degree of control is helped by adding 25 per cent to the main BSP is paid from 22 months of age, steers that are close grazing area as a buffer that can be grazed if grass runs to this age at the end of the grazing season are often short but is conserved if not needed. From mid-season yarded for winter finishing just to claim the subsidy. It grazing needs to expand onto aftermath regrowths on is a strategy that needs to be costed carefully.

areas previously cut for silage or hay.

Grazing performance is undermined if cattle, especially those in their first season of independent grazing,

Dairy beef systems

become infected with parasitic worms that cause gastroenteritis and bronchitis. Effective control programmes

In the UK the main dairy herd calving season is in late include early season treatment to inhibit the build-up

summer and autumn, which contrasts with many other of infective larvae on the sward, mid-season treatment countries where cows calve in the spring. It reflects the accompanied by a move to fresh aftermaths on fields high proportion of milk produced used for year-round previously conserved or mid-season treatment with a liquid consumption.

long-acting bolus to control infection even though pas- Intensive indoor dairy beef systems can utilize calves tures carry infective larvae. The choice depends on farm born at any time of year. However, grass-based systems circumstances (Chapter 19).

work best utilizing calves from the main calving season. Standards for an overwintering and grass finishing At turn-out in the spring these calves are old enough to system are presented in Table 3.4. Because the second make effective use of grazed grass.

Beef Finishing Systems • 19

Cereal (barley) beef

Grass and maize silage beef

Cereal beef is a housed bull beef system feeding an

Grass silage bull beef was popular in the 1980s as a beef all-concentrate diet to slaughter at 11–13 months of age. Carcasses have a creamy white fat which is popular which was made into silage. Its flaw was that it was a system that made very intensive use of grass, all of age. Carcasses have a creamy white fat which is popular with some supermarkets and independent butchers. Its flaw was that it was a system designed for its production capabilities rather than market demand. However, it is advisable to secure market outlets before than market demand. In the original system bulls were commencing production, especially if poor conformation. 16–18 months old at slaughter, by which time the carcasses were too heavy for many buyers and the fat was most profitable when it adds value to home-grown grain coloured by carotene. There are also question marks or home-bred calves.

over the eating quality of beef from bulls over 14

Profitable production is heavily dependent on achieving high target daily gains. The protein content of the months of age.

There is still a place for grass silage bull beef up to

diet is reduced progressively from 18 per cent crude protein in the calf ration to 14 per cent in the final finishing ration. The system could also be used for steers and heifers but, for reasons that have been discussed already, grass-based systems now place emphasis on the utilization of grazed grass.

by about 10 per cent, but their continued use in the EU is controversial. The section on winter finishing (see p. 15) has already

discussed the potential for maize silage in beef production. Standards for cereal beef are shown in Table 3.5.

With up to 30 per cent of the ration DM as maize

Table 3.5

Standards for cereal bull beef.

Breed type

Holstein–Friesian

Limousin × Friesian

Belgian Blue × Friesian

Charolais × Friesian/

Simmental × Friesian

Slaughter age (months)

11.5

12.5

12.5

12.5

Daily gain (kg)^a

1.3

1.3

1.35

1.4

Concentrates (tonnes)^a

1.8

1.9

1.9

2.0

Slaughter weight (kg)

460

500

510

530

Carcass (kg)

250

285

290

295

a From 3 months of age.

Table 3.6

Standards for grass silage bull beef.

Breed type

Holstein–

Hereford ¥

Limousin ¥

Belgian Blue ¥

Charolais ¥ Friesian/

Friesian

Friesian

Friesian

Friesian

Simmental ¥ Friesian

Daily gain (kg)a

1.15

1.05

1.3

1.25

1.3

Slaughter age (months)

14

14

14

14

14

Concentrates (tonnes)a

1.1

0.63

0.63

0.94

1.3

Silage (tonnes @ 22% DM)a

5.7

6.8

6.7

6.1

5.4

Cattle/ha

7.9

6.6

6.7

7.4

8.3

Slaughter weight (kg)

495

465

513

535

555

Carcass (kg)

270

255

290

300

305

a From 3 months of age.

20 • Chapter 3

Table 3.7

Standards for maize silage bull beef production.

Breed type

Holstein–

Hereford ¥

Limousin ¥

Belgian Blue ¥

Charolais ¥ **Friesian/**

Friesian

Friesian

Friesian

Friesian

Simmental ¥ **Friesian**

Daily gain (kg)a

1.25

1.2

1.25

1.3

1.3

Slaughter age (months)

13

12.5

13.5

13.5

13.5

Protein concentrate (tonnes)a

0.50

0.47

0.52

0.52

0.52

Cereal (tonnes)a

1.0

0.7

0.7

0.7

0.75

Silage (tonnes @ 30% DM)a

3.8

4.0

4.4

4.4

4.4

Cattle/hectare

8.7

8.3

7.5

7.5

7.5

Slaughter weight (kg)

490

455

510

530

535

Carcass (kg)

265

245

285

300

300

a From 3 months of age.

silage there is little decline in daily gain from cereal production and quality decline. In addition, there is a beef levels. Experience of the system is limited and so strong case for supplementing calves with about 1 kg the bull beef system presented in Table 3.7 is somewhat concentrate daily for two or three weeks after turn-out speculative. It is based on 25 per cent of the ration DM in the spring until they become accustomed to grazing. as maize silage supplemented with a protein con- Also, supplementation should be reintroduced in late concentrate and cereal. Profitable production is heavily summer to counter rapidly declining grass quality, start-dependent on a high yield of good quality maize silage ing at 1 kg daily and building up to 2 kg daily for a and achieving target daily gains. Continental ¥ Friesian month before housing.

heifers may be suitable for maize silage beef fed to Standards for the system are shown in Table 3.8. The gain about 1.0 kg/day and slaughtered at 435–450 kg overall daily gain of 0.9 kg for Limousin ¥ Friesian

liveweight.

steers is made up of 0.8 kg/day through the rearing winter, 0.8 kg/day during grazing and 1.1 kg/day in the finishing winter. The finishing gain would be achieved

Eighteen-month beef

at an average concentrate consumption of about 2.5 kg

Eighteen-month beef is a grass-based system that utilizes dairy-bred steers born in the autumn. It is the original planned system of production in the UK. Limousin × Friesian heifers would be

managed at an overall daily gain of 0.8 kg/day to a slaughter weight of 450 kg.

Calves are reared through their first winter, grazed from six to twelve months of age and then finished in their second winter on rationed concentrates with silage to

Grass beef

appetite. Heifers may be used for eighteen-month beef, but are better suited to grass finishing.

Grass beef is the most extensive of the planned dairy

Profitability depends heavily on achieving good

beef systems. Farmers see it as a low cost system that grazing performance. In farm practice this has taken maximizes subsidy income. At best, steers qualify for second priority to making a large tonnage of first-cut both BSP payments, an extensification premium and silage. This partly explains the poor financial margins the slaughter premium. Carcasses from continental achieved in recent years. In addition, the system has a cross steers may be too heavy for some buyers. Heifers relatively high concentrate requirement, a high silage are well suited to the system and can be more profitable requirement and, at best, steers only qualify for a single than unsubsidized steers.

BSP payment.

Good grazing gains are essential for profitable

The principles of good grazing management

production. If first season gains are below target,

described for grass finishing (see p. 17) apply equally to

winter store feeding costs are increased to catch up

eighteen-month beef. The cattle graze for the full

lost ground. Ideally, winter gains should be 0.5–

season and so, from mid-season, there is a need to
 0.6 kg/day for heifers and early maturing Hereford ¥
 expand onto fresh aftermath grazing on areas previ-
 Friesian steers and 0.7–0.8 kg/day for continental cross
 ously cut for silage as the cattle grow heavier, but grass
 steers.

Beef Finishing Systems • 21

Table 3.8

Standards for steers in eighteen-month beef.

Breed type

Holstein–

Hereford ¥

Limousin ¥

Belgian Blue

Charolais ¥ Friesian/

Friesian

Friesian

Friesian

¥ Friesian

Simmental ¥ Friesian

Daily gain (kg)

0.85

0.85

0.9

0.95

0.95

Slaughter age (months)

18

17

18

18

18

Concentrates (tonnes)a

1.3

0.9

1.0

1.1

1.2

Silage (tonnes @ 22% DM)a

5.8

5.1

6.0

5.7

5.5

Cattle/hectare

3.4

3.9

3.3

3.5

3.6

Slaughter weight (kg)

500

460

520

525

540

Carcass (kg)

270

250

290

295

300

a From 3 months.

Table 3.9

Standards for steers in grass beef production.

Breed type

Holstein–

Hereford ¥

Limousin ¥

Belgian Blue

Charolais ¥ Friesian/

Friesian

Friesian

Friesian

¥ Friesian

Simmental ¥ Friesian

Daily gain (kg)

0.73

0.68

0.75

0.80

0.83

Slaughter age (months)

22+

22+

22+

22+

22+

Concentrates (tonnes)a

0.4

0.3

0.5

0.5

0.6

Silage (tonnes @ 22% DM)a

7.0

5.7

6.7

6.7

6.7

Cattle/ha

1.6

1.7

1.6

1.6

1.6

Slaughter weight (kg)

555

515

585

595

610

Carcass (kg)

300

280

330

335

340

a From 3 months of age.

In the second grazing season steers need to be marketed as soon as possible after they qualify for the complex leader–follower grazing system was developed in which the younger cattle graze selectively ahead of second BSP to reduce stocking rate as the season progresses. Despite its merits, it is rarely marketed as soon as possible after they qualify for the complex leader–follower grazing system was developed in which the younger cattle graze selectively ahead of second BSP to reduce stocking rate as the season progresses. Marketing of heifers should start in mid-season.

adopted by farmers who prefer to keep the two age
This need to reduce stock numbers from mid-season
groups separate.

in step with declining grass production favours early
The principles of good grazing management
maturing types of cattle. So early maturing Hereford &
described for grass finishing (see p. 17) are applicable
Friesian steers fit the system better than late maturing
to grass beef. As with eighteen-month beef there is a
Holstein–Friesians or Charolais & Friesians that may
strong case for supplementing calves with about 1 kg
fail to finish off grass and have to be housed for a period
concentrate daily for two or three weeks after turn-out
of winter finishing. Similarly, early maturing heifers
in the spring and reintroducing supplementation in late
have advantages over later maturing steers. None-
summer to counter declining grass production and
theless, all the breed types, steers and heifers can be
quality.

utilized in the system provided their perfor-

Standards for steers in the system are presented in

mance characteristics are understood and managed

Table 3.9. As an example of standards for heifers,
accordingly.

Limousin & Friesians would have a lifetime gain of

Grazing management is complicated by the fact that

around 0.6 kg/day to slaughter at 20 months of age

there are two age groups of cattle on the farm. A

weighing 500 kg.

Chapter 4

Dairy Farming

A.H. Andrews and A. Poole

Structure in Europe and the world

22

herds consist of a single cow, often with a calf at foot,

Trends of structure in the United Kingdom

24

or very small numbers of low-yielding cows. This occurs

Milk utilization

26

in many parts of Africa and Asia, and in Europe

Dairy breeds

27

smallish herds are seen in countries such as Greece,

Feeding dairy cows

30

Portugal, Spain and Italy. While there have been very

Dry matter intake

30

gradual changes in the last half-century in Europe and

Energy

31

the United Kingdom, the introduction of milk quotas in

Protein

31

Feeding strategies

32

1984 accelerated these. In the last decade since the first

Minerals and vitamins

33

edition of *Bovine Medicine* was published the changes

Grassland farming

33

have been faster and at the start of the new twenty-first

Grassland production

33

century they have become very rapid indeed. There are

Grassland utilization

35

increasing trends towards a so-called 'global market'

Winter feeding systems

37

with a lowering of tariff barriers. If this continues this

Dairy farm buildings

38

will mean that many areas where milk price is directly or

Parlour and dairy

39

indirectly subsidized will have to deal with lowered

Cow housing and feeding barn

40

price for the product. Many areas with low labour costs

Feed storage area

41

and good weather to grow crops are starting to gear

Calving and isolation boxes

42

themselves up to the challenge of producing low cost

Slurry

42

Herd records

44

milk for export to other parts of the world. This is at

Physical records

44

present particularly occurring in North and South

Breeding records

45

America.

Milk records

45

Over the past few years much thought has been

Financial records

47

invested by the World Trade Organization, among

Management

47

others, about increasing the flow of milk and dairy prod-

Labour

47

ucts across the globe. However, the dairy markets are

Breeding

47

some of the most protected of agricultural products

Culling

47

and the Organization for Economic Co-operation and

Age at first calving

48

Development estimated in the late 1990s that member

Breed of sire used

48

states producer support arrangements were, in absolute

Genetic worth of the sire

48

Health

49

terms, higher than for all other commodities. It is this

Economics of dairy farming

50

background which is causing international trade organ-

Measures of efficiency

50

izations to find ways to reduce export subsidies paid

Farm assurance and herd health schemes

52

by countries and to alter tariff quotas for imported

Organic farming

52

products.

The future

52

The introduction of milk production quotas had a
very profound effect on the European milk industry. Its
aim was to reduce overall European milk production

Structure in Europe and the world

and the changes undertaken by producers to accom-

modate this altered many milk parameters. The structure in 1983 prior to the change is given in Table 4.1a and the present situation within the enlarged EU is shown in Table 4.1b. The position presented in Table 4.1a was reached over the previous 23 years by each country expanding its milk production through an industry and ranges from high-yielding cows in Israel, the USA and Canada as well as within European countries such as the Netherlands, Denmark and the UK to the small, low-yielding herds of Greece, Portugal, Spain and Italy. In many parts of the world milking expanded herd size and increased yield per cow. Total

22

Dairy Farming • 23

Table 4.1a

EEC dairy structure (pre milk quotas).

Cows

Herds

Average herd

Yield

Total

Self-sufficiency

(’000)

(’000)

size

(l/cow)

production

(’000 t)

BF

SNF

(%)

(%)

Germany

5451

363

15.3

4650

26 007

116

136

France

6506

367

19.8

3950

33 337

122

128

Italy

3120

424

7.3

3540

11 030

66

63

Netherlands

2333

58

40.8

5330

12 550

256

116

Belgium

946

45

21.7

3930

4145

104

116

}

Luxembourg

70

2

29.7

4274

320

UK

3257

54

58.2

4906

17 680

86

104

Irish Republic

1528

77

19.9

3910

6 480

298

175

Denmark

913

35

28.2

5585

5 205

212

142

Greece

219

77

3.1

3200

770

—

—

Portugal

369

115

3.2

3021

1 115

—

—

Spain

1885

—

—

3382

6 375

—

—

Source: Milk Marketing Board (1986).

Table 4.1b

EU dairy structure in 2000 (post milk quotas).

Country

Cows

Herds (1997)

Average herd

Yield

Total

Butterfat

Protein

(2000)

(’000)

size (1997)

(2000)

production

content

content

(’000)

(kg/cow)

(2000)

(2000)

(2000)

('000 t)

(% by weight)

(% by weight)

Germany

4 539

186

27.9

6157

28 332

4.22

3.41

France

4 413

146

30.7

5844

24 775

4.08

3.19

Italy

2 172

102

20.5

5113

10 774

3.66

3.26

Netherlands

1 532

37

44.0

7302

11 155

4.40

3.46

Belgium

629

20

32.3

5420

3 383

4.09

3.34

Luxembourg

44

1

36.5

6030

265

4.19

3.34

United

2 339

36

68.8

6133

14 489

4.01

3.30

Kingdom

Irish

1 238

39

32.4

4191

5 280

3.70

3.23

Republic

Denmark

644

13

50.8

7272

4 720

4.28

3.44

Greece

173

24

7.7

4756

805

3.67

3.22

Spain

1 141

106

11.9

5007

5 900

3.75

3.09

Portugal

355

70

5.2

5819

2 057

3.84

3.19

Austria

621

86

8.4

4977

3 340

4.13

3.33

Finland

358

29

13.3

6916

2 524

4.22

3.29

Sweden

426

16

29.6

7748

3 348

4.18

3.31

EU Fifteen

20 624

911

24.0

5885

121 146

4.07

3.31

Sources: various including mainly National Dairy Council (2001).

cow population did not increase to the same extent as

The Common Agricultural Policy (CAP) set out to

the number of dairy producers gradually declined. The

increase food production, maintain farm incomes and

reasons for this were due to both individual national

prevent rural depopulation. This encouraged the very

policies and the then EC requirements.

small farms of much of Europe to survive and expand,

Table 4.2

Production and milk quota ('000 tonnes) for EU

quota producers at a rate of 100 per cent of the target

Member States.

price for milk. On an on-farm basis, provided national

production exceeded quota, each farm paid super-levy

Production

Milk quota

on excess production above its individual quota at

a rate of 75 per cent of the target price for milk. In

1983

1983–84

1991–92

2000–2001

1987–88 further changes to the means of determining

super-levy liability were made so that those producers

Germany

26 429

23 792

22 927

27 865

most over quota paid the highest proportion of the levy,

France

33 230

26 768

24 613

24 236

Italy

11 310

9 914

9 221

10 314

again provided the national quota was exceeded.

Netherlands

12 782

12 197

11 213

11 075

Quota has been transferable between individual

Belgium

4 149

3 643

3 364

3 310

farmers within a member state, either on a sale

Luxembourg

319

294

272

269

(attached to land or without, but both are complex) or

UK

17 878

15 950

14 789

14 602

lease basis (a much simpler procedure) or else on an

Irish Republic

6 380

5 599

5 301

5 342

administratively based system where unused quota

Denmark

5 361

4 933

4 525

4 455

reverts to an authority and is re-allocated. Further reg-

Greece

765

588

581

675

ulation in 1987 imposed a butterfat ceiling on quotas,

Spain

5 917

thereby giving a reduction in national quota if the

Portugal

1 872

average butterfat of 1985–86 was exceeded. Also in

Austria

2 749

1987 further quota cuts were announced, resulting in a

Finland

2 407

Sweden

3 303

further fall by 1989 of 8 per cent. The calculation of any

EU Fifteen

118 391

liability for excess production for a farmer is extremely complex. It depends on the amount of milk over quota

Sources: Milk Marketing Board (1990); National Dairy Council (2001).

and on the average butterfat content of the milk. Each producer has a butterfat reference figure. If a levy is to be charged then the farmer's production volume will be adjusted up or down by the amount that the butterfat while for the larger producers, financial help made dairy average differs from his/her reference figure. Again farming relatively profitable compared with other levies only arise when national production is too sectors of agriculture. Thus total milk production for high.

the 10 members of the EC at that time rose from 87.5

The objective of reducing milk production has been million tonnes in 1965 to 120.7 million tonnes in 1983. achieved. Initially it was envisaged that milk quotas The success of the EU milk quota in controlling would be phased out in the early 2000s. However, this production is demonstrated by the fact that even in has not been the case and the CAP reform has extended the expanded Union total milk production was 120.9 milk quota until 2006. Special increases in quota were million tonnes in 1998.

allocated to Italy, Spain, Greece, Ireland and Northern While the CAP was proving very satisfactory for Ireland in 2000 and 2001 in addition to the increase of dairy farmers, the financial support they were receiving 1.5 per cent given to all countries in three stages from was increasingly threatening the whole EU budget. 2005. The intervention price will be gradually cut from Most of this aid was provided through intervention 2005 and it is expected that the system will have been buying of surplus products. In fact, sale into interven- re-examined in detail by then.

tion became the accepted market rather than the market of last resort. Thus by 1983, self-sufficiency

Trends of structure in the United Kingdom

levels rose within the EC to 125 per cent for butterfat (BF) and 166 per cent for solids-not-fats (SNF) (Table Changes to the structure in dairy farming in the United 4.1a) and the cost of dairy support made up one quarter of the whole EC agricultural budget. Thus at this stage, years in Europe. Thus there are fewer producers and milk quotas were imposed to control milk production slightly fewer cows, but in larger herds and with a higher and each member country had to take a cut of varying yield per cow (Table 4.3). This has been brought about proportions (Table 4.2).

by the virtual elimination of the small herd. In 1972, 20 If national production exceeded the allocated quota per cent of cows were in herds of less than 30 cows, but then a super-levy was imposed. This super-levy was to by 1997 it was about 5 per cent. This acceleration has be calculated either on a dairy basis or on an on-farm

increased at the beginning of the new century as milk basis. On a dairy basis, if national production and the prices dropped, causing most farms either just to break supplying dairy were over quota, then levy was imposed even or to lose money. The foot and mouth disease outbreak on the dairy and hence back to the individual overbreak of 2001 has caused more producers to cease milk



Dairy Farming • 25

production, and present numbers of herds are likely to decrease sharply.

milking installations in 1972, but made up over 45

The December 2001 census showed UK dairy cows

per cent in 1990 (Fig. 4.1), and are the main system down to 2.203 million with in-calf heifers down to 0.442 today.

million. While beef cows were down to 1.673 million, in- The increased yields have been realized by improved calf beef heifer numbers rose.

feeding (see Chapter 9), breeding and management The changes to larger herds have been facilitated by techniques. The breed of cow in the UK has altered, 64 new technology and mechanization and a consequent per cent Friesian in 1965 moving to over 91 per cent loss of labour. In 1972, 67 per cent of farms milked Holstein–Friesian by 1990, which has also made a major through a cowshed, with only 28 per cent in 1990. contribution.

Milk purchase has also undergone some very fundamental changes in the last century. Initially farmers sold direct to dairy companies. However, as the latter

Table 4.3

The structure of dairying in England and Wales, 1965 became larger the individual farmer had problems in

to 2001.

obtaining a fair price. This resulted in Agricultural Marketing Acts in 1931 and 1933, which set up a framework

Year

Producers

Cows

Average

Yield

whereby all the milk produced was taken by the Milk

(’000)

(millions)

herd size

(l/cow)

Marketing Boards who then had to sell it on the producer’s behalf. The Agricultural Act of 1993 allowed the

1965

100.5

2.65

26

3545

1970

80.3

2.71

33

3755

abolition of the Milk Marketing Boards in Great

1975

60.3

2.70

46

4070

Britain and from 1 November 1994 the Milk Marketing

1980

43.4

2.67

58

4715

Scheme was revoked (Northern Ireland from 1 March

1984

39.3

2.70

67

4950

1995). In the deregulated market, producers are free to

1988

31.7

2.38

69

4870

sell their milk wherever they choose including direct

1990

31.5

2.33

70

5020

to dairy companies, or via intermediary organizations

1995

28.1

2.10

74

5380a

managed jointly by the dairy company and producers,

1998

24.7

1.92

77

5770a

or independent producer groups, usually co-operatives,

1999

23.3

1.94

82

5955a

who either then sell to the dairy companies or process

2000

21.8

1.84

85

5940a

the milk themselves. Many sold milk via Milk Marque,

2001

20.2

1.76

which was considered in 1999 to be a monopoly, result-

a

ing in its division into three new regionally based co-

United Kingdom figures.

Source: Milk Marketing Board, 1990; National Dairy Council, 2001.

operatives, namely Axis, Milklink and Zenith, which

Fig. 4.1

Milking in the herringbone

parlour.



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Table 4.4

Utilization of milk in England and Wales (%) to 1989–90 and in the United Kingdom (%) to 2000.

Liquid

Butter

Cheese

Condensed

Cream

Others

milk

milk

1964–65

73

3

11

5

5

3

1969–70

66

10

10

4

7

3

1974–75

62

8

16

4

8

2

1979–80

50

23

15

3

7

2

1982–83

45

31

14

2

6

2

1983–84

45

30

14

2

6

3

1984–85

48

27

15

2

5

3

1987–88

49

23

19

2

3

4

1989–90

51

20

18

2

4

4

1993–94a

86

2

4

2

3

3

2000a

50

2

22

4

2

20

Source: Milk Marketing Board (1986); National Dairy Council (2001).

a Figures on a different basis to previously.

began to operate as fully independent businesses from 1 April 2000. There has since been more rationalization and mergers, with one group involved in milk purchase in Scotland, England and Wales becoming known as British Milk. Some milk purchasers now have their headquarters outside the UK.

The last few years have seen many milk purchasers buying milk from individual farmers or small groups or farmer clubs. In the early part of the twenty-first century the number of purchasers has been reducing and farmers are starting to combine in co-operatives to provide more negotiating power with the purchasers. Milk is now purchased in varying ways, but is usually paid for according to its composition, cell count and bacterial count (undertaken in the UK by a Bactoscan, see Chapter 25). There are also seasonality payments.

Milk utilization

Fig. 4.2

A modern butter-making creamery produces and packs 5 tonnes of butter an hour.

While milk production has increased over the last 25 years, milk consumption has slightly reduced. The amount used as liquid milk has declined from 73 to 45 per cent. However, because of quotas this percentage This changed with increasing amounts of milk being has increased again (Table 4.4). Self-sufficiency in 1999 used for manufacturing. At one stage (Table 4.4) butter compared with 1996 in the UK was 74 per cent (68 per manufacture increased (Fig. 4.2) with much interven- cent) for butter, 64 per cent (69 per cent) for cheese, tion producing the so-called 'butter mountain'. With 128 per cent (129 per cent) for condensed milk, 147 per liquid milk sales realizing the highest return to the pro- cent (147 per cent) for cream and 187 per cent (112 per ducer, the producers in England and Wales should be cent) for skim milk powder.

in a more favourable position than the rest of the EU, Prior to the EU, the UK was mainly concerned with as shown in Table 4.5. The large quantities of other uses supplying the liquid market, with Commonwealth constitute mainly milk powder and milk fed to live-

countries supplying butter and cheese requirements.

stock, which is common practice in some countries.

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Table 4.5

Utilization of whole milk, 1984–1989 (%).

Liquid

Butter

Cheese

Condensed Cream

Others

Milk

milk

'84

'89

'84

'89

'84

'89

'84

'89

'84

'89

'84

'89

Germany

13

14

46

34

13

17

4

3

9

13

15

19

France

9

17

38

36

22

28

1

1

4

5

26

13

Italy

29

28

14

14

44

43

—

—

4

5

9

10

Netherlands

6

6

42

36

27

32

8

6

3

4

14

16

Belgium

15

16

55

51

5

7

—

1

5

8

20

17

Luxembourg

11

13

56

53

3

4

—

—

9

13

21

17

UK

41

48

26

20

14

19

2

3

4

3

13

7

Irish Republic

10

11

62

58

9

13

2

2

3

4

14

12

Denmark

8

6

38

37

30

32

—

—

5

8

19

17

Greece

29

30

3

2

47

53

—

—

2

3

19

12

Source: Milk Marketing Board (1986, 1990).

Table 4.6

Dairy herd breed distribution.

Approximate

Yield

Butterfat

Protein

percentage of

(kg/cow)

(%)

(%)

national herd

Ayrshire

2.0

5887

4.04

3.34

Holstein/Friesian

90.0

6960

3.98

3.27

Guernsey

1.8

4899

4.76

3.58

Jersey

1.6

4708

5.40

3.29

Dairy Shorthorn

0.3

5589

3.88

3.30

Others and crosses

4.3

—

—

—

Sources: various, including National Dairy Council (2001).

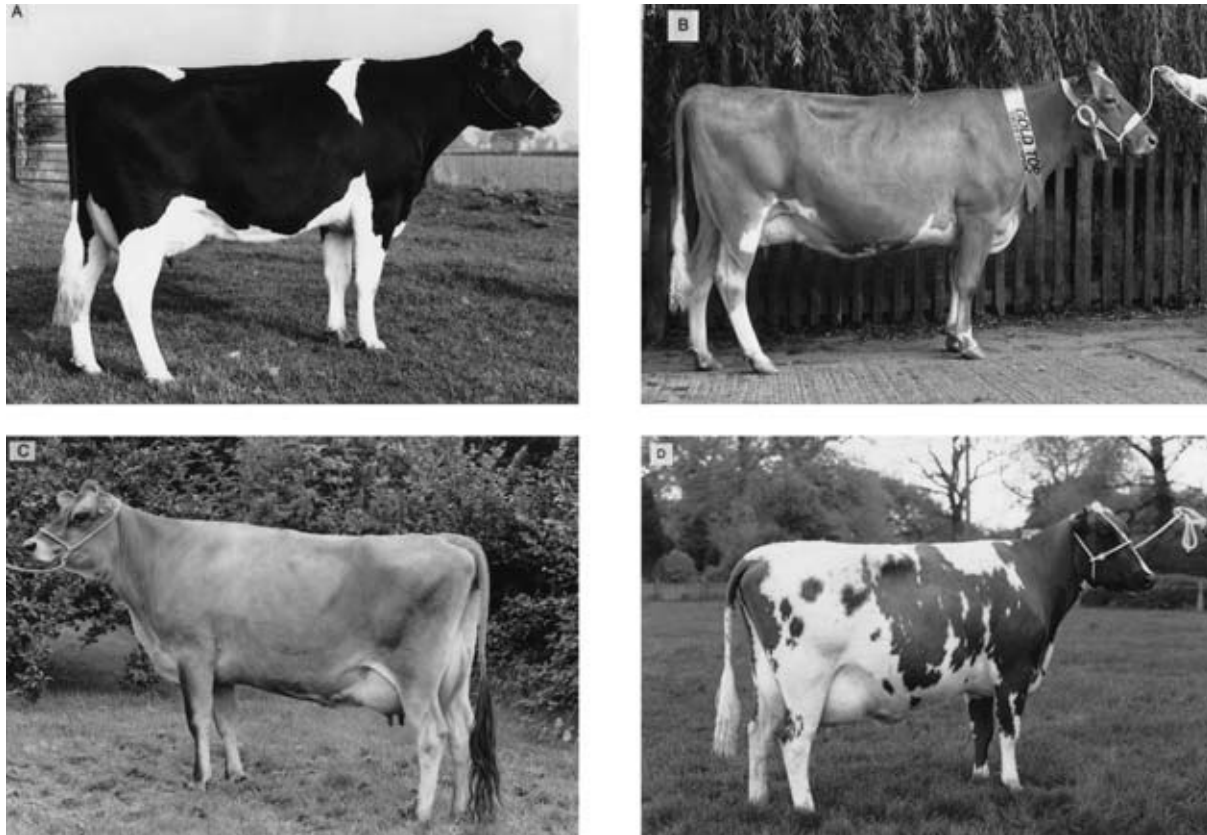
While this table relates to the 1980s, proportions are relatively similar today. The surplus situation in Europe the world. While every breed has its enthusiasts, the vast majority of herds are mainly Holstein–Friesian type consumed being skimmed milk, or semi-skimmed milk, (Fig. 4.3). These black and white cows are renowned for allowing the release of milk for butter production. The their high yields of average quality milk. As they last 20 years have also seen a similar trend in the UK, become more extreme, the surplus males are of less use with about 20 per cent of liquid milk consumed as skim

for beef. Recently the input costs have been examined or semi-skim in the 1980s, 55 per cent in 1994 and 62 by some farmers and there is a slight tendency for those per cent in 1998. The further quota restrictions have who wish low input production to favour animals of a taken this into account.

less extreme Holstein type. These animals are also often favoured by the organic milk producers. However, those aiming at the very high yielding herd are still pursuing a Holstein type of animal. The main drawback of

Dairy breeds

such animals is that any surplus calves produced are of limited use in beef production. Such problems in the The principal dairy breeds of the United Kingdom are future may be overcome by the use of sexed semen and given in Table 4.6. These breeds or their local variations then possibly by the one of embryo transfer in those



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Fig. 4.3

(a) A Friesian cow, (b) a Guernsey cow, (c) a Jersey cow, (d) an Ayrshire cow.

cows not required to produce dairy herd replacements.

decreased, the breed has currently been given a boost

The increased interest in organic farming and sustain-

as its milk is being specifically retailed by some chain

able agriculture is likely to be reflected in an increase

food stores. A smaller cow than the Friesian, it produces

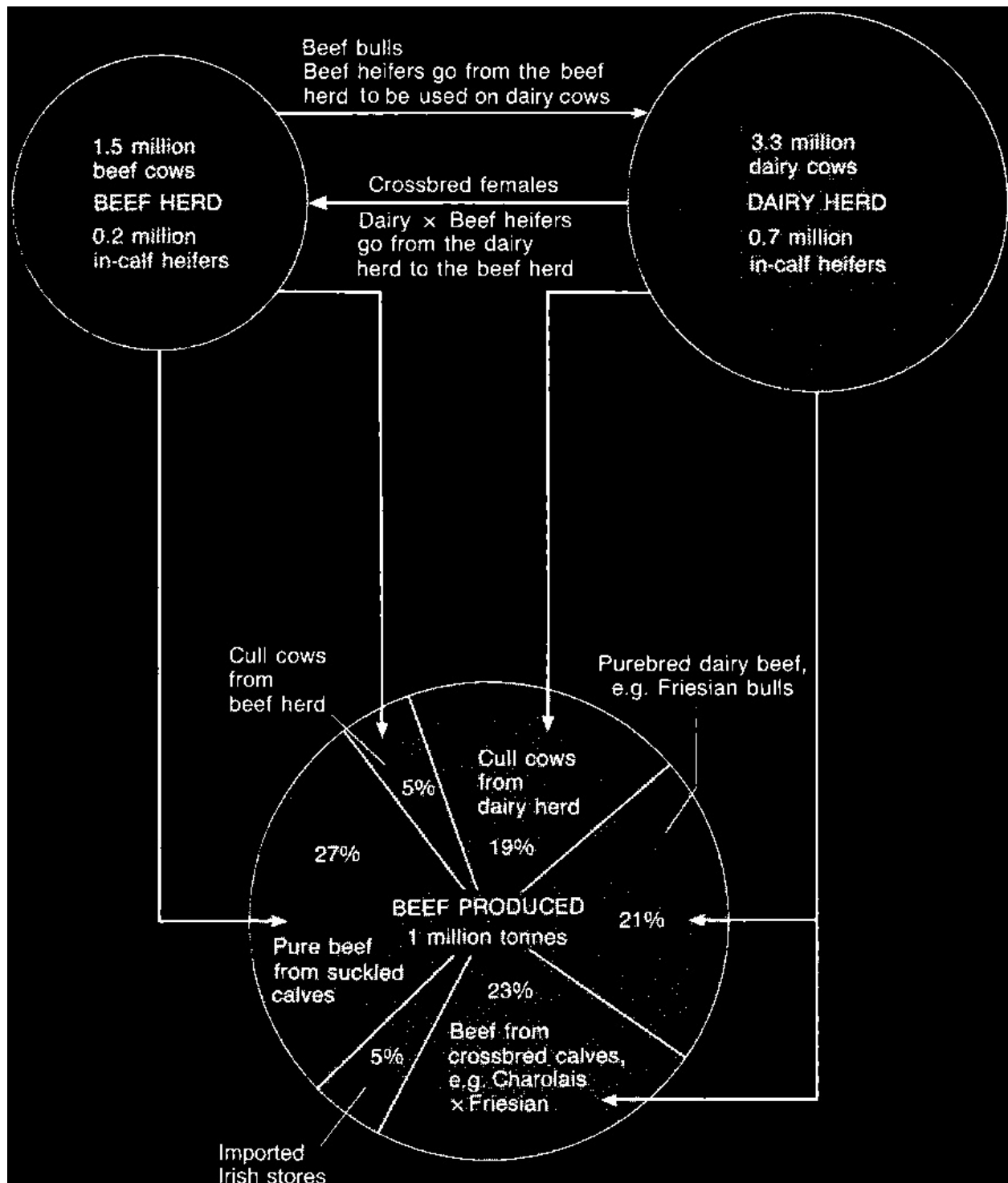
in usage of Friesian-type animals, which are more

lower milk yields but of higher quality. It is a breed adjusted to such production systems than the Holstein. renowned for the dairy type and so is not very suitable The Channel Island breeds, Guernseys and Jerseys, for beef.

are still popular with a minority of breeders. They are The Dairy Shorthorn has suffered from the demise of docile cows producing lower yields of very high quality being the major breed pre World War II, now slipping 'gold top' milk. This sells at a premium to the liquid to almost insignificance. With good beefing qualities, it milk or cream markets. The milk is now often sold tended to fall between two stools, with neither the milk homogenized. The breed does produce very efficiently quantity of the Friesian nor the quality of the Channel on the basis of weight of milk constituents produced. Island breeds. Types of the breed are used in some The beefing qualities are low and the fat tends to countries. Its use may also increase on farms practising be yellow, but the meat can be acceptable when sustainable or organic agriculture.

crossed with one of the continental beef breeds such as The beefing characteristics of the dairy breeds are of Charolais, Limousin, Simmental or Belgian Blue. Dys-importance in England and Wales as the majority of the tokia problems in the Channel Island breeds are few beef produced originates from the dairy herd. Until because of a broad and pliable pelvis that allows delivery of viable live calves when crossed with the large March 1996, the origin of British beef was as in Fig. 4.4. The Over Thirty Month Scheme (OTMS), which pre-beef breeds.

vented the sale of older animals for human consumption. The Ayrshire is still regionally very popular in a few parts of its native Scotland. While its numbers have which paid a premium for calves slaughtered before 20



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Fig. 4.4

Sources of beef in the UK.

Source: Meat and Livestock Commission.

Table 4.7

Sources of home-produced beef. Reproduced with permission from Meat and Livestock Commission (1999).
derived from the dairy herd. Recent BSE problems in other parts of Europe are also altering the types of animal which are entering the meat industry for use

1993

1995

1996

1998

both directly and in manufacturing. In Britain, should

(%)

(%)

(%)

(%)

the OTMS be removed, it will cause another alteration

in the constituents of beef which is consumed. At

UK-bred steers,

78

76

92

100

present, increased proportions of total beef production

heifers and

are being used for manufacturing and catering and so

young bulls

beef herd

34

35

43

52

if human consumption of over thirty-month-old beef

dairy herd

44

41

49

48

is again allowed, there should be a market for the

product.

Cull cows

19

22

8

0

The Friesian breed has played an important part in
beef herd

8

6

3

0

beef production with most farmers producing sufficient
dairy herd

11

16

5

0

of their cows pure to provide the heifer replacements

Adult bulls

1

1

0

0

for the dairy herd, with the remainder being bred to a

Irish cattle

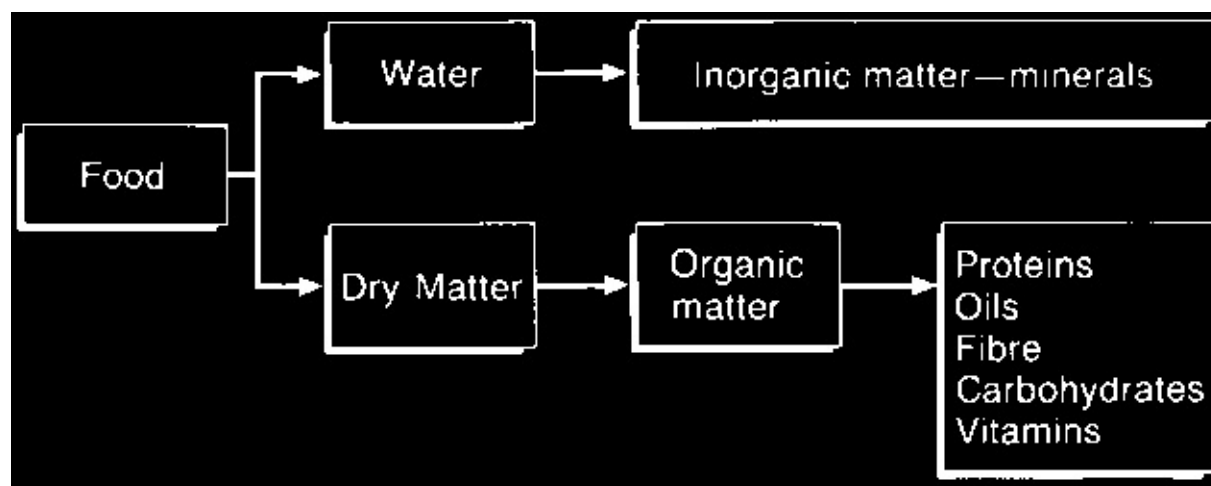
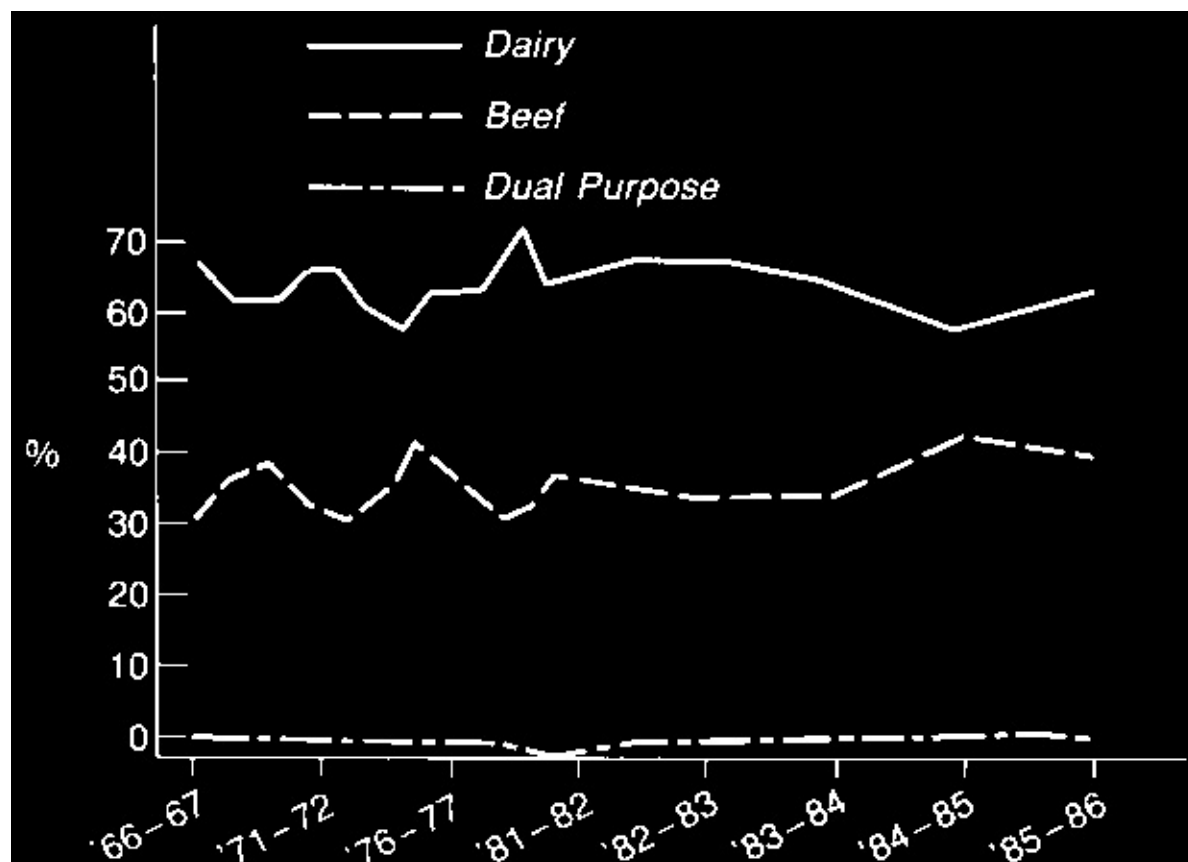
1

1

0

0

beef bull to maximize calf returns. Figure 4.5 shows that, in most years, 60–70 per cent of inseminations have been to a dairy bull, with the remainder to beef breeds. days of age, payable from May 1996 until 31 July 1999, These data cannot at present be updated, but the most altered these proportions so that in the early twenty- numerous breeds of bull used for artificial insemination first century the origins are as in Table 4.7. These are (AI) in 1986 are shown in Table 4.8. While the dairy likely to alter with an increase in the amount of beef inseminations follow the pattern of breed distribution,



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Fig. 4.6

Food components.

dependent on the price and the quality of the bulls

offered.

Fig. 4.5

Dairy, dual purpose and beef inseminations as per-

centages of total inseminations. Source: *Report of Breeding and Production Organization*, MMB (1986).

Feeding dairy cows (see also Chapter 9)

A vital feature of dairy cow management is correct feeding. Food, either purchased or home produced,

Table 4.8

AI breed demand in MMB areas, 1986 (%).

amounts to 60 per cent of the variable costs of production so the efficiency of feed use is extremely im-

Ayrshire

0.7

portant. In the past, most high-energy feeds were

Friesian/Holstein

57.8

purchased as concentrates from feed manufacturing

Guernsey

0.8

companies. The onset of bigger herds, access to machin-

Jersey

1.2

ery to mix up feeds and the ability to buy feed con-

Shorthorn

0.2

Total dairy breeds

60.7

stituents at competitive prices have meant that many

Aberdeen Angus

2.8

dairy and beef farmers now produce their own rations,

Belgian Blue

1.1

often feeding them as complete feeds or total mixed

Charolais

7.4

rations (TMR).

Hereford

12.0

The main components of feed are shown in Fig. 4.6.

Limousin

12.7

All feeds contain water in varying amounts. Dry feeds

Simmental

1.7

such as cereals will only have about 14 per cent water,

Others

1.6

while other feeds such as mangels and other root crops

Total beef breeds

39.3

will be up to 90 per cent water. While all cows require

water it is the dry matter proportion of the total food

Source: *Report of the Breeding and Production Organization*, MMB

(1986).

that supplies the nutrients for maintenance and pro-

duction. In general, the higher the dry matter of the

overall diet, the higher will be the dry matter intake

(DMI). Apart from the need for minerals and vitamins,

the main aspects to consider in dairy cow feeding are

with beef the continental breeds are replacing the tra-

DMI and energy and protein levels.

ditional British beef breeds, especially the Hereford.

The Belgian Blue is more widely used in the UK than shown in Table 4.8, but again recent figures are unob-

Dry matter intake

tainable. The Aberdeen Angus continues to enjoy some support because of the ease of calving, particularly

Dry matter intake (DMI) is affected by the liveweight when used on maiden heifers or small cows. It also has of the cow, stage of lactation, milk yield, type of feed some marketing advantages in that Angus beef can and frequency of feeding. As a guide for estimating demand a premium.

DMI the following equation is used:

While there is an increasing amount of embryo trans-

$$\text{DMI (kg/day)} = 0.025 \times \text{liveweight (kg)} + 0.1$$

fer occurring and costs have reduced, it is still an

¥ milk yield (kg/day)

uncommon procedure. However, the possible use of sexed semen does appear to offer considerable possi-

At calving DMI will be 2–3 kg/day lower than calculations over the next few years. Its uptake will be very

lated, but it will then increase to peak at three to five

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Table 4.9

Energy requirements of dairy cows.

Breed

Liveweight

Maintenance

Production

Liveweight gain

Pregnancy

(kg)

(MJ/day)

(MJ/kg milk)

(MJ/kg gain)

(MJ/day)

Ayrshire

500

54

5.3

34

23

Friesian

600

63

5.2

34

23

Guernsey

450

49

5.8

34

23

Jersey

350

40

5.9

34

23

Shorthorn

550

59

5.1

34

23

Table 4.10

Digestible crude protein requirements of dairy cows.

Breed

Liveweight

Maintenance

Production

Pregnancy

(kg)

(g/day)

(g/kg milk)

(g/day)

Ayrshire

500

300

60

150

Friesian

600

350

55

150

Guernsey

450

275

70

150

Jersey

350

225

70

150

Shorthorn

550

325

55

150

months after calving. After peak milk production,

Maintenance

63 MJ

intake decreases slowly through lactation and is lower

Production (20 ¥ 5.2)

104 MJ

during the dry period. DMI can be increased by feeding

Liveweight gain (0.5 ¥ 34)

17 MJ

highly or more digestible feeds such as maize silage.

Pregnancy

23 MJ

Thus cows will eat greater quantities of a 70 per cent

Total energy required

207 MJ/day

digestibility (D) silage then one of 64 D. Also succulent

feeds such as brewers' grains, kale and roots consider-

Protein (see Chapter 9)

ably enhance intakes in an otherwise dry diet of hay or

silage with concentrates. When feeding large quantities

Protein is required for both maintenance and produc-

of concentrates, splitting the feed into three or four

tion. In contrast to the case for energy, body tissues

feeds rather than the conventional two in the parlour

cannot be used to make up a shortfall in protein and so will also increase intake and reduce rumen acidity. This milk yields will suffer if protein is in deficit, especially is also why TMR can work well and again it will increase in early lactation. Protein is often measured as crude feed intake.

protein (CP) and is given as percentage of dry matter.

In rations it used to be measured in grammes of digestible crude protein (DCP) per kilogram DM.

Energy (see Chapter 9)

Requirements for maintenance and production are

The energy requirements of a dairy cow depend on shown in Table 4.10.

her needs for maintenance, lactation, pregnancy and

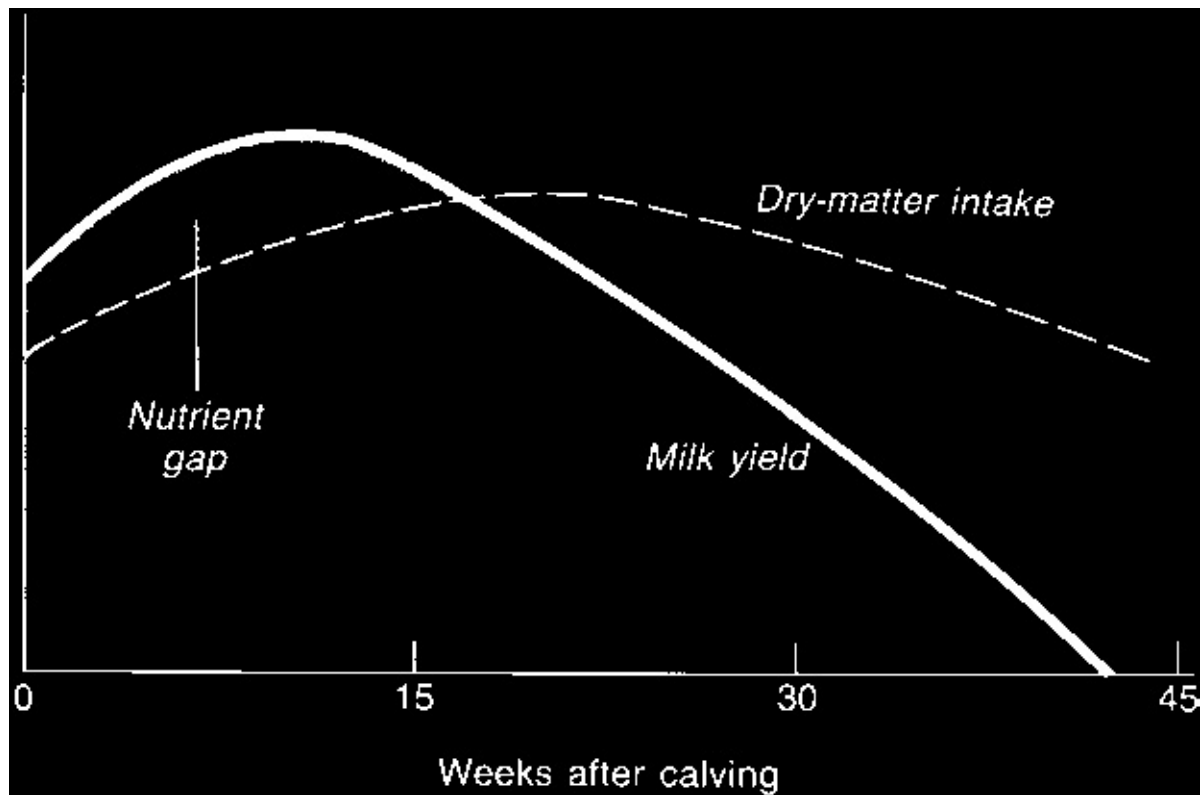
A refinement of the DCP system is the division

whether she is increasing or decreasing weight (Table of rumen degradable protein (RDP) and rumen

4.9). Energy in diets in Britain is measured as metabo- undegradable protein (UDP). A dairy cow has a need

lizable energy (ME) and the units are megajoules (MJ) for both types of protein. The system has been further

per kilogramme dry matter (DM). If dairy cows are in energy deficit then body fat will be mobilized, causing liveweight loss to make up for some or all of the deficit. This looks at the rate at which protein becomes available within the rumen and is given as quickly degradable protein (QDP) and slowly degradable protein (SDP). These two (QDP + SDP) form what is known as the effective rumen degradable protein (ERDP). A cow producing 20 kg/day of milk, in calf and gaining 0.5 kg/day liveweight would require: quick and slow) and rumen undegradable protein.



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Different feeds contain different proportions or ratings for the first 120 days of lactation. As yield declines and

of these. Thus silage is about 80 per cent degradable and appetite increases then balance should be reached soya is about 60 per cent degradable. Also, degradability and then any liveweight loss in early lactation can be ity does depend on the form of the feed, thus heating replaced in mid to late lactation (see Fig. 4.7 and or formalin treatment reduces rumen degradability. The Chapter 9). As will be discussed later, a compromise has rate of passage out of the rumen also has an effect, with to be achieved between biological efficiency and eco-higher passage rates increasing the UDP. nomic efficiency.

In many instances, although the protein supplied may Very approximately, the most common feeds for be sufficient in terms of its CP or DCP basis, it may still dairy cows (grazing grass, grass silage and concentrates) be deficient in either RDP or UDP. In early lactation have a relative cost of 1 : 2 : 3. Therefore, greater profits on a predominantly grass silage diet, any shortfall is will be shown as the proportion of grass and silage in likely to be in UDP; this explains the observed good

the total diet increases provided performance does not response when fishmeal or soya was added. Meat and suffer (see Fig. 4.8).

bone meal are also a good source of UDP but were While flat rate feeding of concentrates is popular, banned in Great Britain in 1988 for ruminants and in particularly with total mixed rations (TMR), most the rest of the EU finally in 2001. The EU also decided farmers feed at least partly to yield. In most instances to ban the use of fishmeal in ruminant diets from 1 January 2001, resulting in the loss of a major practical source of UDP. However, it may return.

Feeding strategies

Many textbooks have been written on strategies for feeding dairy cows. The main principles are (i) to maximize DMI and (ii) to provide the cow's requirements as cheaply as possible. These two requirements are associated as the higher the DMI then the lower the energy concentration in the diet and hence the cheaper the production of the ration.

In early lactation of high-yielding cows, it will be dif-

ficult to satisfy their requirements within appetite. This will result in a loss of bodyweight, perhaps 0.5 kg/day,

Fig. 4.7

Nutrient gap.

Fig. 4.8

Feeding silage at a barrier.

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Table 4.11

Examples of possible cow diets.

Table 4.12

Main mineral requirement of dairy cows.

Stage of lactation

Ca

P

Mg

Na

Early

Mid

Late

Maintenance (g/day)

Liveweight (kg)

Concentrates (kg DM/day)

8

6

2

400

14

19

6

7

Silage (kg DM/day)

8

12

12

500

18

26

8

9

Total DMI (kg/day)

16

18

14

600

21

32

9

10

Energy supplied (MJ/day)

180

195

145

Energy required (MJ/day)

195

186

133

Production (g/10 kg milk)

Fat content of milk (%)

4.0

28

17

6

6

this is important as dairy cows are not similar enough

5.0

30

17

6

6

in their genetic make-up to allow feeding as if a single animal. It also cannot work unless cows are all at the same stage of lactation rather than calving over wide-spread periods or all the year round. Thus most farmers do to a certain extent 'feed to yield' or requirements, consider are grassland production and grassland whereby concentrates are allocated on an individual utilization.

basis depending on the yield attained and/or condition loss. In consequence, more concentrates are fed early

Grassland production

on in lactation and are reduced as lactation progresses.

Appetite is dependent on DMI so as concentrate con-

To get optimum production from grassland the basics sumption increases, forage intake reduces. This has the

must be correct. These include climate and soil type effect of limiting forage intake in early lactation about which little can be done. Grass grows best on a as concentrates are normally eaten in preference to medium loam soil with adequate summer rainfall. forages. Table 4.11 shows the effect of this on the cow's These conditions are most often found in the west of diet. For correct digestion the dry matter ratio between England and Wales. Where soil is heavy and water-concentrates and forage should be in the range 70 : 30 logged, then drainage can give good results. Conversely, to 30 : 70 in early to mid lactation. Any lower and per- in dry areas, irrigation has a part to play, although the formance will suffer, any higher and digestion will economics of grassland irrigation in Britain are ques- suffer (see pages 829–32).

tionable on all except the driest farms. Soil acidity should be corrected to a pH of 5.5–6.5.

Having achieved as near optimum as possible with

Minerals and vitamins

the basics the next area to consider is grass varieties.

The daily requirements for these must be adequately met. For grazing, most seed mixtures are based on varieties of perennial ryegrass (Fig. 4.9). There are many recommended varieties in the National Institute of Agricultural Botany (NIAB) lists. Timothy is often sown, which adds some palatability and midsummer growth. Older grass varieties such as meadow fescue and cocksfoot are not often sown now. Most mixtures will also include some white clover. This provides nutrients by fixing atmospheric nitrogen and also increases intake by concentrates are fed these will usually produce improving the palatability of the sward. But clover is sufficient of these nutrients for the productive cow.

difficult to establish and maintain in grassland receiv-

However, where rations are all or nearly all TMR then
ing high levels of fertilizer nitrogen.

addition of adequate amounts of vitamins and minerals

Once established, and with good management, a
is essential.

grazing sward should remain highly productive for 10
years or more. The main enemy to this is grazing in too
wet conditions, when treading and poaching will quickly

Grassland farming

destroy a sward and allow weed grasses to establish.

Swards mainly for cutting are usually based on a shorter

Grass and its derivatives, hay and silage, form an impor-

term perennial ryegrass or Italian ryegrass. These have

tant part of dairy farming. The two major aspects to

a more erect growth habit and are easier to cut but are



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Fig. 4.9

Cow set stocking, grazing a rye-grass sward.

Fig. 4.10

A good response is shown from

nitrogen fertilizer.

not so persistent. They are often grown as a grass break

Having established the grass the fertilizer require-

in an arable rotation for two to three years. Often the

ment must be satisfied. Normal recommendations for

species are sown pure. If clover is required then red

grazing would be an annual application of 300 kg/ha

clover is more suited to a cutting regimen than white

nitrogen, 40 kg/ha phosphate and 40 kg/ha potash.

clover. In between these extremes are many dual

Under a cutting regimen the nitrogen and potash

purpose mixtures that are both grazed and cut during a

would be increased to 350 and 150 kg respectively.

season.

Whilst phosphate and potash are essential for grass

The best time to sow grass is in August–September.

growth the main response is shown to nitrogen. Much

This gives it a chance to establish before winter and

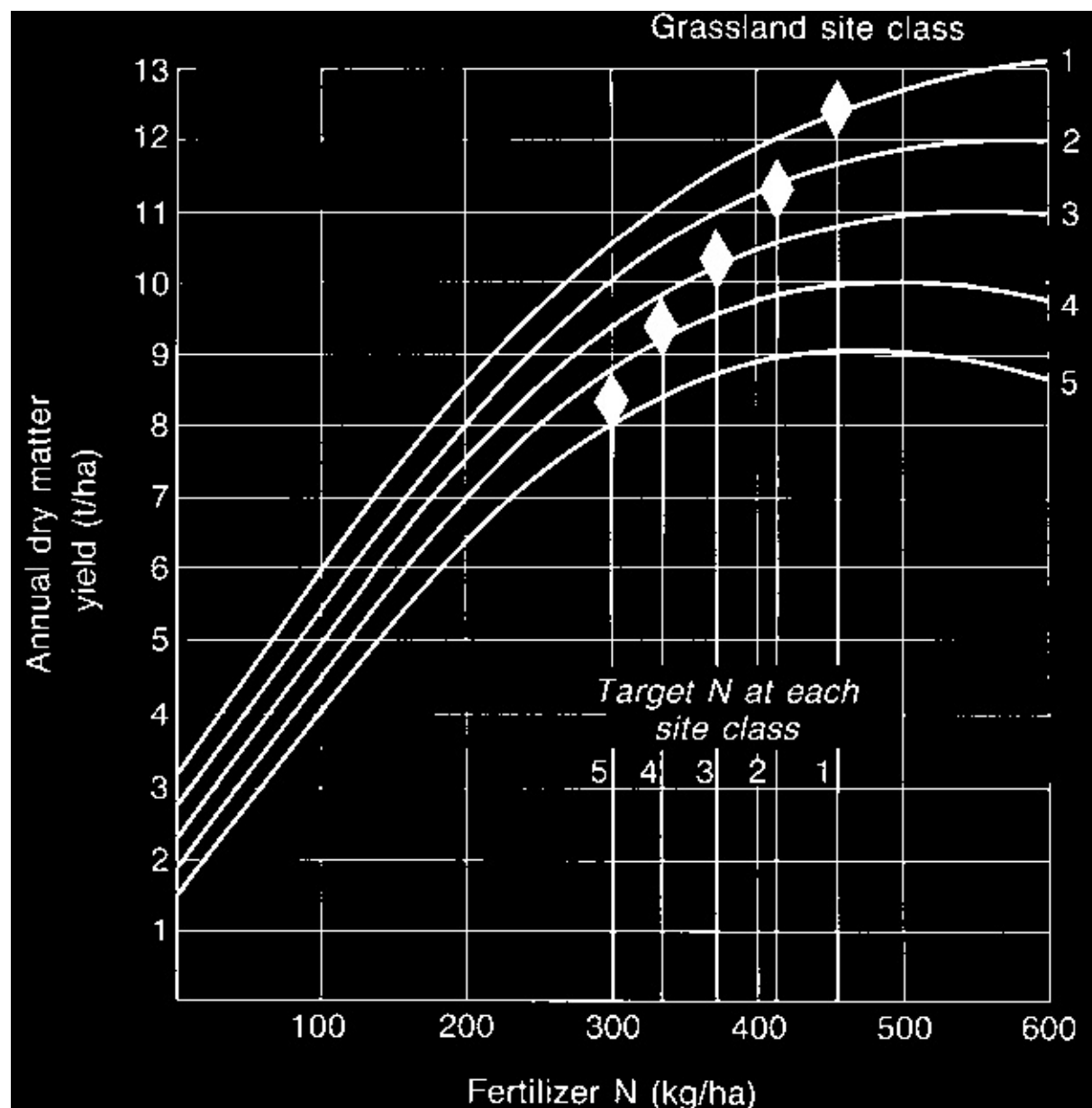
trial work has been undertaken showing a good

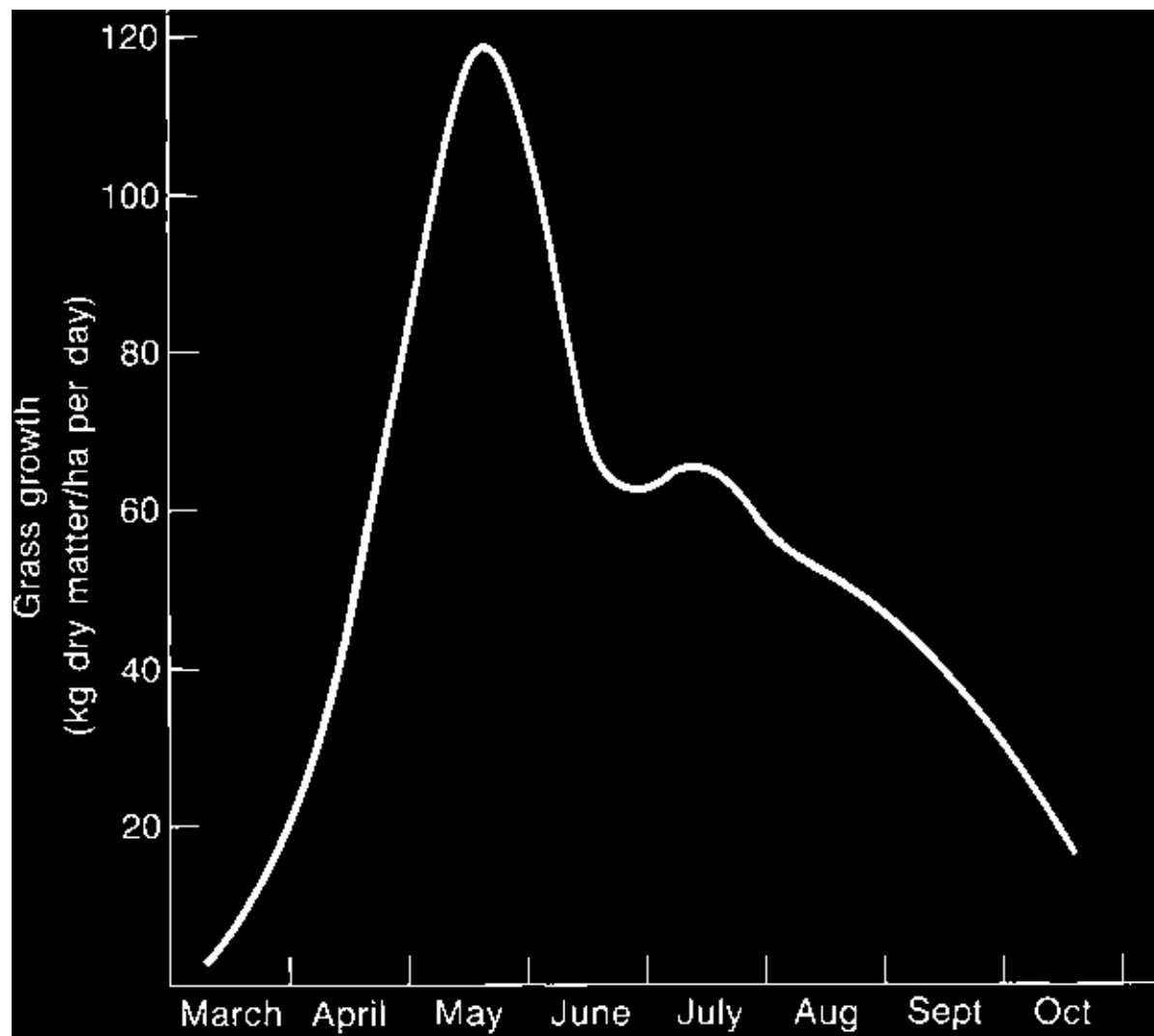
to be in full production the following spring. A

response up to 400 kg/ha, but the average nitrogen

spring reseed in April–May will lose much of the first
use is still under 200 kg/ha (Fig. 4.10). Researchers
year’s production and can be vulnerable to summer
have defined a target nitrogen use as being the point
drought.

in the response curve where 10 kg of grass dry





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Fig. 4.12

Seasonal pattern of grass growth. Source: *Milk from Grass* (GRI and ICI).

Fig. 4.11

Target nitrogen on five site classes. Source: *Milk from Grass* (GRI and ICI).

matter are produced for each 1 kg of fertilizer

maximum area to be cut without affecting milk nitrogen. Nitrogen use is now controlled in many production. Maintenance plus 20–25 l/day of milk can areas.

easily be obtained from spring grass. For an autumn-
Figure 4.11 shows the target point for five site classes, calving cow, as grass declines so milk yield declines and class 1 being the most suited to grass growth and class there should be no need to introduce supplementary 5 the least suitable.

feeding. Spring-calving cows may need supplementary feeding to sustain yields in the summer. This may be concentrates or forage crops such as rape or stubble

Grassland utilization

turnips.

Grass growth is uneven through the year with a peak in
Efficient conservation is a vital part of grassland the spring and then falling away through the growing management. It both provides a feed for winter use by season (Fig. 4.12). Grass growth will also vary greatly the cows and aids grassland management by utilizing

between seasons depending mainly on the summer grass surplus to the grazing requirement. The main rainfall. Nor are the animal requirements even over the options for conservation are either hay or silage. There season. For all these reasons any system of grassland is a continuing move away from hay towards silage on utilization must be flexible and adaptable to cover any dairy farms because:

situation.

- it is more flexible and integrates better with grazing;

The overall aim is to provide the cow with her

- silage is a better feed for dairy cows;

maintenance requirement and as much of her produc-

- silage can be made in poorer (not poor) weather
- tion as possible for each day of the year. This will be conditions; and

achieved with an integrated grazing and cutting system

- silage can respond to higher levels of fertilizer

whereby in early season about one-third of the grass nitrogen use.

area is grazed and two-thirds is cut. This is reversed in

mid season and then the whole farm is grazed in the autumn. However, silage making does require a higher capital investment in both machinery and storage areas (Fig. 4.13).

The most common systems of grazing management are either continuous grazing or rotational grazing with the use of contractors and with the advent of big bale paddocks or strip grazing. There is little to choose between systems in terms of output so choice is down to personal preference and ease of management. These have given even the smallest dairy farm the opportunity to improve grassland utilization and profitability by changing to silage making.

Under any system, cows should be stocked at a heavy density (6–8/ha) in the spring, which will allow a reference must be made to other forage crops that may have



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Fig. 4.13

Silage making with a precision
chop forage harvester.

a place on dairy farms by producing heavier yields

Kale

than grass or producing feed in a season when grass is

This is a high protein green feed grown mainly for

not available. The most popular forage crops are

grazing in the autumn and early winter. It produces a

maize, fodder beet, kale, lucerne, and stubble turnips

high yield of succulent feed that stimulates milk pro-

and rape.

duction. It can be cut and fed to dairy cows in the yards but is more commonly grazed, which means that it

Maize

should be grown in dry fields with good access. Mud, dirty cows and bad feet can be a problem. However, This is increasing in popularity in southern England and some have successfully made silage.

is normally made into silage but can be fed green. To be successful it requires high summer temperatures so even in southern England a sheltered field should be

Lucerne

used. It produces a similar or heavier yield per hectare than grass and has the advantage of being harvested in This is a drought-resistant legume that grows well on one cut. The resultant silage is a very palatable, high light chalky soils. Being a legume it fixes atmospheric energy, low protein feed that improves a winter ration. nitrogen and hence is cheap to grow once established.

It is often mixed with grass silage in the proportion

Cut four or five times during the season it provides

1 : 1 to 1 : 4 maize: grass but can successfully be fed on

good yields of high protein silage, but it can be difficult its own.

to get a good fermentation in the silage. A useful crop for organic farms with suitable soils.

Fodder beet

Stubble turnips and rape

This has about the highest yield of dry matter per unit area of any fodder crop. It can be grown successfully Although these are not the same they are used for the anywhere but prefers a heavier soil. It is an arable crop same purpose – to provide a succulent green feed either and the farmer needs arable techniques to grow it in the summer or autumn. For summer use they need successfully, especially for establishment, weed control sowing in the spring but autumn grazing can be and harvesting. It is harvested in October/November achieved by drilling after harvesting an early cereal and stored for winter use. It provides a palatable high crop. They both provide grazing when feed might energy feed, which helps butterfat percentages. otherwise be short.



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Fig. 4.14

Self-feeding silage.

Winter feeding systems

silage quality is even throughout the clamp, that the

face is not more than 2 m high and that there is enough

A winter feeding system will be partially dictated by the

space at the face, 24 hour access of at least 40 cm/cow.

objectives, high or low milk yields, and by the facilities

To avoid secondary fermentation the silage face

available.

needs to move back by at least 150 cm/day. Self-feeding

In all cases the first requirement is to know the quan-

is labour saving and requires no machinery. It is better

tity of bulk feeds available. Over a 200-day winter a

but not essential to have a roof over the silo and intakes
Friesian cow may well consume about 3.5 t of dry
will improve if a light is left on at night. Keep cows back
matter. If there are 2.2 t of silage dry matter per cow
from the silage face with a barrier, either a solid rail or
then this will leave 1.3 t to be made up with concentrates
an electric wire. The latter, however, can often reduce
or other purchased feed. Provided there are about 1.8 t
intake. To prevent waste the face must be kept tidy and
of forage dry matter for a 200-day winter then a rea-
waste silage removed.

sonable feeding system can be devised. This equates to
Mechanical feeding of silage will either involve
about 7 t/cow of fresh weight silage. Anything much less
putting the silage in a trough or behind a barrier with
than this will create problems, not so much from the
a tractor and fore-loader/blockcutter or else loading the
cow's point of view but from the economic side as it will
silage into a forage wagon that will spread it behind the
tend to be an unprofitable winter. Anything more than
barrier. Many people claim that this will increase intake

this will be a bonus and an asset to the farm especially compared with self-feeding by 10 per cent or more. Two if of a high D value.

simple barrier designs are shown in Fig. 4.15.

All feed changes should be done gradually to prevent

For 24-hour access the herd will need a minimum

digestive upsets. Thus silage should be gradually intro-

feed space of 40 cm/cow, but space of up to 75 cm is now

duced on a limited access basis from calving or by mid-

often recommended for best intakes. Again cleanliness

September, whichever is the earlier. The herd should be

is important and waste silage should be removed on a

housed at night by some time in October depending on

regular basis, preferably at least once daily. It is impor-

conditions and then housed continually from mid-

tant to ensure that silage is always available *ad libitum*.

October onwards, again depending on weather condi-

Leaving the trough empty for two to three hours before

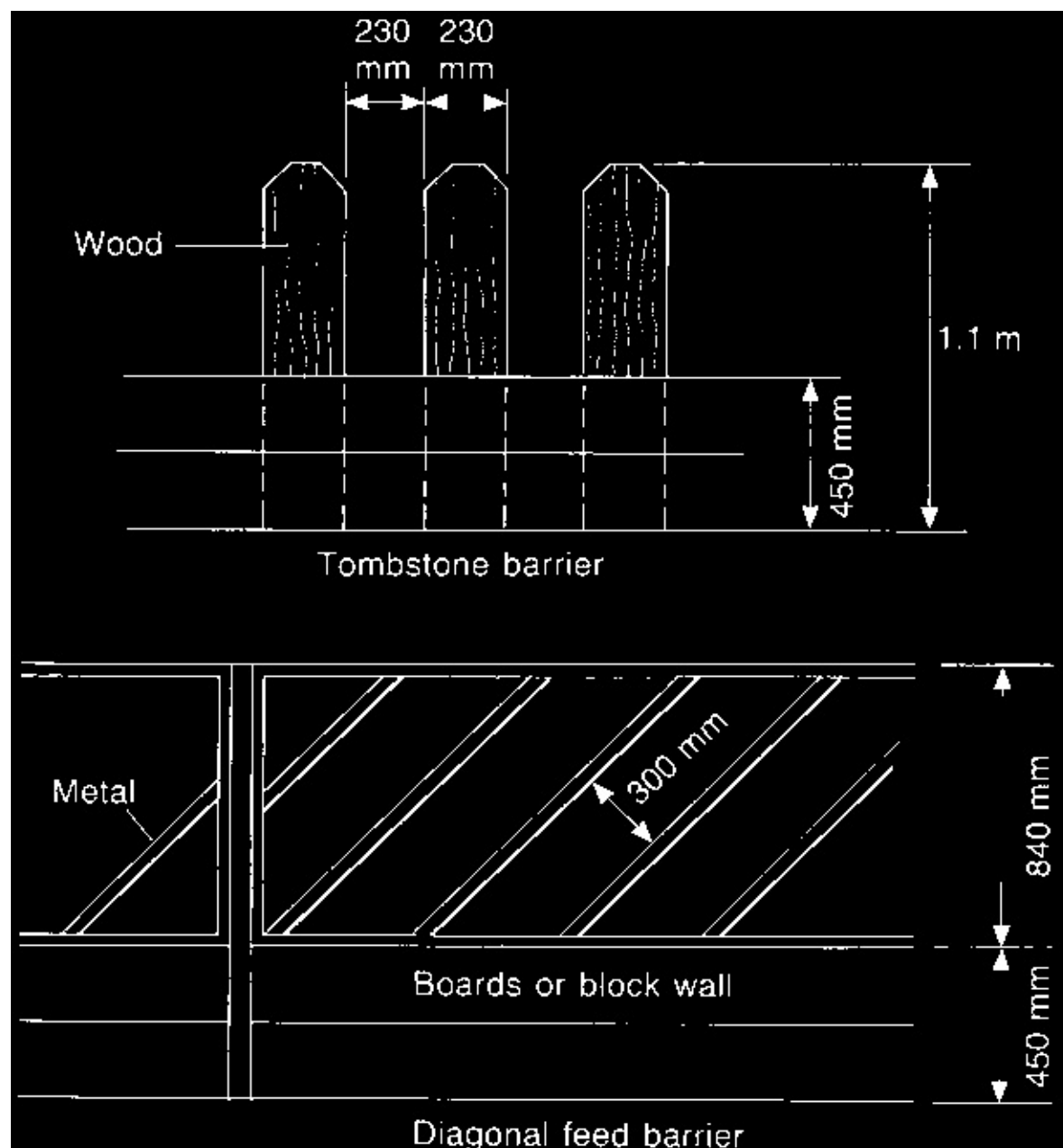
tions. Over this period silage consumption will increase

feeding will suppress intake and can cause digestive

and other feeds can be gradually introduced. The same

problems. It is a tremendous advantage to a winter principle applies in the spring, when ideally the herd feeding system to have a trough or barrier available as should be kept inside at night on silage for seven to ten almost any feed can be fed behind it, from silage to hay, days after turning out with grazing time gradually roots, brewers grains, sugar beet pulp or concentrates. increased.

This gives a great flexibility. When feeds such as Silage will either be self-fed (Fig. 4.14) or fed dried beet pulp or concentrates are fed it is essential to mechanically behind a barrier or in a trough. Self- have at least 60 cm/cow of trough space to ensure a re-feeding works well for low yields, provided that the sonably even intake among cows.





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Fig. 4.16

Cows at a computerized out-of-parlour feed station.

Fig. 4.15

Two types of feed barrier.

A sophistication of this system is complete diet with alternative feeds such as cereals, maize gluten or feeding or total mixed rations (TMR). Here all the sugar beet pulp.

feeds are placed in a mixer wagon, which mixes them

A sophistication of this system is with computerized together and delivers the diet behind a feeding barrier.

out-of-parlour feeders (Fig. 4.16). Each cow has a

Because of the degree of mixing the cow is unable to

transponder identifying her to a feeder station. This is separate out, say, barley from silage and so eats the then programmed to allow a specified amount of feed whole mixture uniformly. Ideally the wagon has weigh per day in a number of feeds. The cow can then eat up cells so giving a very accurate measurement of feeds. to her allowance but no more. Although the capital cost Use of a mixer wagon allows the feeding of a wide range of these installations is high they make for very accurate feeds that may represent a very economical buy, rate feeding, based on the sound principle of 'little and but which are normally either difficult to feed or often'. They perhaps demonstrate the way dairying will unpalatable, e.g. molasses and rapeseed meal. It also go in the future. Printouts can be obtained for cows not allows much more satisfactory feed digestion within the consuming their allocated amounts.

rumen and further down the alimentary tract. Some In all feeding systems water must not be overlooked. farmers are now buying straights and producing their With milk being 86 per cent water any shortage of water

own complete diets, usually with the help of a nutritionist. Attention is being focused on straight feeds and need 60–100 kg of water per day and being creatures of habit tend to all want to drink at the same time, especially late afternoon. This makes a large reservoir and proof. The introduction of herd health and quality fast flow of water essential. The volume drunk will assurance schemes has drawn attention to how feeds increase as milk yield increases and in dry hot weather, are stored. On some farms forage boxes are used so that 120 litres or more is not uncommon.

instead of mixer wagons, but these do not really mix up the feed.

Most milking parlours have facilities to feed concentrates

Dairy farm buildings

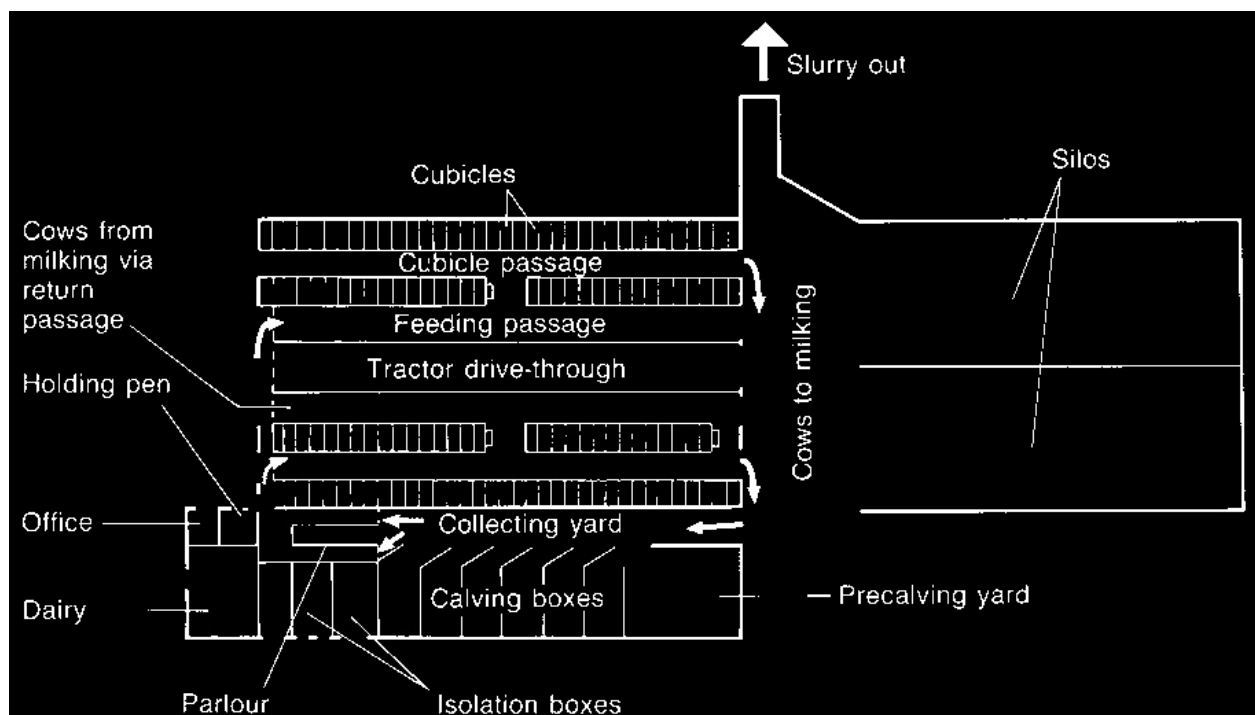
and it is usual to feed these twice per day at

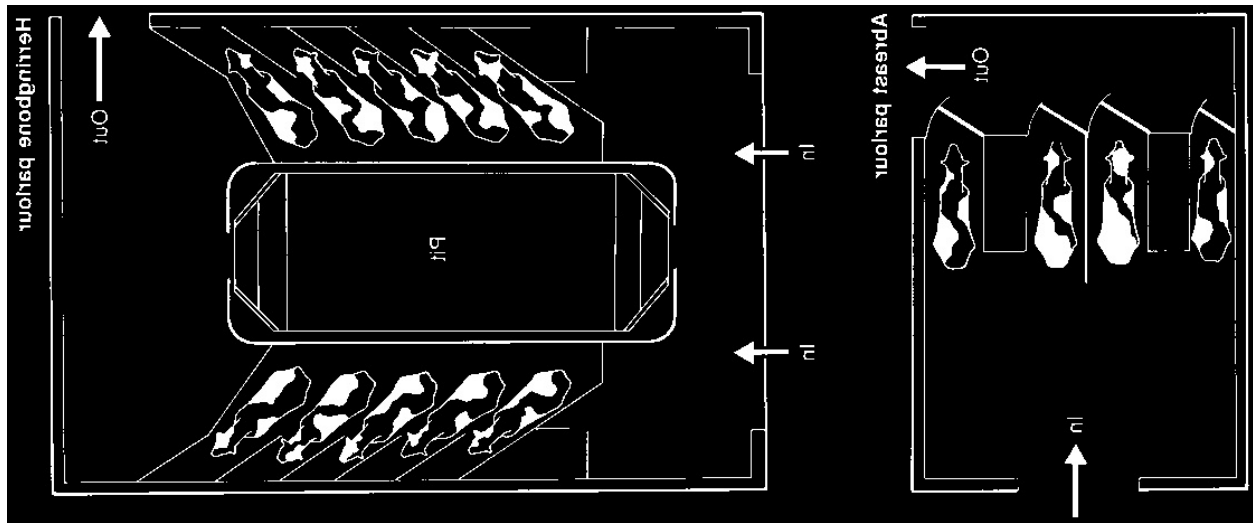
milking time. A limit to be fed during each milking

Because of the diverse nature of dairy farming there are

would be 4–5 kg. If anything above this is needed then almost as many different designs of dairy building units as there are dairy farmers. an out-of-parlour feed at the barrier is beneficial. This

not only splits the feeds, which helps digestion, but also However, if starting from scratch the main needs are allows a proportion of the concentrate to be replaced for a milking parlour and dairy, winter housing and





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Fig. 4.17

Typical layout of a dairy unit.

feeding barn, food storage area, calving and isolation boxes, slurry handling area and various collecting yards and access concrete. A typical set of dairy buildings is shown in Fig. 4.17.

Important considerations when designing a dairy unit include the following.

- Cow-free access for the milk tanker so that collection can be made at any time.
- Good cow flow so that cows come from the bedded area through the parlour and back with a minimum of narrow passages and sharp corners.

- Good feeding access so that cows can be fed without having to work amongst them.
- Access to silage storage areas away from the cows so that silage making can take place while the cows are in the yards or in for milking.
- Good handling arrangements for veterinary treatment and AI.
- Adequate slurry storage as spreading may be restricted in nitrogen vulnerable zones.
- Thought should be given to the way the buildings blend in with the surroundings. Choice of materials and site are important.
- Adequate loose boxes for calving, sick animals, etc.

Parlour and dairy

This is the key to the whole dairy unit, being used twice (or three times) a day 365 days of the year. Cowsheds are very labour intensive and outdated while rotary parlours in the past have proved unreliable, with no benefits over a static parlour but are now becoming more common again. Automatic (robotic) parlours are also now being introduced. Figure 4.18 shows the most common par-

lours used, an abreast and a herringbone parlour.

Fig. 4.18

An abreast and a herringbone parlour.



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The most simple parlour is the abreast, which is particularly suitable for herds of up to 80 cows with one man milking either with three milking units and six standings or six units and six standings. The cows enter and leave one at a time so a degree of individual treatment for a slow milker or slow eater is possible. The parlour should have a loft above to hold the concentrates, which fall by gravity to the feeder situated

between each pair of cows. Milking rate will be about 30–40 cows/hour.

In a herringbone parlour, by far the most common, the milker stands in a pit, which removes most of the bending required. The cows come in and out of the parlour in batches and stand at an angle to the pit on either side. A herringbone parlour is suitable for any sized herd and one man could comfortably milk 100 cows in two hours with a 10 : 10 herringbone, i.e. 10 milking units, 10 standings. For 300 cows or more larger herringbones with two or three milkers are common. A loft above the parlour will hold the concentrates and each standing will have a feeder. In some parlours feeding has been discontinued.

Automatic cluster removers (ACRs) are now available that make it possible for one man to handle more machines without the danger of overmilking.

Fig. 4.19

A feed/sleep arrangement with metal cubicles.

In any parlour the work routine is to let the cow or group of cows in, feed them, dry wipe the udder or wash

and dry if dirty, wash the udder, test the foremilk, attach (3 tonnes per cow), which creates work to cart in and the cluster, remove the cluster, teat dip or spray, transfer the milk and let the cows out. Pre-milking teat dipping is practised in some herds, especially those with high levels of environmental mastitis. Cubicles (see p. 422) have gained in popularity and, well designed, can be a very trouble-free system with a low demand on labour (Fig. 4.19). The main requirement for comfort is to make the cubicle large enough, contain some dairy equipment such as the vacuum reg-ulator, milk pump, water heater and washing equipment. The bulk milk tank will also be situated here. The surface. They can be made of metal or timber and a capacity of the bulk tank is important and must be sufficient to hold the likely peak milk production. Except

The dairy should be adjacent to the parlour and will about 1.2 m wide and 2.2 m long, with a good lying ment. The bulk milk tank will also be situated here. The surface. They can be made of metal or timber and a capacity of the bulk tank is important and must be sufficient to hold the likely peak milk production. Except

Friesian cows and does not offer sufficient flexibility for in a herd with a very compact calving pattern about many high-yielding cows and so ones such as in Figs 30–35 litres/cow capacity is normally sufficient.

4.21 and 4.22 are used. Other new models are also available. Both the parlour and dairy must be kept scrupulously clean and in Britain will be inspected regularly by the Table 4.13.

authorities. The motors and refrigeration unit should The cubicle should slope from the head to tail end be housed separately in an engine house. It is also and the best surface is concrete with mattresses, mats useful to have a small area near the parlour where or sand as a bed. A strategically placed headrail is the cowman can keep the records necessary for herd essential to ensure the cow dungs in the scraper passageway. management.

Passageways should be at least 2.4 m (8 ft) wide to allow for good cow flow and sufficient width for the scraper.

Cow housing and feeding barn (Chapter 56)

The whole cubicle and scraper passageway can either

The choice for cow housing will rest between housing
be situated within an open span building or be part of
in straw yards and cubicles.

a purpose-built cubicle building. For all situations it is

Cows often appear more comfortable in a straw yard

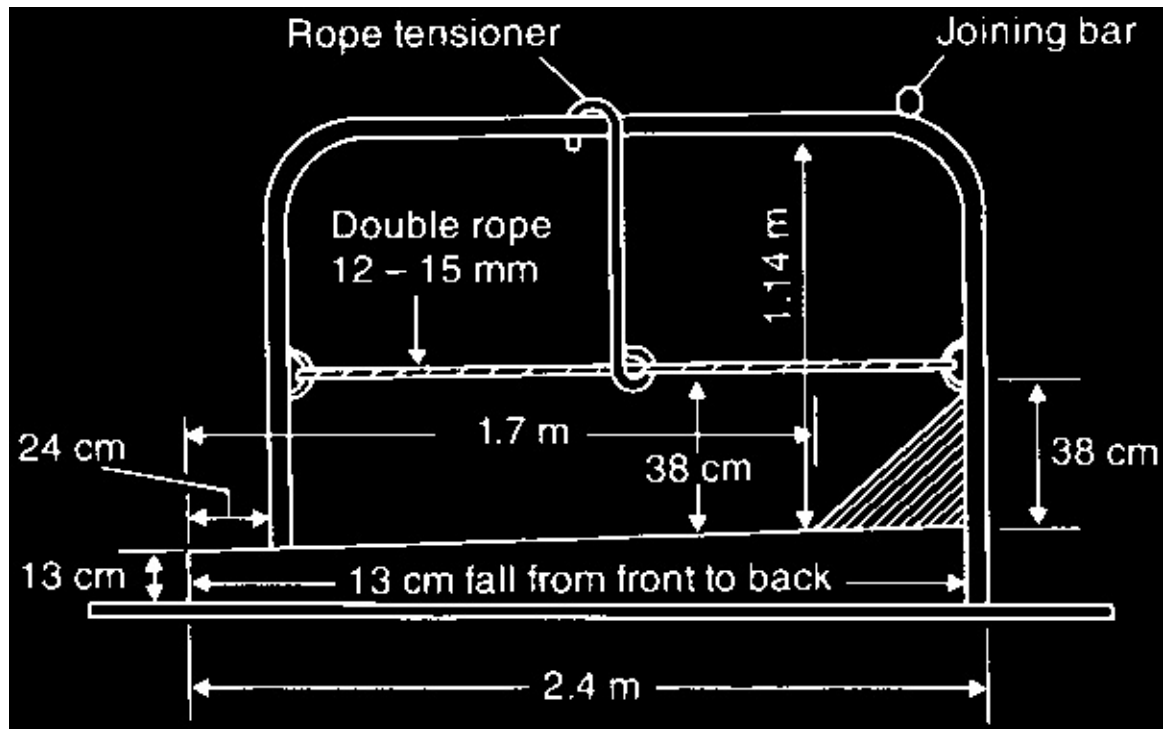
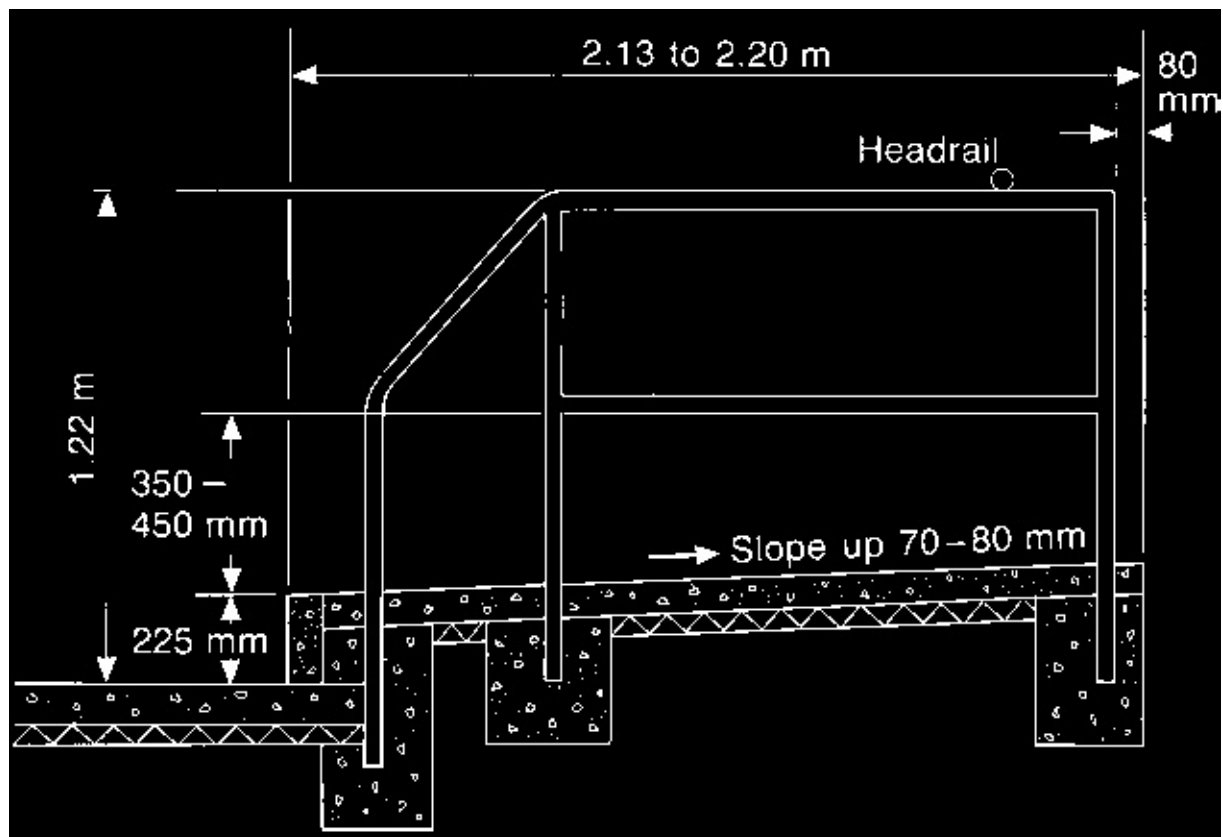
important to have a draught-free environment with

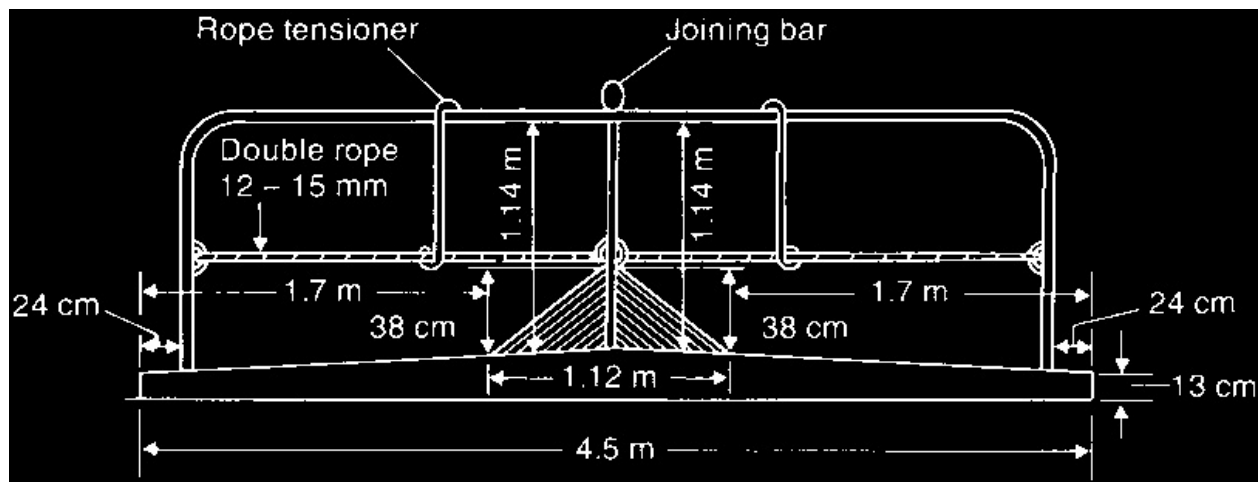
but if they are to keep clean they need about 6 m²/cow.

plenty of ventilation. This is normally achieved by the

They also use a lot of straw over a winter (about 2.5–

use of Yorkshire boarding (vertical boarding with gaps





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to allow free movement of air) or with an open ridge to the whole herd into groups, by milk yield or calving the building or kennel.

date, which can assist management.

With either straw yards or cubicle beds, the feeding

There has been increasing emphasis on cow comfort,

areas can be either inside or outside the building. An

particularly from the point of view of preventing lame-

inside area is preferable but will add to the cost. An

ness and providing suitable conditions to ensure that

example of an arrangement with a centre feed passage

cows lie down for similar lengths of time as they do when

is shown in Fig. 4.23.

at grass. This is difficult or impossible to achieve with

The advantages of this type of arrangement are that cubicles and so some farms have altered accommodation the cows can be shut in the feed area while the cubicles to straw yards (see Figs 4.24, 4.25 and 4.26). Such alterations are scraped and vice versa. Feed can be put out without alterations do require sufficient space for each cow (see Table 4.14). They can result in rapid improvement of locomotion scores, and considerably reduce lameness due to white line disease and solar ulceration (Hughes, 2000). However, mastitis levels can rapidly increase, particularly due to *Strep. uberis* and *E. coli*, unless great care is taken in producing a satisfactory straw bed. The keys to success include making available a large quantity of straw for each cow (about 2.5–3 tonnes) and ensuring that it is kept dry when stored and the bed remains dry. Regular cleaning out every 4–6 weeks may help prevent mastitis problems, but this is possibly less important than the quality of the straw used. Good ventilation is also required to help keep the straw bed dry.

Feed storage area

The major item here is the silage storage facility. This can range from a tower silo to a simple hardcore pad.

Fig. 4.20

A metal cubicle division.

Construction of a simple silo is worthwhile to make handling easier and to prevent waste. Clamp silos are the most common and are usually constructed with a concrete base and walls of earth, sleepers or concrete. Safety is essential and in Britain walls must be up to DEFRA (Department for the Environment, Food and Rural Affairs) standards bearing in mind the walls have to contain not only the weight of the silage but also pressure from the tractor when consolidating the clamp. For open silos, guide rails must be provided 900 mm above the wall and for roofed silos sufficient height (5.5 m) must be allowed for tractor clearance. All effluent must be contained in a sealed tank that can be pumped out. For self-feeding of silage the height of clamp must not

Fig. 4.21

A cubicle suitable for a Holstein cow (courtesy of
be more than 2 m and capacity will need to be about

J. Hughes).

12–15 m³ per cow for a winter's storage. If other feeds

Fig. 4.22

A double fronted cubicle suitable for

Holstein cows (courtesy of J. Hughes).

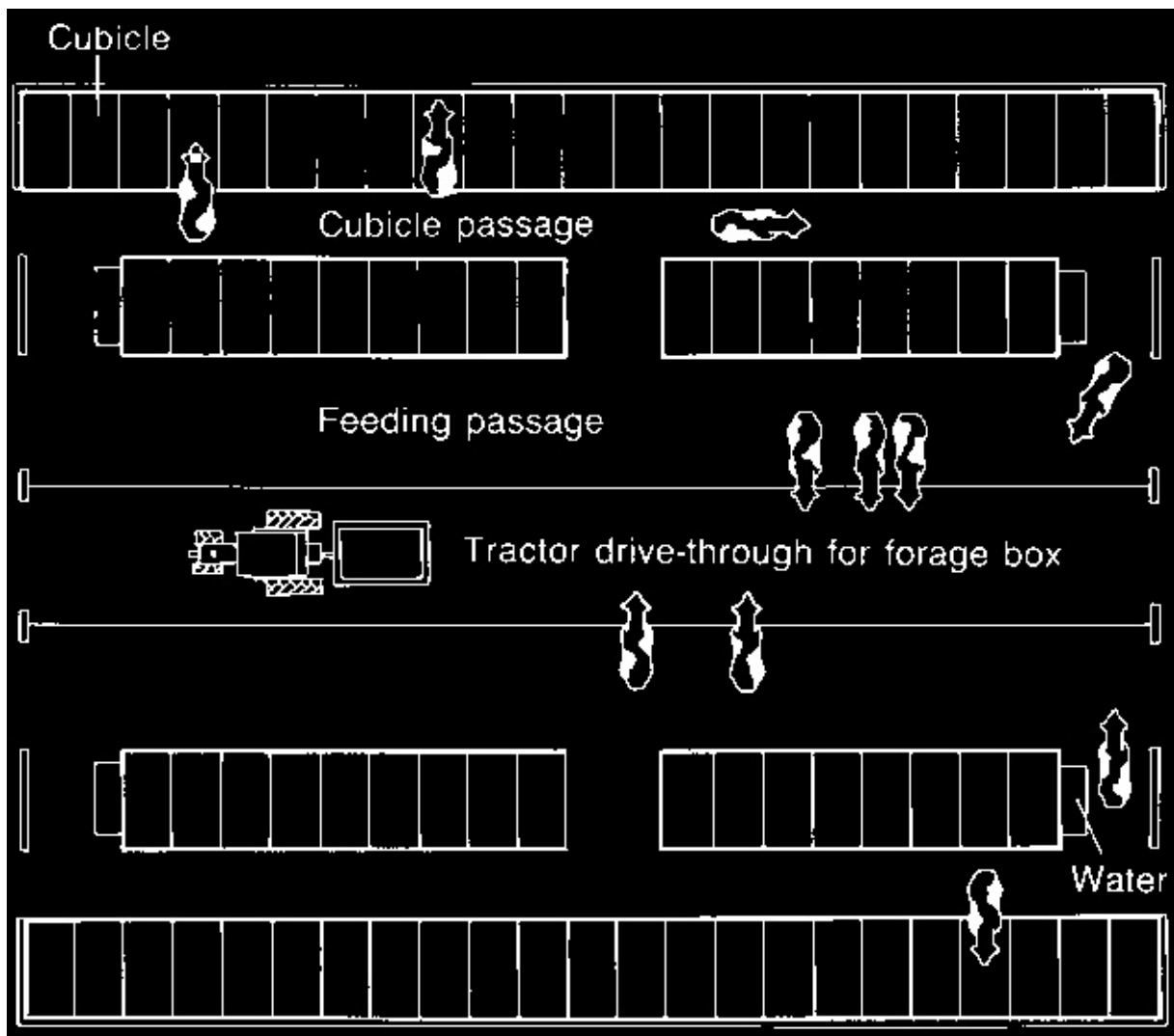


Table 4.13

Measurements of cubicles suitable for high yielding Holstein cattle as determined in Britain (adapted from Hughes, 2000).

Measurement

Metric (Imperial)

Length

2.4 m (8 ft)

If double fronted cubicles with concrete fillet so maximum forward lungeing space length can be reduced to 2.28 m (7 ft 6 inches) Width

1.2 m (4 ft)

If width 1.14 m (3 ft 9 in) there must be a flexible lower barrier Rear step

<150 mm (6 inches or less)

Fall (front to rear)

100–125 mm (4–5 inches)

Division height

1.14 m (3 ft 9 inches)

Lower division rail (flexible)

400 mm (1 ft 4 inches)

Barrier thickness ideally flexible – at least

19 mm (3/4 inch)

Brisket board

100 mm (4 inches)

Brisket board from front

0.75 m (2 ft 6 inches)

Brisket board from rear

1.7 m (5 ft 8 inches)

Sloped concrete fillet (instead of brisket board) height

38 cm (1 ft 3 inches)

Concrete fillet from heel stone

1.7 m (5 ft 8 inches)

Barrier (above fillet) if head to head cubicles

76 cm (2 ft 6 inches)

Head rail (below average wither height) 1/5 cubicle length from front 150–250 mm (6–10 inches)

cows can be restrained for veterinary treatment or insemination. They require a separate air space.

Boxes should be reasonably spacious and must have a tying-up ring or some other method to aid cow restraint. Walls must be rendered so that disinfection can take place. It is also useful to have a water bowl and feed rack, tractor access for cleaning out and, maybe, a milking point so that cows can be milked in the box.

Sick cows may die or have to be destroyed in the boxes so good access for the slaughterer's lorry is essential.

For a herd with spread calving pattern, one box for every 40–50 cows should suffice. Placing hinges and a swinging gate against a wall with the fixed end about 0.5 m (2 ft) from a corner is a useful method of restraint. It allows the gate to be swung round to restrain a cow between the gate and the wall.

Fig. 4.23

Sleep and feed arrangements.

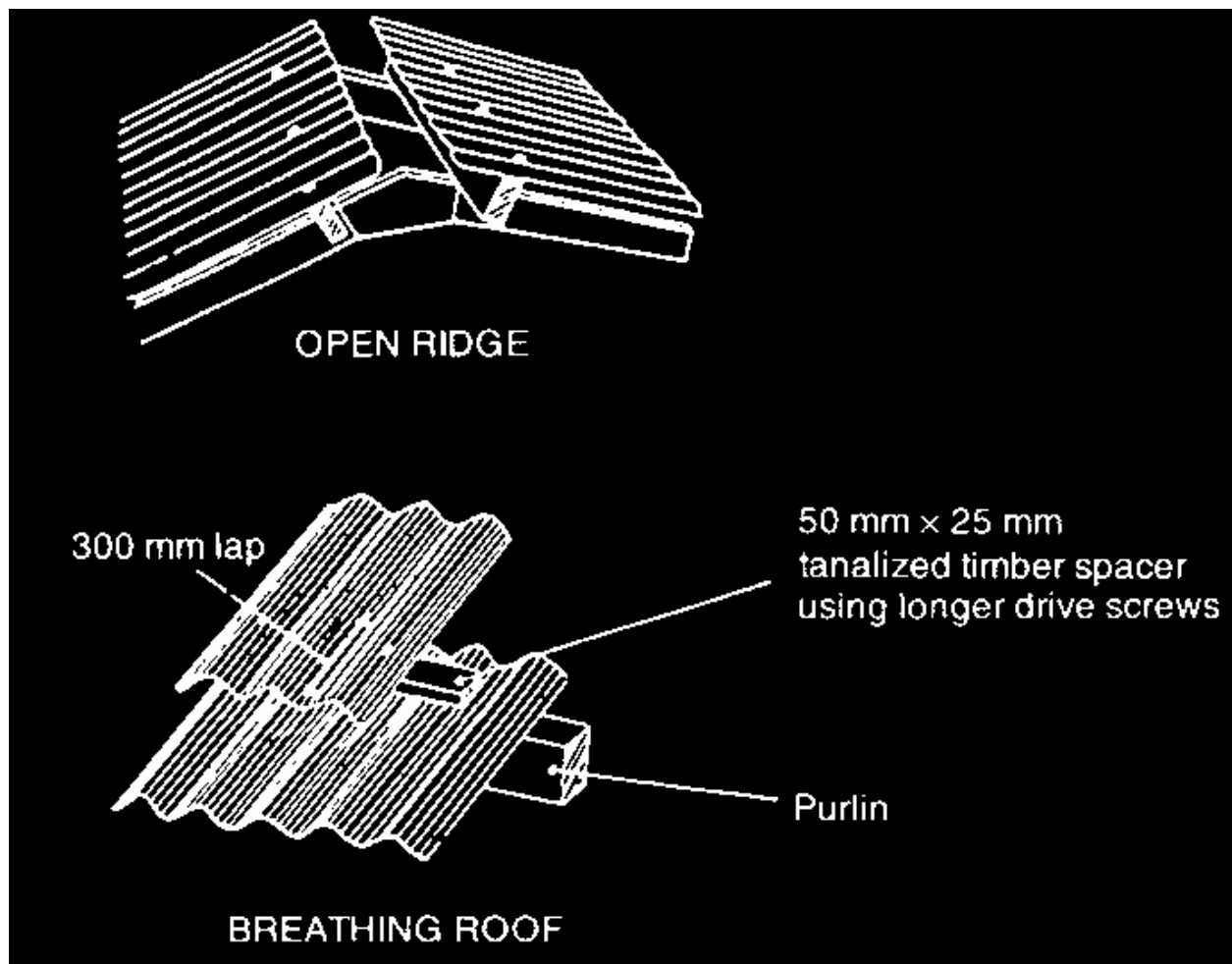
Slurry

With more and more farmers using a loose housing and cubicle system, disposal of slurry is becoming an increasing problem. Also, water authorities are rightly demanding more control of pollution. A dairy cow will produce about 7000–8000 l (7–8 m³) of slurry over a year and dry.

winter. This has quite a high fertilizer value (10 000 l will contain 25 kg N, 10 kg P₂O₅, 45 kg K₂O) but much of this will be lost by leaching if it is spread during the winter

Calving and isolation boxes

when the soil is saturated. Also, the pollution risk is
These need siting in a convenient position to allow for
much increased. In the UK there are laws concerning
ease of entry and exit for cows. The area would
application in nitrogen vulnerable zones (NVZs).
also probably include a cow handling area where
Therefore, winter storage becomes almost essential.



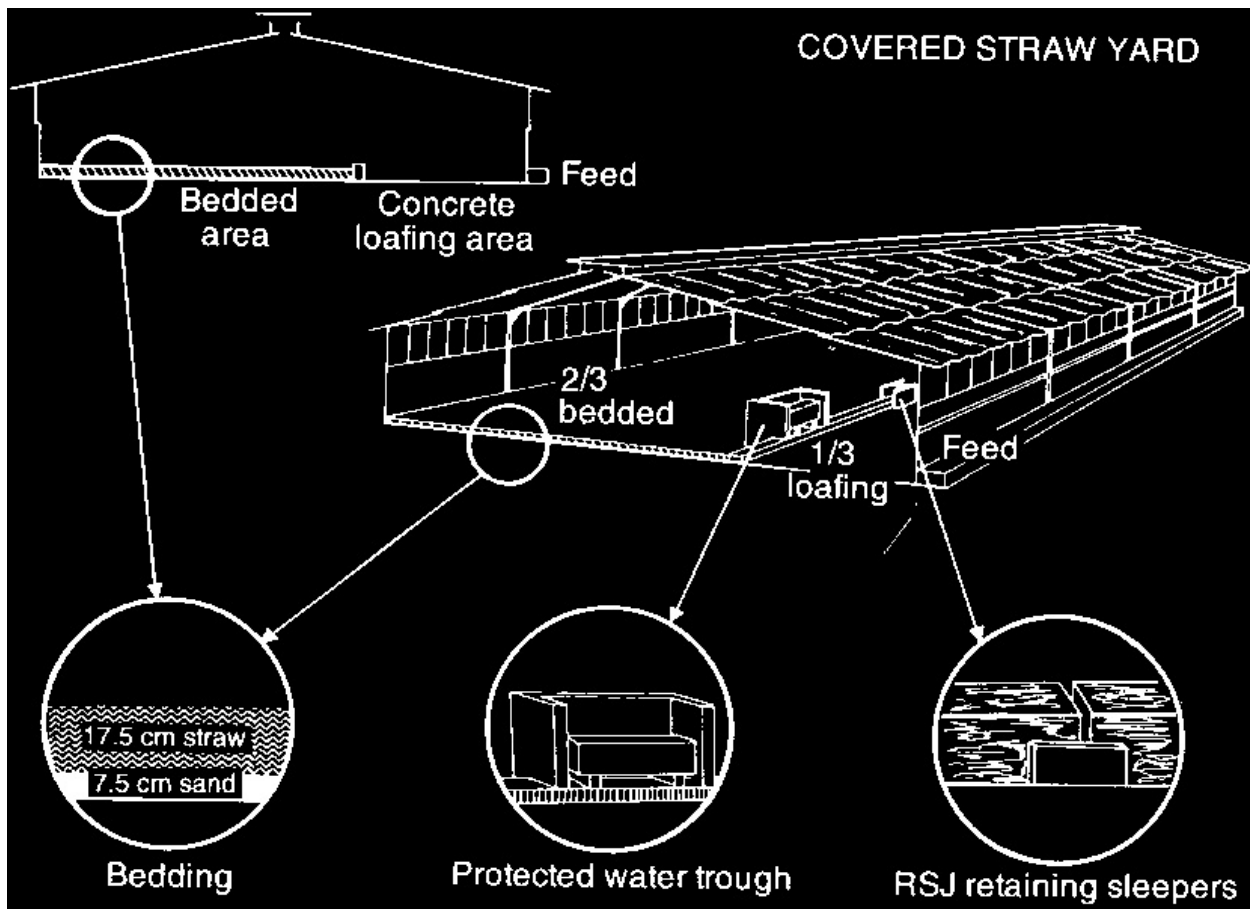
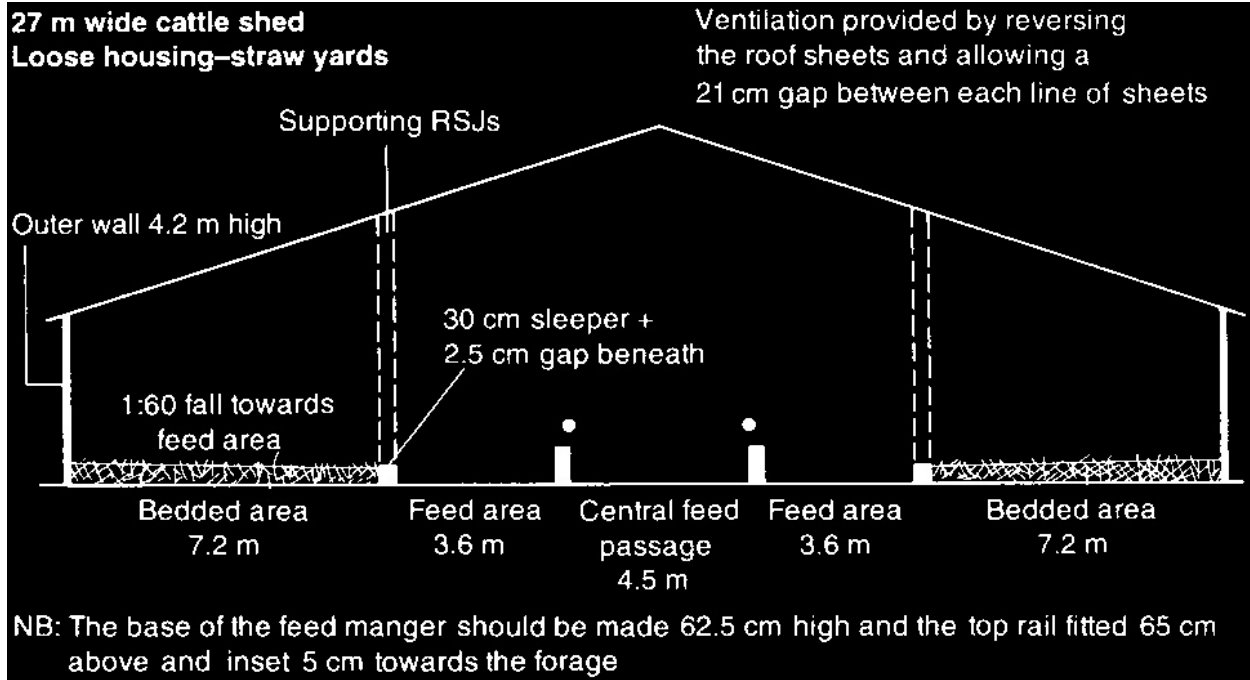


Fig. 4.24

A 27.5 m (90 ft) wide
cattle shed with loose housing
(reprinted from Hughes, 2000).

Fig. 4.25

Structures within a covered straw
yard (reprinted from Hughes, 2000).

Slurry is usually scraped with a tractor-mounted
squeegee to the store. The store must be able to cope
with slurry, water, waste feed and bedding. It will reduce
the volume considerably if clean water from roofs is
diverted to a separate soak-away or drain.

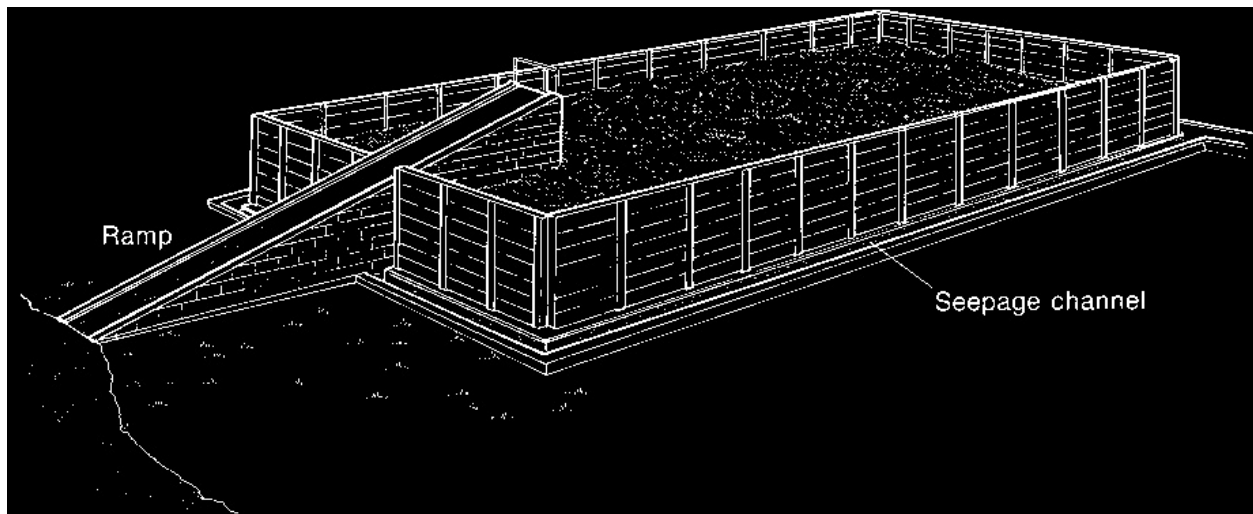
Long-term storage can be provided in a sealed tower
or a storage compound. With a slurry tower, the slurry
is scraped into a reception pit and then pumped into the
sealed tower. This is kept agitated to prevent settling or
crusting of material. For emptying, the slurry runs back
by gravity to the reception pit and is then pumped into
the slurry spreader. It provides a good pollution-free
system but is expensive to install and can create prob-
lems if management is not good leading to crusting and

blockage of pumps.

By contrast the storage compound, although not cheap to construct, provides a simple-to-manage system

Fig. 4.26

Ventilation for a covered straw yard (reprinted from with very few problems. The compound should be large Hughes, 2000).



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enough to hold a winter's slurry, have a concrete or hard sleepers (Fig. 4.27). This allows water to drain from the base to facilitate emptying and a shallow ramp to push slurry, which is collected and spread separately. This the slurry up for filling. Slurry is pushed in all winter considerably reduces the volume of material needing to

and then at a suitable time emptying takes place with a be stored for the winter and improves the handling of tractor and fore-loader direct into spreaders. A sophis- the remaining material.

tication is a 'weepy wall' silo with walls constructed of timber or concrete sleepers with gaps between the

Herd records

Herd records are essential for management of the herd

Table 4.14

Space allowances for straw yards suitable for high but they must be simple to keep, understood by the yielding Holstein cattle as determined in Britain (adapted from Hughes, 2000).

farmer and his staff and of use in the herd management.

Before records can be kept, cows must be individually

Space allocation

Metric

identified. With herds becoming larger and to make identifying cows by relief staff possible, this identifica-

Strawed area

tion must be permanent and clear.

Freshly calved cow

6.5 m² (70 ft²)

Freeze branding with liquid nitrogen kills the pig-

Mid lactation cow

5.6 m² (60 ft²)

ment cells in hair and results in a clear white number

Dry cow

4.6 m² (50 ft²)

on black cows but is not so successful with light

Plus

coloured cows. Ear tags are also often used and can be

very clear if large. However, there is always the possi-

Loafing area

bility of losing tags. With the coming of new technology

All production stages minimum

2.3 m² (25 ft²)

Thus total minimum

it is possible that most cow identification in the future

Freshly calved cow

8.8 m² (95 ft²)

will be by transponder, either implanted into the cow

Mid lactation cow

7.9 m² (85 ft²)

or worn on a collar or ear tag or placed in the rumen.

Dry cow

6.9 m² (75 ft²)

For short-term cow identification, e.g. for a cow needing treatment, etc., spray cans of different coloured

Optimum number per pen = 40 cows

paints or tail tape, sticky coloured tape, can be success-

Area per pen of 40 cows

fully used.

Freshly calved cows

350 m² (450 yd²)

Computers are now used for keeping records for

Mid lactation cows

320 m² (380 yd²)

most large herds, but this is by no means essential and

Dry cow

275 m² (335 yd²)

the capital cost will result in a place for manual record-

- Maximum per pen with sufficient space = 60

ing in small herds for many years to come. Records kept

- Drainage in feeding area = 1 in 60

will be either physical or financial. In many countries

- Straw usage = 2.5t per cow

treatment records must also be kept.

- Feeding passage never less than 3.7m (12ft) wide

- Easy access from bedded area

Physical records

- Water – recess into bedded area but no access from bed

- Ventilation must allow moisture to escape – roof open

The notebook: The herdsman or farmer should always

ridged, possibly breathing or gap roof

have a notebook in his pocket to record information

Fig. 4.27

A weeping-walled slurry

compound.

Cow record card									
Cow name _____		Pedigree number _____			Ear tag number _____				
Sire _____		Date of birth _____							
Dam _____									
Lactation									
1	Calving date	Sex of calf	Sire	Milk yield	Days	Fat %	Protein %	Lactose %	Date dry
	Service dates				Veterinary treatment				
2	Calving date	Sex of calf	Sire	Milk yield	Days	Fat %	Protein %	Lactose %	Date dry
	Service dates				Veterinary treatment				
3	Calving date	Sex of calf	Sire	Milk yield	Days	Fat %	Protein %	Lactose %	Date dry
	Service dates				Veterinary treatment				

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Fig. 4.28

Cow record card.

such as bulling cows when the event is observed, before successfully maintained with written entries on a card transferring this to more permanent records.

but often a more visual record is kept. This is usually a rotary board with pins or magnetic blocks of different

Cow record cards: This will be the cow's 'log book' and colours used to identify cows. These are placed on the

will give a concise picture of each cow's history. It must board to record events so any cow 'out of line' is quickly contain basic details such as name, pedigree number, obvious and can be investigated. If this method is used it ear tag number, herd number, date of birth, service is important to have a more permanent back-up in case dates, calving dates, sex and sire of calf data, production the pins are accidentally removed. Figure 4.29 shows details, health records and details of veterinary treatment. An example is shown in Fig. 4.28. Much of this systems are now available and they are being increasingly used in larger herds. These will usually allow the each animal also has a Cattle Passport.

creation of various action lists such as those cows to separate out for routine veterinary examination, those due to come on heat that week, those due to be served, those

Breeding records

due to be dried off, those due to calve, etc.

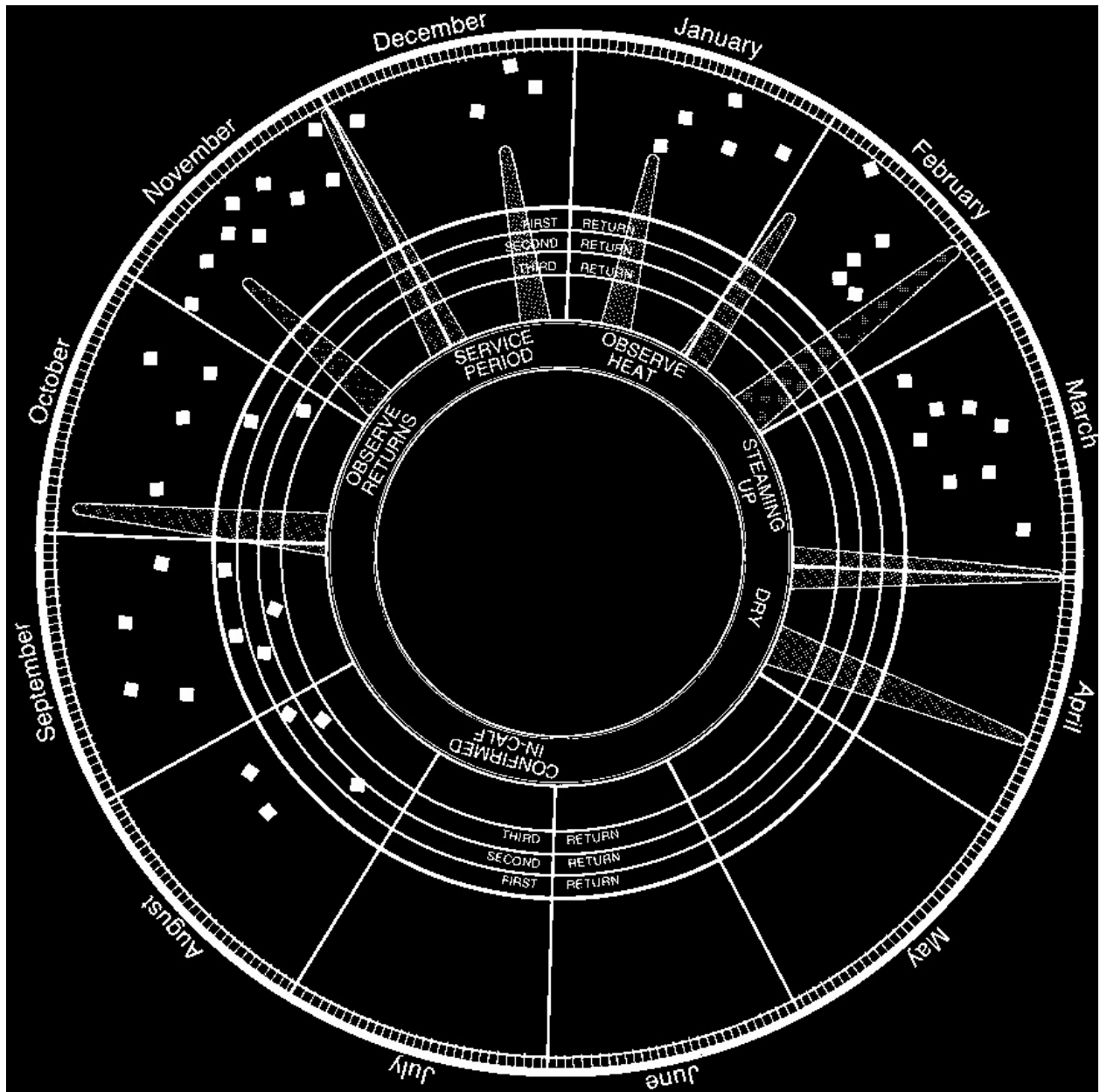
Apart from information kept on the cow record card a simple breeding record should be kept. This will be used

Milk records

to keep dates of calving, pre-service oestrus, services and identification of sire and expected calving dates. These

Whilst it is possible to record milk yields from cows records should allow the farmer or herdsman to quickly personally, most farmers who wish to record milk use identify any problem cows, such as those not cycling or

National Milk Records. The scheme supplies a milk not holding to service, so that treatment can be carried recorder to measure the milk and take a sample, which out before the calving interval slips. These records can be is then sent to a laboratory for testing for the percent-





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Fig. 4.29

Breeding board.

Fig. 4.30

The milk recorder sampling milk
in the parlour.

age of butterfat, protein and lactose and cell count. The commencement of lactation. At the end of the lactation recorder also enters dates of calving, service, drying off a cow record card is produced. From the dates provided and culling (Fig. 4.30). The results are sent back to the an action list is produced for the next month with lists farmer within a week and contain records not only for

of cows due to calve, to dry off, to serve or to pregnancy that month but also on a cumulative basis from the diagnose.

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Milk records are used for pedigree purposes, to

A herdsman will be one of the most skilled workers provide information to prepare Improved Contempo- employed on the farm. He must enjoy working with rary Comparisons (ICC) for bulls and to assist with cow cows and be prepared to work the long hours made nec- selection for breeding purposes.

essary by milking twice a day, seven days a week. He

Apart from this, milk records can be used for man- must be observant to see signs of unhealthy cows, to see agement purposes for individually rationing cows or for cows in oestrus or about to calve and to see signs of use in a milk prediction scheme.

under- or overfeeding. He must be capable of keeping records accurately.

Financial records

He must be clean, to produce high quality milk and

to prevent a dirty environment. He must be strong as Farmers keep many financial records with varying degrees of sophistication to assist with the management feeding and calving especially. Fortunately, the herdsman's job provides a high degree of job satisfaction and interest for a dedicated man.

vided by agricultural organizations or consultants. All Most herdsmen are paid a fixed wage for the job but of these measure the main output from the dairy herd, may also have bonus schemes related to such things as milk income, and relate this to the main variable cost of milk yield per cow (less common since quotas), margin production, namely feed. This then gives a margin over over feed costs, calving percentage or calves sold.

feed analysis, expressed on the basis of per herd, per A part from the main herdsman, relief will be needed hectare, per cow or per litre. For example, the gross for weekends, holidays and sickness. Often this relief

margin per litre in England and Wales in 1995–96 was 15.34 p/litre and 13.24 p/litre in 1997–98; and that per Failing this, relief agencies exist which can provide a cow was £897/cow in 1995–96 and £809/cow in 1997–98. herdsman often at short notice. At present numbers Margins in some of the next few years were considerably lower (e.g. £807 per cow and 14 pence per litre in November 2002, taking into account inflation). individual stock person.

These services are all computerized and rely on the farmer supplying information monthly on cow numbers, milk quantity and quality, feed used and price, fer-

Breeding

tilizer used and price, and land area used.

The aim of a breeding policy must be to get the cows in The farmer then has the results returned to him and calf regularly and to provide sufficient replacements of often has a league table comparing his results with

high genetic potential to maintain the herd size at the other farmers or comparing his results for this year with optimum level. This will depend on the culling rate, the previous year or budget.

the age at first calving, the breed of sire used and the Differences between the various schemes are small genetic worth of that sire.

but some form of financial monitoring provides an essential part of the dairy herd management.

Culling

Management

Studies have shown that the majority of culls are sold for reasons such as poor breeding, mastitis, bad feet, injury, etc. (Table 4.15). These might be termed

Labour

No dairy system will work efficiently without good

Table 4.15

Reasons for culling (%). Sources: Cull cow surveys labour. In many cases this is supplied by the farmer and 1984 – FMS Information Unit; Milk Development Council, 2000.

his family but for larger herds employing a herdsman is

common. In the milk costs survey for 1986–87, 63 per

1984

2000

cent of herds only used family labour. From the same survey there was an average labour use of 35 hours/cow

Poor yield/quality

12

10

per year. This ranged from 79 hours/cow for herds of

Poor breeder

21

28

below 30 cows to 27 hours/cow for those above 100

Bad legs or feet

9

7

cows. About half of this time was spent milking the cows

Mastitis/cell count

15

18

Udder

8

6

and the other half tending them. Labour costs will vary

Old age

10

5

depending on factors such as herd size, facilities

Injury

5

4

available and type of person employed but will range

Others

20

22

between £250 and £300/cow or about 4 pence per litre.

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unplanned reasons and will not lead to overall herd

With many first calf heifers still being bred to a beef
improvement.

bull this gives very limited scope to do other than breed

With good management it should be possible to keep

pure with the dairy cows especially if management is the annual culling rate to 20 per cent. But the national below average. However, where choice is available then average is about 25 per cent. Also, because so much cows with the highest cow genetic index (CGI) should of that culling is for the unplanned reasons outlined be used to breed replacements.

above, the rate of genetic progress is considerably As pre-sexed semen becomes more available and slowed. In some countries such as North America affordable it will become increasingly possible to pro-culling percentages tend to be higher, while in others duce all high genetic merit heifers from those cows such as New Zealand they are lower.

chosen to breed replacements. The remainder can then be bred to a beef bull and again it will be possible to choose the calf's sex. Otherwise embryo transfer can be

Age at first calving (see Chapter 5)

used to solve both these management problems.

This is very much linked with culling rate. Table 4.16 shows the effect of three culling rates and three differ-

Genetic worth of the sire

ent ages of first calving. The number of heifer replacements needed more than doubles between options.

The choice here will rest between using natural service

Although heifers need a higher plane of nutrition to

and AI. The chances of being able to use a bull of high

reach sufficient weight to calve at two years old their

genetic merit which was referred to as 'ICC' for natural

lifetime yield will exceed those calving at three years

service are very remote, so AI is to be recommended.

(Table 4.17).

Also, AI gives choice for traits other than production

such as ease of calving, conformation or size. Within the

overall traits desired, bulls with the highest ICCs for

Breed of sire used

weight of fat plus protein should be used. Also, it is

Most dairy farmers breed sufficient cows pure to get the

important to choose bulls with a reliable proof, i.e. with

desired number of replacements, the remainder being

a weighting of +40 or above, with daughters in over 20

put to a beef sire to maximize the value of the calf.

herds and with less than 25 per cent of daughters in the
Depending on the factors outlined above this can mean
two herds with most daughters. A wide selection of
between 40 and 80 per cent of the herd will need to be
bulls are available meeting these requirements (Fig.
bred pure.

4.31) (see also Chapters 5, 12). Genetic merit has been
converted into various numbers such as the production
index number (PIN), which is a comparison of milk

Table 4.16

Number of replacement heifers needed per 100
volume and weight of the milk constituents, and index
cows.

of total economic merit (ITEM), which includes both
production and conformation parameters.

Culling rate (%)

Age at first calving (years)

It has always been difficult for farmers to assess a
high genetic merit (HGM) bull in terms of their own

3

herds. The advent of a large number of companies
selling semen and the different methods used to express

15

30

38

45

genetic merit in various countries have also not helped.

20

40

50

60

It has resulted in the production of conversion formu-

25

50

63

75

lae to predict performance. Production information is

Table 4.17

Production associated with age at first calving. Source: Rearing replacements for
beef and dairy herds (MLC and MMB).

Age at first calving (months)

23–25

26–28

29–31

32–34

35–37

Herd life (years)

4.0

4.0

3.8

3.8

3.8

Lifetime yield (kg)

18 725

18 708

17 943

17 970

17 637

Yield/day in herd (kg)

13.1

13.2

13.1

13.1

13.2

Yield/day of life (kg)

8.8

8.4

7.9

7.5

7.3



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Fig. 4.31

Peckforton Citation River ET—

a high ICC Friesian bull.

expressed as predicted transmitting abilities (PTAs). In

(2)

At service: ensure the cow is on a rising plane of the UK, production (profit) index number (PIN) has nutrition and not losing weight. Oestrous detection has been used for several years, but was biased towards detection is a skill found in a good herdsman. Four milk quantity and constituents. This was superseded by observation periods during the day of at least 20 the index of total economic merit (ITEM) for a time, minutes each will aid oestrous detection. Oestrous which included conformation traits as well as those of detectors such as tail paint, dyes or vasectomized production. More recently the PLI (production plus bulls can be used to help detection.

lifespan index) has also been included to provide an

(3)

Post service: cows should still be closely observed indication of production and lifetime performance.

for repeat heats. Cows apparently in calf should

Other indices have been introduced including mastitis

be pregnancy tested at 24 days after service with
and calving interval.

a milk progesterone test or at six to eight weeks

In 2001 a new genetic evaluation system was intro-
after service with a veterinary examination.

duced for Holstein cattle in the UK. The system is based

To achieve a regular 365-day calving interval, first
on the Swedish interbull evaluations for production and
service should be at 50–60 days from calving, which
conformation. The procedure, developed in 1995, is
allows time for one repeat service before losing time.

known as the multiple trait across country evaluations

An MMB report on Checkmate, the Board's fertility
(MACE) and involves the incorporation of data on a
monitoring service, showed a wide difference between
particular bull from its own country and also from any-
the top and bottom 10 per cent selected on the interval
where else it has been used plus parent information from
between calving and assumed conception (Table 4.18).
the home country and abroad. As a bull is used in differ-
New techniques to improve the rate of genetic pro-

ent countries it reduces the evaluation contribution of
gress will doubtless be developed further over the
the parents to the overall genetic levels such as PIN. This
next few years. These mainly revolve around embryo
parental contribution can initially be up to a third when
transplants and include possibilities with sexed embryos
the bull has not produced daughters in another country,
and super-ovulated cows producing many embryos for
but reduces to about 1 per cent when it has been widely
non-surgical transplants. As the cost comes down and
used in that country and two or more others. The infor-
the success rate improves these techniques will be used
mation will still be expressed in PIN and PLI values.
on more commercial farms.

The other objective of obtaining a calf per year will
largely depend on herd management. Very few cows are
infertile but many fail to breed regularly. Key manage-

Health

ment times are as follows:

This subject will be dealt with very fully in the main

(1)

Post calving: ensure the cow is clean, has no dis-
text of the book. Suffice to say here that cow health is
charges and is cycling regularly.
paramount to success in dairying.

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Table 4.18

Example of overall herd fertility results. Source; Checkmate report, 1985
(FMS Information Unit, MMB).

Whole sample

Top 10%

Bottom 10%

Average interval calving:
assumed conception (days)

99

81

130

Average interval calving:
first service (days)

71

64

80

Services/assumed conception

1.82

1.65

2.22

Table 4.19

Average, bottom 25% and top 25% of farms based on retained profit/litre.

Source: ADAS and HSBC data to April 2000 (after Lott, 2000).

Bottom 25%

Average

Top 25%

Farm size (hectares)

99

102

114

Herd size

146

157

180

Yield per cow (litres)

6 860

6 967

6 863

Total milk produced (litres)

999 977

1 093 000

1 237 000

Output (pence per litre)

20.31

20.55

21.26

Variable costs (ppl)

7.73

7.17

6.78

Gross margin (ppl)

12.58

13.37

14.40

Direct overhead costs (ppl)

8.01

7.23

6.87

Rent, finance and quota (ppl)

4.07

3.66

3.08

Total overheads

12.08

10.89

9.95

Profit before drawings/tax (ppl)

0.50

2.48

4.54

Drawings/ tax (ppl)

2.90

2.58

2.45

Retained profit/ loss (ppl)

-2.40

-0.09

+2.45

Total cost of production (ppl)

22.71

20.64

18.81

Veterinary and medicine charges are usually between costs are paid wages, power and machinery, sundries, £35–80/cow or 0.4–0.8 p/litre and are made up of drugs, property charges, interest and depreciation.

particularly anthelmintics and antibiotics, and veteri-

Each year various farm advisory services produce nary charges. The main diseases that cause losses to the reports looking at the economics of specialist dairy farmer through loss of production, cost of treatment farms. These analyse the average as well as the top and or even death of the cow are mastitis (see Chapter 23), bottom 25% of farms selected by profit/hectare. The hypocalcaemia (see p. 781), hypomagnesaemia (see p. results in Table 4.19 are an example and highlight the 787), lameness (see Chapter 32) and calving problems. differences. The system is now known as benchmarking, which compares or measures a level of achievement which is seen as the target for that individual or

Economics of dairy farming

business.

For all the major factors those in the top group out-perform the others showing the enormous range of

Measures of efficiency

results found on fairly similar farms. However, on very

The overall measure of efficiency on any dairy farm is

many farms full records are not available to carry out

profit. Profit is calculated by deducting from the whole

this type of analysis and simpler records must be used.

farm gross margin the overhead costs of the business.

There are many, such as margin over concentrates,

The gross margin (GM) is the output of milk, calves and

margin over all purchased feed (MOPF) or gross

cull cows less the herd replacement charge to give the

margin, all per litre, per cow or per hectare. Statistical

gross output. From this are deducted the variable costs,

analysis shows none of these measures to be closely

of production, purchased feed, forage costs, veterinary

correlated to profit. The best measures are those where

costs, and medicine and sundry costs. The overhead

land area, and hence intensity of farming, are taken into

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Table 4.20

Analysis by margin over purchased feeds (MOPF) (£/cow).

MOPF (£/cow)

< 575

575–625

625–675

675–725

> 725

Milk yield (l/cow)

4741

5118

5414

5724

6245

Concentrate use (kg/l)

0.30

0.28

0.27

0.27

0.27

MOPF (p/l)

10.95

11.76

12.02

12.26

12.38

MOPF (£/cow)

519

602

651

702

773

Stocking ratea (LSU/ha)

2.24

2.15

2.18

2.18

2.27

MOPF (£/ha)

1079

1195

1314

1425

1634

a MOPF: margin over purchased feeds; LSU: livestock stocking units.

Table 4.21

Analysis by margin over purchased feeds (MOPF) (p/l).

MOPF (p/l)

< 10.5

10.5–11.1

11.1–11.7

11.7–12.3

> 12.3

Milk yield (l/cow)

5730

5677

5621

5494

5270

Concentrate use (kg/l)

0.33

0.29

0.27

0.24

0.21

MOPF (p/l)

9.7

10.8

11.3

11.9

12.6

MOPF (£/cow)

616

663

686

697

700

Stocking ratea (LSU/ha)

2.30

2.24

2.20

2.14

2.11

MOPF (£/ha)

1275

1370

1401

1395

1398

a MOPF: margin over purchased feeds; LSU: livestock stocking units.

Table 4.22

Analysis by margin over purchased feeds (MOPF) (£/ha).

MOPF (£/ha)

< 1100

1100–1300

1300–1500

1500–1700

> 1700

Milk yield (l/cow)

4975

5309

5538

5821

6172

Concentrate use (kg/l)

0.27

0.26

0.27

0.27

0.29

MOPF (p/l)

11.76

12.09

12.19

12.18

11.83

MOPF (£/ cow)

585

642

675

709

730

Stocking ratea (LSU/ha)

1.73

2.02

2.23

2.42

2.80

MOPF (£/ ha)

940

1195

1389

1598

1916

a MOPF: margin over purchased feeds; LSU: livestock stocking units.

account. Provided the overhead costs are not excep-

Milkminder report, which analysed data from 2459

tionally high, then margin over feed/ha (Table 4.22) will

herds, and *Genus Management Costed Dairy Farming*

have a large influence on profit.

1989–90.

However, with the quota system now limiting pro-

A high margin per cow results from a high yield per

duction on farms, many people are now saying that

cow with modest concentrate use. Stocking rate does

margin per quota volume and hence margin per litre not change substantially over the range of results. has become the most important measure (see Table 4.19). Farms with a high margin per litre had a lower milk yield per cow but with a much lower concentrate usage. Stocking rate also fell so there was little improvement in margin per hectare. Those with a high margin per

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Table 4.23

Details of spring- and autumn-calving systems.

an audit and accountability trail from the animal to the end consumer. They will usually involve regular peri-

Spring

Autumn

odic inspections of the accommodation, management, health and record keeping of the farm. These visits may

Yield (l/cow)

5228

5634

be undertaken by veterinary surgeons or other inspec-

Concentrate use (kg/cow)

946

1409

tors. The aim is to provide reports and, where necessary,

MOPF (p/l)

13.0

12.4

to enforce improvements within given time periods. In

MOPF (£/cow)

678

698

Britain the schemes are mainly run or approved by the

Stocking rate (cows/ha)

2.29

2.30

MOPF (£/ha)

1553

1605

milk purchasers. While at present they are limited in

their effectiveness they do provide the mechanisms to improve both animal health and welfare.

hectare had a higher yield produced with increased concentrate usage and with a much higher stocking rate.

Organic farming

The intensity of farming was much greater.

All these different systems of producing milk can

In the United Kingdom and other parts of Europe there lead to good profits if carried out efficiently. Therefore,

has been rising interest in organic farming. At present depending on the farm situation and the circumstances

in Great Britain only about 5 per cent of milk is produced in this way but the volume is growing. While

different systems can suit different farms.

A vigorous debate also ensues about the merits of seasonal calving and its effect on profits. Both spring calving and autumn calving are very defined systems that

are rewarded with a higher milk price. However, as stocking rates are lower and also costs of organic feeds

can be successful and profitable if operated efficiently.

are high, margins tend not to be greater than in

Table 4.23 shows the differences between the systems.

conventional farms.

A British spring-calving herd (calving February–

This method of food production has been receiving

April) produces a lower yield with less purchased feed

increasing attention over the last 15 years. Its main aim

than an autumn-calving herd (September–December).

is to ensure that production is undertaken in harmony

This gives a higher margin per litre but lower margin

with the environment and that as much is returned to

per cow and per hectare.

the land as is removed from it. The system has been

Both systems can be equally profitable but as the

taken up in many countries especially those of Europe.

margin per cow is lower for the spring calver than the

Organic farming is defined in EU law under EU Regu-

overhead costs must also be lower to show the same

lination 1804/99. The system involves meeting various cri-

profit.

teria before a farm can be defined as organic and this

Thus the level of overhead costs goes a long way to involves a conversion period which involves the land determining the farm system. Where overhead costs are and, in Britain, usually takes about 2.5 years. The objective very low (i.e. an owner-occupied farm with no rent, no taxes of the system are to sustain the animals in good borrowed money, no paid labour and little machinery), health by the use of effective management systems, then a very extensive system with low yields and low good stockmanship and suitable diets and by preventing outputs can be practised and still show a good profit. ing conditions where remedial treatments have

However, if the overhead costs are high then the farm previously been used. The use of alternative and complementary must be intensively run with high yields and high stockplementary medicines is indirectly encouraged by the ing rates, putting surplus land into arable crops to generate increase in stated withdrawal periods, usually by at least erate a high gross output. Only then will a satisfactory 100 per cent. The EU regulations only allow three 'sympoint profit be generated.

thetic drug treatments' a year (except for vaccines and antiparasitic preparations). At present a premium is paid for organic milk, which makes it an attractive proposition for some dairy farmers, but this is reducing the position for some dairy farmers, but this is reducing the use of land, labour, capital, stock and quota most efficiently.

The future

Farm assurance and herd

health schemes

While milk quotas remain within the EU it is probable that they will be further reduced for most nations in the short term and then gradually or rapidly abandoned. These are another initiative to ensure high management and health standards on dairy farms. They help provide The commodity price will eventually have to become

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closer to world milk prices under the direction of markets and the internet, or indirectly via shops of all various World Trade Organization initiatives. In several

types.

parts of the world cow numbers and milk production

Those doing the job well will survive although they

are being geared up to meet this. In most European

will have to keep their eye very much on the various

countries there will be a reduction in dairy cow

trends which develop concerning milk and its products.

numbers as yields increase. There will also be fewer

Whilst political decisions and the further reduction or

farms with larger numbers of cattle and proportionately

demise of milk quotas will close many doors, it is

fewer people employed. This will inevitably lead to

probable that others will open and there will be good

more cows or animals per stock person and an

opportunities in dairy farming for many producers.

increased requirement for mechanisation. Feeding

practices will continue to change to allow the expres-

sion of genetic merit with its increased yields per cow.

References

Sexed semen will facilitate a reduction in the number

of cows made pregnant to produce herd replacements

Farm Management Services Information (1990) Checkmate

and will also then allow poorer producing cows to be

report. Milk Marketing Board, Thames Ditton, pp. 1–17.

bred to beef bulls again, often with sexed semen.

Federation of United Kingdom Milk Marketing Board (1984)

Increasing movement of animal products may lead to

United Kingdom Dairy Facts and Figures, Thames Ditton,

more interest being paid to the methods of production

pp. 1–200.

in those countries which are primarily exporters. The

Federation of United Kingdom Milk Marketing Board (1990)

global transportation of food will probably also result

United Kingdom Dairy Facts and Figures, Thames Ditton,

pp. 1–209.

in increased levels of disease in those which are major

Genus Management (1990) An Analysis of Genus Man-

importers of animals and animal products. Unless

agement Costed Dairy Farming 1989–90, Crewe, pp.

disease levels in importing countries are allowed to rise,

1–24.

increased emphasis must be placed on health control

Hughes, J. (2000) Internal cattle building design and cow and monitoring in the exporting regions.

tracks. In *The Health of Dairy Cattle* (ed. by A.H. Andrews), Dairy farm incomes are likely to be further squeezed, pp. 278–98. Blackwell Science, Oxford.

resulting in the formation of various producer groups

Lott, E. (2000) Benchmarking – a vital tool to British agricul-to give improved bargaining power with milk buyers.

ture. *UK Vet*, 5, No. 6, 28–31.

Such milk producer groups will also be able to purchase

Meat and Livestock Commission (1999) Life after CPAS. *Beef* creameries and milk processing plants to improve inte-Management Matters, No. 6, 1–16.

gration of the supply chain. Much of the price structure

Milk Development Council (2000) Longevity – Controlling

Culling to Improve Herd Profitability. Publication 51

will still depend on the prices which the main super-

(03/00), Cirencester, pp. 1–12.

markets are prepared to pay. However, as prosperity

Milk Marketing Board (1986) EEC Dairy Facts and Figures,

increases more people will eat less at home and more

Thames Ditton, pp. 1–178.

outside the home. This will allow the development of

Milk Marketing Board (1990) EEC Dairy Facts and Figures,
the catering market at all levels and again will provide
Thames Ditton, pp. 1–210.

marketing initiatives. There will be more niche market-

National Dairy Council (2001) Dairy Facts and Figures 2001
ing, either direct to the public via farm shops, farmer
Edition, London, pp. 1–275.

Chapter 5

Heifer Rearing – 12 Weeks To Calving

B. Drew

Introduction

54

more intensive methods, particularly where the land

Age at calving

55

involved could be used for alternative, more profitable,

Month of calving

55

enterprises.

Rearing management, 3–6 months

55

The low priority given to heifer rearing is supported

Management and housing

55

by DAISY survey data (Kossaibati & Esslemont, 1996)

Feeding

56

which stated that in the average herd, 22 per cent of

Rearing management, 6–12 months

56

dairy heifers born were lost before they started their

Management at turnout

56

Supplementary feeding

56

first lactation, with a further 14 per cent culled before

Optimum growth rates

56

they completed their first lactation, while in the worst

Grazing systems

57

herds, almost 60 per cent of heifers failed to calve for a

Autumn management

58

second time.

Rearing management, 12–15 months

58

Calving at two years is more profitable than calving

Housing and management

58

at two and a half to three years as little over half the

Feeding for fertility

58

area of land is required, lifetime milk yield is greater

Stress

59

and the amount of feed, housing, labour and working

Breeding policy

59

capital required for the rearing enterprise is reduced.

Sire selection: breeding replacements from heifers

60

A policy of calving at two years can only be achieved

Selecting a sire

60

if the majority of cows calve within a relatively short

Selecting for longevity

60

Selecting for a low incidence of dystokia

61

period or a proportion of herd replacements are taken

Service management

61

from heifers.

Controlled breeding

61

The management of a dairy heifer between 12 weeks

Rearing management, 15–18 months

62

old and calving has a considerable effect on her poten-

Rearing management, 18–22 months

62

tial for milk yield, fertility, the incidence of dystokia and

Rearing management, 22–24 months (2 months precalving)

62

on longevity. Insufficient growth rates during the

Growth rates in pregnancy

62

rearing period result in the production of small-framed

Fly prevention

63

heifers with disappointing milk yields due to their inabi-

Cubicle training

63

lity to compete with older cows during the first lactation.

Minerals and trace elements

63

Poor growth rates at around the time of service result in

Management factors affecting dystokia and calf mortality

64

low pregnancy rates and delayed entry into the herd.

Management prior to calving

64

Management at calving

65

The faster a heifer grows, the more efficient she is in

Targets for growth for two-year calving

65

converting feed into liveweight gain. This could suggest

Management during the first lactation

65

that heifers should be fed to grow as rapidly as possi-

Survival rates

66

ble, especially during the summer months when grass provides an abundant and relatively inexpensive source of feed. However, in some circumstances, high growth

Introduction

rates during the first year can result in lower milk yields due to the effect of rapid growth on the relative

Dairy heifer rearing is generally considered to be a proportion of fat and milk secretory cells in the non-intensive, low-profit enterprise. Indeed, few farmers developing udder.

would rear heifers at all if it were not for their interest

The potential of a heifer will only be maximized

in herd improvement and disease control. However, if she is reared to a plan with careful control over planned heifer rearing is the starting point for performance at all stages of the rearing period. Target profitable dairying. An increasing number of farmers growth rates and intermediate target weights should be now realize the importance of this and are adopting set as soon as the predicted date of first calving has been

54

Heifer Rearing – 12 Weeks to Calving • 55

decided. Optimal growth rates will depend on age at

Table 5.1

Effect of age at calving on first lactation milk yield.

calving, estimated mature body size, management after

Source: Furniss *et al.* (1986).

calving and average herd yield.

Dairy heifer rearing is a specific farm enterprise

Age (years)

involving capital, land and labour. It should be managed

2

3

and recorded with the same attention to detail as
that given to the other enterprises on the farm, with

Milk yield (305 days) (kg)

4544

4980

preplanned target rates of growth being achieved

Calving interval (days)

386

401

throughout the rearing period.

A detailed description of the management and
targets required for autumn-born heifers reared to
months. It is known that the average life expectancy
calve at two years is given in this chapter. The pattern
of two-year calvers is greater and the lifetime milk
of growth and targets for weight-for-age for spring-born
production higher than heifers calving at three years
heifers calving at two years are similar. There is less
(Table 4.17).

pressure to maintain steady weight gains when the aim

There are a few circumstances when it is preferable

is to calve at an older age and there are periods when to adopt a policy of calving heifers at two and a half to lower growth rates are acceptable.

three years. Calving at an older age reduces the management pressures imposed by the two-year system and

Age at calving

permits the utilization of marginal land unsuitable for cropping. It also enables replacements to be taken from cows of high genetic merit even if they calve

The younger a heifer is at calving the less land, housing later in the season. Calving at three years can only be and capital are required during her rearing period but justified when there is a large area of outlying rough heifers should not calve at less than 23 months because a grazing.

higher incidence of calving problems can be expected and subsequent milk yields are disappointing. With a high stocking rate of 0.4 ha/livestock unit, the area

Month of calving

of land required for each replacement unit is 0.4 ha for heifers calving at two years, 0.5 ha for heifers calving

Most dairy farmers plan to calve their heifers in a batch at two and a half years and 0.7 ha for heifers calving at one season of the year. Autumn-calving herds find three years. When the stocking rate is 0.5 ha/livestock difficulty in maintaining this seasonality in the pattern unit the area required for heifers calving at three years of calving unless either herd fertility is high or a is twice that required for heifers calving at two. The proportion of the calves reared for replacements are housing and capital required to sustain a two year system taken from heifers. The optimum month of calving is also reduced. For instance, a 100 cow, autumn-calving varies with the relative prices of milk and compound herd calving at two years needs 40 replacements required and with the management pressures from other ing accommodation for calves and yearling heifers. enterprises. Once the optimum month of calving for a However, a similar herd calving at three years must herd has been established the heifers should be mated maintain 60 heifers and house the third-year heifers to calve at the same time or shortly before the earliest

during their third winter when space requirements are calving cows.

approaching those of adult cattle (Table 4.16).

Calving heifers before the cows reduces the stress

The first lactation milk yields of heifers calving at two imposed following introduction to the herd and allows years are likely to be lower than if the age of first for a longer interval between calving and service. How-calving is delayed. Milk yields of two-year-old calving ever, some farmers prefer to calve heifers at the same heifers at the ADAS Bridgets Research Centre aver-time as the early calving cows as they find that they aged 4544 kg milk as compared with a yield of 4980 kg are then easier to manage in the parlour.

for heifers calving at three years (Furniss *et al.* , 1986), However, when differences in the mean calving

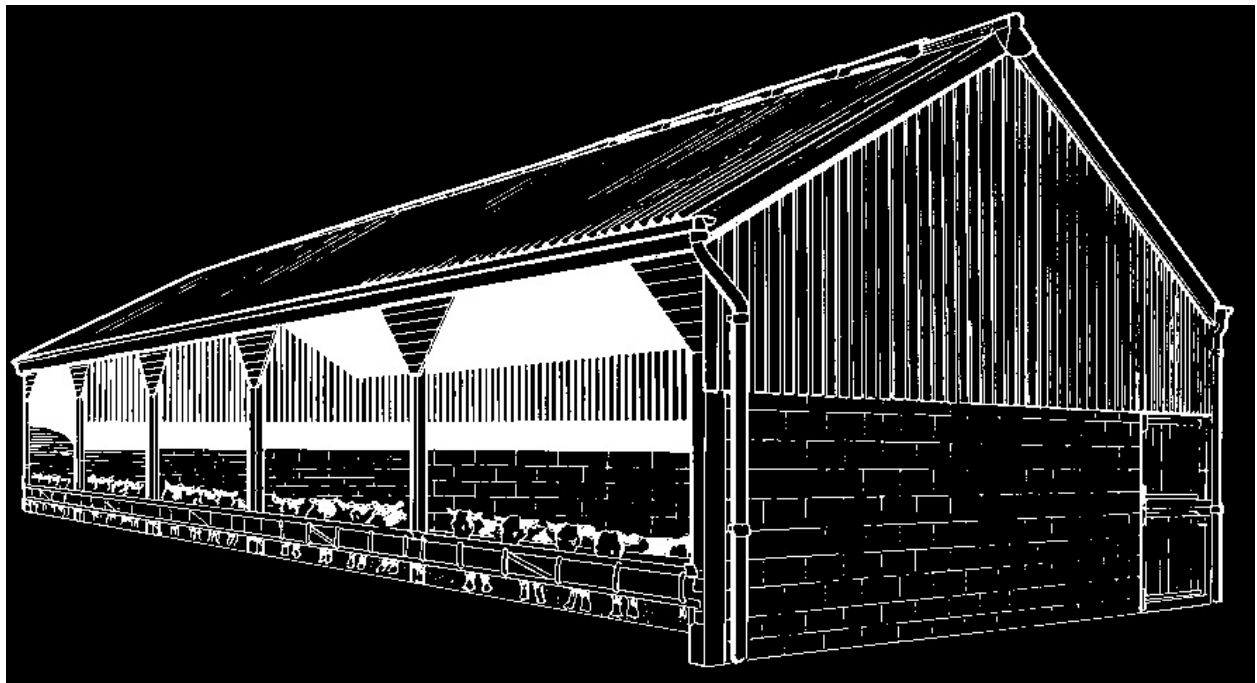
interval are taken into account the effect on yield was

Rearing management, 3–6 months

marginal (Table 5.1). This is supported by more recent research (Pirlo *et al.*, 2000) which showed that as age of

Management and housing

first calving increased there was generally an increase in milk yield and milk fat percentage, but at the same time rearing costs increased. The optimum age for first calving still appears to be approximately 23 to 24 months as much fresh air as possible with a draught-free lying



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Fig. 5.1

Example of a well-ventilated calf house.

area. Autumn-born heifers should be housed through-

Rearing management, 6–12 months

out the period but spring-born heifers may be turned out towards the end of the period if aftermaths are

Management at turnout

available.

Autumn-born heifers should be turned out to pasture when weather and soil conditions permit. After turnout the winter ration should be continued until the forage

Feeding

intake declines, generally within one to two weeks. The Minimum target growth rates during the first year of life compound can then be withdrawn. Spring-born heifers should be based on estimated mature body weight. A are normally housed until aftermaths become available. steady liveweight gain at a rate equivalent to the estimated mature body weight in g/day is required. Thus a

Supplementary feeding

Jersey heifer with a mature weight of 500 kg should be predicted to grow at 500 g/day (0.5 kg) while a Holstein Following withdrawal of compound feed soon after should be expected to grow at a minimum of 700 g/day

turnout, no supplementary feed should be required until (0.7 kg).

late August or early September. Attention should be

It is important to maintain a constant rate of gain.

given to mineral and trace element supplementation,

Poor growth rates, whether caused by an inadequate especially in areas of known deficiency (see Chapter 21).

diet or disease, are likely to lead to compensatory

gain at grass, which can have an adverse effect on

Optimum growth rates

subsequent milk yield.

From 12 weeks of age the calves should be fed to

Stocking rates should be adjusted to maintain a steady

achieve target growth rates, with either a silage, hay or

rate of liveweight gain over the summer. During May

straw based diet offered, supplemented with concen-

and June when grass is of high quality it is not unusual

trate feed as either a rearing nut or home mix. The total

for heifers to grow at 0.8–1.0 kg/day unless a tight stock-

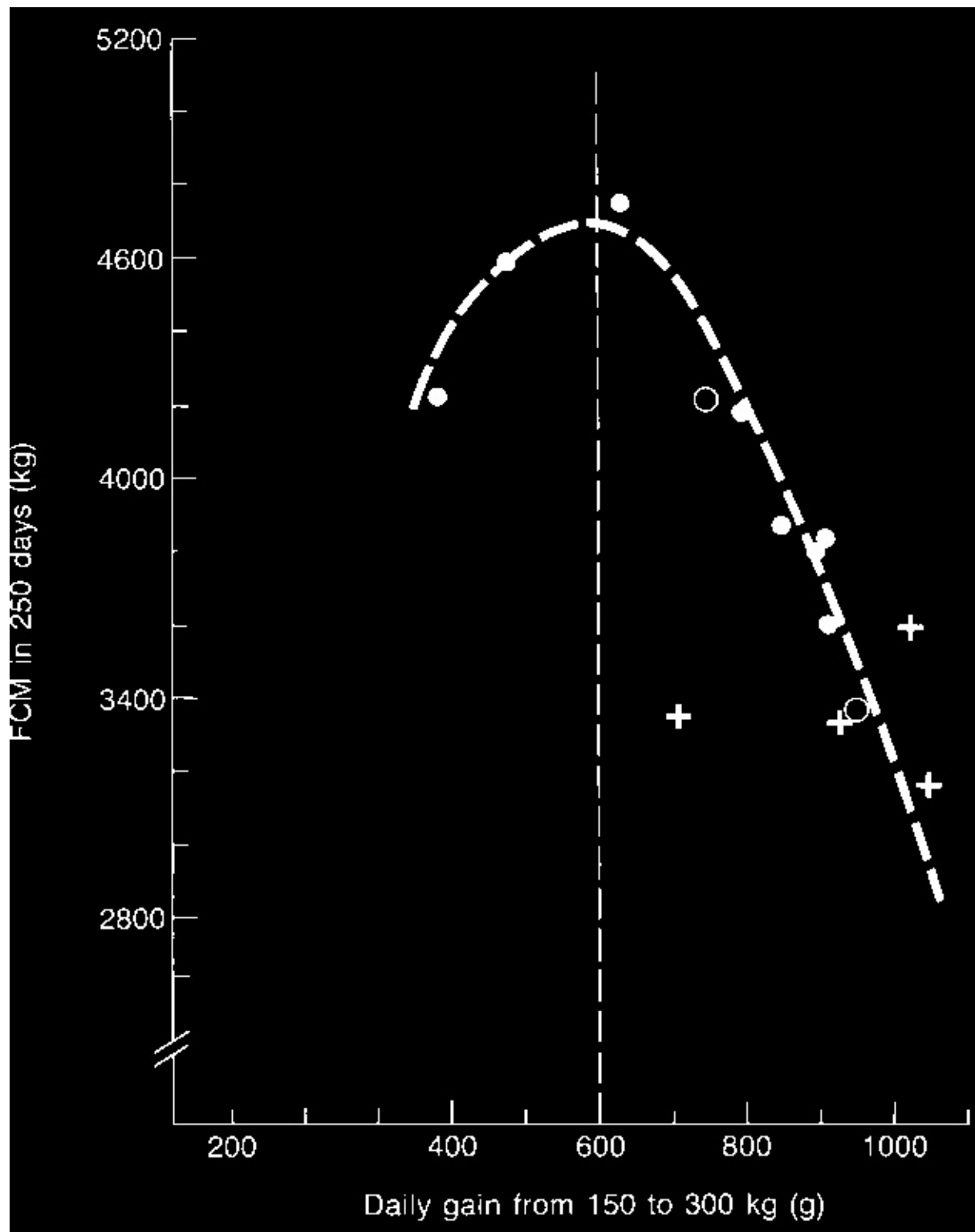
diet at this stage would typically be approximately 16

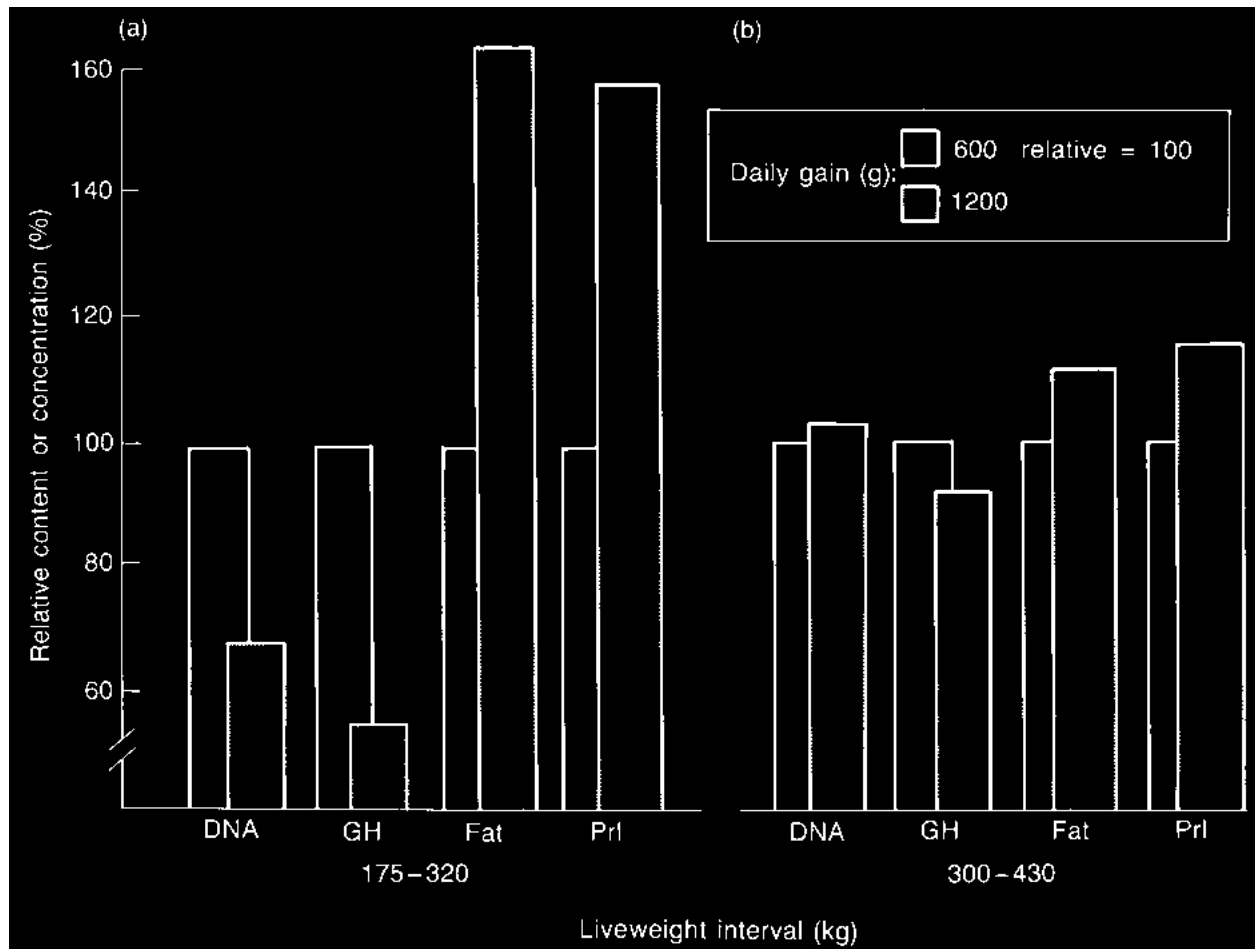
ing density is maintained. In the months leading up to

per cent crude protein. The amount of concentrate feed
puberty it is desirable to restrict daily liveweight gains
offered should be varied according to forage quality
to approximately 0.7 kg/day for Holsteins or equiva-
and the growth rates required. Forage should be of high
lent for smaller breeds based on the animals' predicted
quality, fresh and palatable. Calves should not be given
mature weight.

forage that would be unacceptable to cows. Calves fed
It is known that high growth rates in prepubertal
silage should also be offered clean barley straw and can
heifers are detrimental to milk production (Little &
be expected to take about 5 per cent of their roughage
Kay, 1979; Drew & Altman, 1982). A typical relation-
in this form. Regular monthly weighings are essential
ship between early liveweight gain and the first lacta-
to monitor progress and adjust nutrition. On infected
tion milk yield of heifers is shown in Fig. 5.2. Mammary
farms consideration should be given to vaccination
growth and development is under hormonal control
against lungworm prior to turnout (see p. 274).

and serum concentrations of some of the hormones





Heifer Rearing – 12 Weeks to Calving • 57

involved are affected by plane of nutrition (Sejrsen & these hormones. Rapid growth before puberty has Foldager, 1992; Sejrsen, 1994). It is likely that the negative influence of a high plane of nutrition on mammary the mammary epithelium, decreased serum growth growth is caused by changes in the secretion of hormone and elevated serum prolactin concentrations

(Foldager & Sejrsen, 1982). After puberty there is little or no effect (Fig. 5.3).

Summary evidence from recent studies at ADAS

Bridgets suggests that there are production and economic benefits from restricting the rate of liveweight gain (to around 0.7 kg/day) in the prepubertal period, in terms of subsequent milk yield performance, with little or no loss in terms of onset of puberty or first calving age and weight. There is also a potential benefit to be gained from a period of cheaper feeding followed by compensatory growth.

Grazing systems

Ideally, calves should be turned out to pasture with a low level of parasite challenge. Up to late June this can be classified as land that falls into one of the following categories: new seeds after an arable rotation and grassland used only for conservation in the previous year.

After mid July a pasture can be assumed clean if either it is an aftermath or not grazed by cattle earlier in the year (Chapters 19, 60).

If parasite-free calves are turned out to and remain

on a clean pasture it should be safe to graze for the remainder of the season. Unfortunately, in intensive dairy systems it is seldom possible to provide clean

Fig. 5.2

First lactation milk production in relation to early life grazing as such. On permanent pasture, helminth egg liveweight gain (after Foldager & Sejrsen, 1982).

output can be suppressed by anthelmintic treatment.

Fig. 5.3

Relative content of DNA

and fat in mammary glands and serum

growth hormone (GH) and prolactin

(Prl) concentrations in (a) prepubertal

and (b) post pubertal heifers raised on

two planes of nutrition (after Foldager

& Sejrsen, 1982).

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Overall it is important to reduce parasitic challenge in veterinary treatments required during the period. They the heifers' first grazing season whilst allowing suffi- should be housed in semi-covered yards with a total

cient challenge for them to build up natural immunity.
space allowance of approximately 6 m²/animal. A clean
Individual parasitic control programmes should be
concrete loafing area should be included to encourage
drawn up for each herd (Chapter 19).
good hoof development. A special area of cubicles can
In continuous grazing systems the stocking rate
be left to train the heifers.
should be adjusted to match the grass available and the
required growth rate, aiming for a sward height of 5–6

Feeding for fertility

cm in spring, 7–8 cm in mid summer and 9–10 cm in
autumn. In what is known as the '1–2–3' grazing system
It is known that the level of nutrition over the service
the grazing area dedicated to the heifers is divided into
period is important in ensuring high pregnancy rates
three blocks. In spring the heifers graze one area while
and entry into the herd at the optimum time. In a study
the other two blocks are cut for silage. Later the heifers
undertaken on six commercial dairy farms, the fertility
graze the two silage aftermaths while the grazed block

of heifers fed rations considered to be adequate by
is cut for silage. At the end of the season all three blocks
the farmers was compared with the fertility of heifers
can be used for grazing.

fed the same diets supplemented with cereal (Drew &
An alternative grazing system is described as the
Pointer, 1977). The supplemented diets were calculated
'leader-follower' system. This is a rotational grazing
to provide an additional 20 MJ/day metabolizable
system of eight to twelve paddocks where the youngest
energy (ME) and were fed for a 12-week period
heifers graze each paddock ahead of older heifers
commencing six weeks prior to service. Ovulation was
(Kossaibati & Esslemont, 1986).

controlled and the heifers were inseminated at a fixed
Grass growth and supply should be monitored
time. Semen and inseminators were used equally over
throughout the summer to achieve the required
all groups.

growth targets. Concentrate supplementation should be
The calving rates to fixed time insemination are

provided if required.

shown in Table 5.2. There was a wide variation in fertility between farms, but on each farm a higher pro-

Autumn management

portion of heifers fed the improved ration calved to the synchronized service. The calving rates to first service During the period May–August target liveweight gains for the heifers on the farm ration ranged from 32 to 67 should be achieved without difficulty. At the end of this per cent and for the supplemented groups from 59 to period the heifers should be well grown for their age 79 per cent. In this study, no significant relationship and breed without any obvious fatness (Table 5.9). between body condition score and fertility was found. Heifers calving at two years require supplementary However, other workers have reported a relationship feeding from late August or early September depending between body condition and fertility, with lower pregnancy rates occurring at each end of the score range. rates decline rapidly from this time and if additional

It is likely that change in body condition score energy is not supplied liveweight gains will be low. A has a greater effect on fertility than the actual score compound feed or home mixed cereal ration supplemented with minerals and vitamins should be fed. require to grow at a rate of approximately 0.8 kg/day in Regular weighings are essential at this time for order to maintain body condition. As it has been shown both autumn- and spring-born heifers programmed for calving at two years. Heifers being reared to calve at three years can be allowed a store period.

Table 5.2

The effect of level of nutrition on the fertility of dairy Autumn-born heifers should be assessed for size and heifers.

suitability for service. Actual weights should be compared with targets. If any animal is found to be below

Farm

Number of

Percentage calved to the

heifers

synchronized insemination

target but, with additional feeding, could attain the
minimum service weight, she should be housed at the

Control

Supplemented

beginning of September and fed an appropriate ration.

1

62

40

67

2

58

59

75

Rearing management, 12–15 months

3

56

32

59

4

37

67

79

Housing and management

5

78

54

69

6

73

53

67

Heifers reared for two-year calving should be housed

Mean

61

50.0

68.9

at least six weeks prior to service and given any routine

Heifer Rearing – 12 Weeks to Calving • 59

that loss of body condition is associated with low preg-

Table 5.3

The effect of the parity of the dam on lactation
nancy rates a ration calculated to provide for maintenance of heifers.

nance and at least 0.8 kg/day liveweight gain should be
provided to Holstein/Friesian heifers over the service

Dam

period. Rations for other breeds should be adjusted

Heifer

Cow

according to relative body size.

Attempts to reduce the period of supplementary

Weight at first service (kg)

370

360

feeding to three weeks before and after service have

Weight at calving (kg)

496

503

been only partially successful. Reducing the period of

First lactation yield (305-day) (kg)

4977

4742

supplementary feeding to three weeks prior to service

Proportion rebred

0.8

0.8

should only be considered if Friesian heifers are in condition score 2.5–3.0 (Holstein and Jersey 2.0–2.5), or heifers are to be mated by natural service or observed oestrus artificial insemination (AI) and the reduced period of feeding does not affect the time of housing.

must be good to achieve acceptable results and avoid

The period of supplementary feeding after service disappointment.

should not be shortened as it is likely to increase the

The cost of keeping a bull is often less than AI but incidence of early embryonic loss.

the risk of disease and injury are greater. The increased

In addition to providing a ration to satisfy the above cost of using AI can only be justified if a pure bred sire standards it is important to avoid major changes to is used and the female calves reared for herd replace-

the composition of the diet during the ten days before
ments. A few farmers are still reluctant to take replace-
and after service. Changes in diet are almost always
ments from heifers, preferring to use mature cows with
associated with disappointing pregnancy rates.

a proven record of performance. The rate of genetic
progress will be greater when replacements are taken
from heifers, providing a sire of high genetic merit is

Stress

used.

Stress, such as that caused by noise, physical trauma,

A study by Furniss *et al.* (1988) of the first lacta-

overcrowding or some routine veterinary treatments,

tion and breeding records of 569 heifers calving in a

can alter the concentration and pattern of secretion

Friesian/Holstein-based herd showed that those heifers

of the reproductive hormones and is, therefore, poten-

born to heifers yielded significantly more milk in 305-

tially detrimental to fertility. In intensive manage-

day lactation than those from cows (Table 5.3).

ment systems it is difficult to eliminate the possibility

Rearing replacements from heifers and the earliest of stress, but with careful planning it can be reduced to calving cows enables a two-year calving policy to a level at which pregnancy rates are not likely to be adopted, with herd replacements entering the herd be affected.

at the optimum time. The management of the heifer

Housing should be undertaken six weeks prior to

rearing enterprise is simplified as it should only be

service as oestrous behaviour is suppressed during

necessary to rear calves born over a six to eight

the first oestrous cycle after housing. It is important to

week period. It also assists in the maintenance of the

ensure that sufficient trough space is provided to avoid

optimum calving pattern as the later calving cows can

competition. There should be an adequate loafing area,

be mated to a beef bull.

the building should be well ventilated and the floor such

The incidence of dystokia need be no greater with

that animals in oestrus will not slip. If AI is to be used,

a selected AI Holstein/Friesian bull, than a Hereford

the handling facilities should be designed to allow for bull used in natural service. In a survey of 61 herds quiet and efficient movement of animals. The heifer recording aspects of rearing and calving experience should be presented standing at the same level as the with heifer calvings, the incidence of dystokia associated with Hereford sires, averaged over all data, was almost identical to that associated with Friesians (Table 5.4). There was no difference between the breed in calf

Breeding policy

mortality rates. When using Holstein/Friesian AI bulls for heifer inseminations there is no need to restrict the Some farmers consider oestrous detection in heifers to selection to those noted to be easy calving although be too difficult and time consuming for AI to be practical. Those recorded as having greater than average calving cal. This is likely to be a problem when groups of heifers difficulties should generally be avoided. The most graze outlying fields away from satisfactory handling critical factor is overall heifer rearing management to

facilities. Synchronization offers a possible solution to ensure that they are large but not fat at the time of this problem but the management standards on farms calving.

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Sire selection: breeding

many can lead to unnecessary complication and lack

replacements from heifers

of uniformity in the herd. There are two key economic indices used to describe the overall production potential of sires in the UK: these are profitable life index

A survey of farmers breeding replacements from

(£PLI) and production index (£PIN). £PLI and £PIN

heifers showed that the main selection criterion was

are used to describe the average net margin per cow per

‘ease of calving’. It would appear that it is not unusual

year which the bull is likely to pass to his daughters and

for this issue completely to override all the principles

bulls can be ranked on these values for comparison. A

taken into account when selecting replacements from

short list of sires based on their £PLI and their overall

cows. Any heifer mated to a pure-bred bull is potentially a dam of the next generation. The bull used must be at least as good as those used on the cows and should have genetic merit of the existing herd, with high genetic merit also be genetically superior to the sire of the heifer to which it is mated. Only by adopting these basic principles can genetic progress be made. When a bull is selected to be a sire of the next generation, attention should be given to the effect that he may have on the incidence of dystokia.

ability (PTA) indicates the relative production potential in terms of milk volume and composition. The reliability of PTAs should be considered: the higher the

Selecting a sire (see p. 48–9)

ability of PTAs should be considered: the higher the

reliability, the greater the confidence in the predicted Genetic improvement has long-term effects on herd values. Generally, sires with a PTA reliability of 70 per performance and profitability. The effect is cumulative or greater should be selected. Once a short list of five, making the inclusion of heifers in the breeding top production bulls has been produced, the selection of replacements important for maximizing genetic should be refined using factors such as milk composition (Table 5.5). It is advisable to select a 'team' of three and functional conformation traits including several sires (typically three or four) for a herd, including udders, legs and feet, SCC and calving difficulty. Using two or more which are suitable for use on maiden heifers. Using several sires reduces risk, although too

Selecting for longevity

Few studies have been undertaken in this country on

Table 5.4

Effect of breed of sire on dystokia in heifers.

the relationship between conformation (type), production and longevity. Many studies of these relationships

Breed of sire

Percentage of calvings

have been undertaken in the USA and show that first lactation yield is the best early indicator of longevity

(No. of heifers)

Unassisted

Assisted

Difficult

and lifetime yield of dairy cattle. Type scores may add a little to the accuracy of prediction, but not nearly as

Friesian (1181)

63

25

12

much as is commonly assumed by breeders. To a great

Hereford (753)

63

24

13

extent, longevity in cows depends on the management

Table 5.5

Example of accumulating genetic merit in younger animals.

Lactation No.

No. of animals

Average £PINa

Average PTA_b

Average

Milk (kg)

Fat (kg)

Prot (kg)

Fat (%)

Prot (%)

reliability

Young stock

289

53

460

13.0

14.6

-0.08

-0.01

27

1

97

48

417

11.6

13.3

-0.07

-0.01

44

2

130

37

314

10.1

10.1

-0.04

0.00

51

3

90

34

312

9.3

9.5

-0.05

-0.01

54

4

73

23

224

5.6

6.5

-0.05

-0.01

59

5

31

10

148

4.1

3.1

-0.03

-0.03

61

a PIN, production (profit) index number.

b PTA, predicted (production) transmission ability.

Heifer Rearing – 12 Weeks to Calving • 61

decisions of the farmer. However, differences in bulls that were the easiest calving overall, while other longevity between cows within the same herd are farmers reported that heifers calved without assistance now believed to be heritable and factors thought to influence longevity are being included in genetic selection view of the considerable variation between farms and parameters.

farm/bull interactions it is not possible to be certain whether dystokia will occur on an individual farm.

Heifer calves are generally smaller than bull calves

Selecting for a low incidence of dystokia

and with the advent of sexed semen it is now possible

The service sire has a considerable effect on dystokia.

to inseminate maiden heifers with 'female' Holstein

The results of a detailed analysis of 1485 heifer calvings
semen to ensure maximum genetic progress in the herd
to six Friesian/Holstein bulls used equally on 58 farms
and at the same time reduce calving difficulties.

showed that the differences between the bulls used in

Other factors involved in dystokia are discussed in
the percentage of assisted and difficult calvings were
the section on 'Management at calving' (see p. 65).

statistically highly significant (Table 5.6). However, the
effect of the bull was far less marked than the manage-
ment on the farm on which the heifer calved. Of the 58

Service management

farms in the study, 18 reported mean calf mortality rates
of 1.3 per cent born dead or died within 24 hours and

Controlled breeding (see also Chapter 42)

recorded a low incidence of dystokia with all bulls.

Twelve farms reported mean calf mortality rates of 26.9

The introduction of synchronization techniques in the
per cent, which on some farms was associated with a
mid 1970s offered farmers a unique opportunity to

high incidence of dystokia. On the farms with low mortality, none of the bulls was associated with an incidence of dystokia of more than 4 per cent, while on the high rate individuals from a group for service. The results of numerous studies have shown that the pregnancy mortality farms no bulls resulted in an incidence of less than 20 per cent. In this study the bulls used were rates following prostaglandin or progestagen treatment known to be breed average or less for both dystokia on are similar to those obtained with untreated controls cows (<2.7 per cent) and gestation length (<281 days). inseminated at observed oestrus. Despite these findings, In Table 5.6 only bulls 1 and 3 were specifically the uptake of commercial techniques to control the recommended for ease of calving. This highlights the bovine oestrous cycle have fallen well short of expectations, as in the field results can be disappointing based on heifer calvings, that the bull has been used

where herd management or supervision of the breeding programme are not of a sufficiently high standard. In general, bulls with short gestation lengths give easier calvings. The mean gestation lengths of bulls in this study ranged from 273 to 279 days. Providing a bull spread use. Initially, farmer expectation was too high. is not known to be above breed average for dystokia on Many had been accustomed to running heifers with a cows, he can be considered for use on heifers, especially natural service bull for perhaps nine to twelve weeks. if the gestation length is also less than the mean for the By this time around 90 per cent should have become breed. However, some farmers reported dystokia with pregnant, even with modest levels of performance. Farmers have, therefore, become used to finding the majority of heifers confirmed in calf when manually examined as a group and tend to expect similar results from a single AI mating.

Table 5.6

The effect of bull on dystokia in heifers.

Compared with natural service, the cost of a con-

Bull (number of

Percentage calvings

trolled breeding programme with AI is likely to be at

calvings)

least £25–£35 if a double insemination is given. If only

Normal

Assisted

Difficult

40 per cent of served heifers produce a calf and 50 per

cent of those are male the service cost per heifer calf

1 (236)

52

37

11

born could easily be in excess of £100.

2 (231)

70

25

5

Although pregnancy rates of 40 per cent are not

3 (249)

79

19

2

unusual, the management factors affecting fertility in

4 (221)

71

24

5

dairy heifers are generally well understood. Providing

5 (261)

79

19

2

the recommendations given in the section 'Rearing

6 (263)

63

31

6

management, 12–15 months' (see p. 58) are followed,

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pregnancy rates of 65–75 per cent should be achieved.

levels according to size at this time enables the heifer

At this level of fertility the use of synchronization

group to become more evenly matched. High-quality

becomes more attractive if used only for a proportion

silage and barley straw without further supplementation

of the group.

should be sufficient for well-grown heifers. A mineral

Several synchronization techniques (p. 678) are now

mix should be fed to heifers given silage and straw alone.

routinely used for controlled breeding of heifers,

including the use of prostaglandins, progestagen and

GnRH. The oestrous response following synchroniza-

Rearing management, 18–22 months

tion in heifers is generally precise and pregnancy rates

to fixed time insemination satisfactory. However, a

Autumn-born heifers should be turned out as soon as

proportion of heifers do not show oestrus within the

soil and weather conditions permit. Nitrogenous fertil-

expected window after treatment and programmes may
izer with phosphate and potash according to soil
therefore recommend AI only at observed oestrus or
requirements should be applied. The sward height
AI at a fixed time followed by either AI at subsequent
should be maintained at about 6–8 cm. Target stocking
observed oestrus or natural service.

rate at this stage should be six to seven livestock

An example synchronization programme would be a
units/ha.

system whereby the heifers are housed or moved to a
field adjacent to the handling facilities six weeks before
commencement of service. At the same time the heifers

Rearing management, 22–24 months

are weighed and the diet adjusted to ensure a minimum
gain of 0.8 kg/day with adequate mineral supplementa-

(2 months precalving)

tion. Any routine veterinary treatments necessary are
undertaken and a check made to ensure that the heifers

In the two months before calving heifers should be
can be clearly and easily identified. After a minimum of

housed or grazed with the dry cows of a similar stage four weeks all heifers are injected with prostaglandin of gestation as this reduces the stress of introducing and observed closely for standing oestrus. Heifers not them to the milking herd post calving. If heifers are inseminated after the first injection are given a second to be managed and milked as a separate group during injection and inseminated at a fixed time, either once their first lactation, precalving heifers can be managed at around 84 hours after treatment or twice at 72 and as a separate group. In the last two months of pregnancy 96 hours after injection. Following the synchronized the growth rate of heifers should be controlled to mating either the heifers are observed for repeat AI, or approximately 0.7 kg/day.

a bull of another breed is turned in as soon as the period of intense oestrous behaviour has subsided, which is

Growth rates in pregnancy

usually on the fifth or sixth day after the second injection.

Target growth rates between the third to eighth month

of pregnancy should be approximately 0.8 kg/day, but should be adjusted to ensure adequate calving weights.

Rearing management, 15–18 months

The target calving weight for a heifer depends on her estimated mature body size and the management

Housed heifers should be allowed approximately routine during the first lactation. The target calving 7 m²/head from this age. The preservice ration should be weight for a Holstein heifer is around 650 kg precalv-maintained for at least six weeks. As soon as it is known ing. In management systems where heifers are fed as a that pregnancy has been established in the majority of heifer group in the first lactation there is obviously less heifers, decisions can be taken on the level of nutrition competition than where heifers are competing with for the remainder of the period. Their weights should be older cows.

recorded and compared with target (Table 5.9). They

Two-year calving heifers competing with cows in self-should be grouped according to size. Required growth fed silage systems are at the greatest risk as not only are

rates should be calculated using the target calving
they relatively small in weight and stature but they are
weight and feed adjusted accordingly, with higher levels
also changing their teeth at this time.

of supplementary concentrate feed offered to the

In an investigation involving Friesian/Holstein
smaller heifers. Growth rates of up to 1 kg/day or more
heifers calving at two years, Keown (1986) showed that
are not uncommon in Holstein heifers at this age. It is
growth rate during pregnancy has a significant effect
likely that, at this age, the variation in weights of heifers
on milk production, with the heavier heifers at calving
of similar age will be around 80–100 kg. Adjusting feed
producing the most milk (Fig. 5.4).

Heifer Rearing – 12 Weeks to Calving • 63

900

800

700

600

500

400

300

200

Increase in milk yield (kg)

100

0

Fig. 5.4

The effect of weight at calving on

408

431

454

476

499

522

544

567

590

612

635

658

680

first lactation yield (increase in first lactation

Body weight post calving (kg) based on American data (Keown, 1986) milk yield). Data used with permission.

Weight, height and pelvic length at calving were

Cubicle training

found to affect milk yield. All these relationships were

Some dairy heifers can show clinical signs of laminitis statistically significant and suggest that while rapid early in their first lactation. This is frequently a stress-growth in the prepubertal heifer is detrimental to milk related condition associated with changes in the environment, social grouping and feeding, which occur at yield, the converse applies during pregnancy.

The initial results suggested that there is a positive

the time of first calving. It has been shown that housing relationship between body condition score and milk heifers for a few weeks during a period when cubicles yield throughout the condition score range. However, a are not occupied by cows has two advantages. Firstly, more detailed analysis showed that when heifers are fed the heifers become accustomed to using cubicles and adequately during the first lactation there is no benefi-

are less likely to reject them following introduction
cial effect on milk yield of feeding to calve them at body
into the herd, and secondly, as one of the postparturi-
condition scores above 2.5 for Friesians or greater than
ent stresses is eliminated, the incidence of laminitis and
2.0 for Holsteins or the Channel Island breeds. This
other stress-related metabolic diseases is reduced.
information is reassuring in view of the fact that an
Providing a clean concrete loafing area whenever the
increased level of assistance at calving was required
animals are housed, throughout the heifer rearing
when heifers calved at condition scores of 3.0 or more
phase, encourages better hoof formation and reduces
(see pp. 10–11).

problems when heifers move into cubicle yards post
To a considerable extent, dystokia can be avoided by
calving.

careful management in the ninth month of pregnancy.

Growth rate should be restricted at this time.

Minerals and trace elements

A detailed description of some of the minerals and

Fly prevention

trace elements of particular significance to the dairy
Summer mastitis can be a cause of loss in heifers (see
heifer is given in Chapters 21, 46. In view of the fact
Chapter 24). It is most likely to occur between the fifth
that dairy heifers receive relatively little compound
and eighth month of pregnancy, usually in July, August
feed, deficiencies are more likely than with the milking
and September. The incidence of summer mastitis can
herd. The advice given regarding minerals such as
be reduced by grazing large open fields away from
phosphorus, copper, cobalt, selenium and manganese
woods and streams and by the use of fly repellent meas-
fed to dairy cows applies equally to dairy heifers (Chap-
ures. Impregnated ear tags, sprays and pour-ons have
ters 18, 21).

proved to be of benefit in preventing disturbance and
Although iodine deficiency may not be a widespread
irritation caused by flies and reducing the incidence of
problem, it can be responsible for serious losses on indi-
New Forest eye and summer mastitis. The incidence of

vidual farms (see pp. 253–60). If perinatal calf mortality can also be reduced by regular (e.g. weekly) treatment with proprietary fly repellents.

previous years an investigation into the iodine status of

64 • Chapter 5

the herd is recommended early in the season and treat-

Table 5.7

Effect of weight gain during pregnancy on dystokia.

ment undertaken during the fifth to eighth months of pregnancy.

Weight gain

Calf

Percentage calvings

Magnesium supplementation is normally given to

(kg/day)

mortality

(%)

Normal

Assisted

Difficult

dairy cows prior to calving in order to reduce the incidence of hypocalcaemia. As heifers are less prone to

<0.4 (49)

19

61

35

7

this condition, supplementary magnesium is not always

0.41–0.60 (348)

10

74

25

3

provided (see Chapter 46). Data from ADAS trials

0.61–0.80 (854)

11

69

26

5

suggest that magnesium supplementation in the month

>8.0 (199)

14

64

28

8

prior to calving reduces the incidence of dystokia.

As the energy intake should be restricted at this time

Number of cattle recorded at each weight gain is given in brackets.

it is usually most convenient to provide this via the
water.

Table 5.8

Farms with calf mortality rates below 5 per cent compared with farms with calf mortality rates above 20 per cent.

Management factors affecting

Heifer measurements

Low

High

dystokia and calf mortality

mortality

mortality

Management at around the time of calving has a major

Number of farms

18

12

effect on dystokia and calf mortality. Management

Number of heifers

447

212

Average number/farm

25

18

prior to service and during the first eight months of

Mean age at calving (days)

731

734

pregnancy is generally considered to have little effect,

Mean weight at service (kg)

330

323

although opinion is divided. The results of a large-

Mean weight at calving (kg)

504

492

scale investigation into the factors affecting dystokia

Mean height at service (cm)

117

118

showed a tendency for calf mortality and dystokia to

Mean height at calving (cm)

128

127

be greater at each end of the growth rate range

Mean distance hook to pin

44

43

(Table 5.7).

at service (cm)

On the basis of this study, high weight gains during

Mean distance hook to tail

38

38

pregnancy should not result in unacceptable levels

head at service (cm)

of dystokia, especially if liveweight gain is restricted

during the final month of pregnancy. An analysis of the precalving weight and height records showed there to be no significant relationships with dystokia.

relating to the management of the heifer at around the

In order to study the effect of management on the time of calving.

factors affecting calving difficulty and calf mortality, the performance of the 18 farms with calf mortality rates below 5 per cent was compared with the performance

Management prior to calving

of the 12 farms with calf mortality rates over 20 per cent. There was no difference between the groups in the Providing weather conditions permit, it is preferable to growth rates of the heifers (Table 5.8).

graze heifers in a field or paddock adjacent to the dairy

These figures suggest that growth rate of the heifer complex, where the forage is naturally sparse or can be has little effect on dystokia. Equally, there appeared to deliberately restricted to ensure that the optimum body be no differences between the groups in the size of the condition at calving is achieved. The heifers should have

calves born, farmers with both low and high mortality the opportunity for exercise. The diet offered should be rates recording a similar proportion of small, medium a correctly formulated transition diet which introduces and large calves. The high mortality rate farms assisted the heifer to the main forages and feeds which she will a greater proportion of heifers in all of the size group be fed post calving. Mineral supplementation remains categories and classified 58 per cent of large calves as as for dry cows. Supplementary magnesium should be difficult calvings compared with only 8 per cent of the provided.

large calves on the low mortality rate farms.

The group should be observed at least four to five

There is no evidence from these data to suggest

times daily from three weeks prior to the estimated date

that the management of the heifer during pregnancy

of the first calving. Care should be taken to adjust the

accounted for the differences obtained. The differences

‘due to calve’ dates for the gestation length of the bulls

would, therefore, appear to be due mainly to factors

used.

Heifer Rearing – 12 Weeks to Calving • 65

Management at calving

A study involving 179 small-framed Friesian heifers calving at two years, all the progeny of one sire, compared the milk yields and longevity of heifers fed with Grazed heifers should calve in their allotted field or paddock if possible; housed heifers should calve in or without competition during their first lactation. The buildings with which they are familiar.

first lactation milk yields and survival rates to the end of the fifth lactation of heifers at a range of service a calving box increases the risk of dystokia and there-weights are shown in Table 5.10.

fore it is preferable to avoid movement unless this is On this farm no allowances were made for the size essential for adequate assistance (Chapter 67). The field of heifer or the effect of competition on milk yield. A should be well fenced to avoid the possibility of heifers higher percentage of heifers in the group fed with cows

rolling into positions from where it would be difficult to were therefore culled on grounds of disappointing milk assist. The herdsman should be trained to recognize yields.

fear, abnormal pain or distress and instructed on the

In a subsequent study, the lactation records of 1346 correct use of calving aids.

cows on 54 farms were examined to determine the extent to which heifers were affected by competition with cows. The farms were allocated to one of three groups according to the method of management during

Targets for growth for

the first lactation:

two-year calving

Group 1: fed as a heifer group throughout the winter period.

The targets shown in Table 5.9 refer to heifers calving

Group 2: housed with cows. Fed a complete diet or at two years. The target growth rates for heifers calving manger fed.

at 2.5 or 3.0 years are less as they can grow at a slower

Group 3: housed with cows. Fed on self-feed silage.
rate overall and during growth they can be allowed a
store period. The maximum growth rates suggested for
Heifers fed as a heifer group (group 1) gave more milk
the first year and the minimum growth rates during the
than those fed in competition with cows (Table 5.11).
immediate preservice period apply.

Farmers electing to feed cows in groups are more
likely to be aiming for high yields than those where the

Management during the first lactation

Table 5.10

The effect of competition on first lactation milk yield
and survival rates to the end of the fifth lactation.

Heifers fed in competition with cows during their first
lactation are likely to give less milk in the first lactation

Weight at service (kg)

Milk yield (kg)

and have less chance of surviving to calve for a second
time than when fed in a heifer group.

Competition

No competition

225–259

2892 (33)^a

3852 (37)

260–279

3334 (46)

4070 (73)

280–299

3639 (18)

3992 (52)

300–349

3131 (21)

3857 (32)

Table 5.9

Growth targets for two-year calving of Holstein

heifers.

^a Numbers in parentheses represent survival rate percentage.

From

To

Daily Target

liveweight liveweight

(kg)

gain

(at end of

(kg)

period)

Table 5.11

Mean first lactation milk yields.

Birth

Weaning

0.50

65

Group 305-day

yield

Total

Lactation

Weaning

4 months

0.90

150

(No.)

lactation

length

4 months

10 months

0.65

280

Milk

Fat

Protein

yield

10 months

13 months

0.80

350

13 months

17 months

0.90

460

1 (207)

5200

209

170

5369

319

17 months

22 months

0.90

595

2 (580)

4504

180

146

4641

308

22 months

Calving

0.65

630 (pre calving)

3 (559)

4274

174

140

4358

whole herd is managed as a single unit. When the milk

Survival rates

yields are compared with the average herd yield it can

be seen that the true effect in heifers between herds is

On average, cows in Britain fail to survive in the herd

some 5 per cent of mature potential (Table 5.12).

for sufficient time to fulfil their mature yield potential.

Although heifers fed in competition with cows during

The mean length of herd life is less than 3.5 lactations,

their first lactation are likely to yield less milk, it is not while mature yields are not obtained until the fourth,

always possible, feasible or economic to manage, milk

fifth or even the sixth lactation. It is also estimated that

and feed them separately.

some 40 per cent of all heifer calves born alive fail to

The effect of competition can be minimized by ensur-

calve for a second time. The main reasons for disposal

ing that heifers are well grown prior to calving. Size is

are fertility and low milk production. It is evident that

important, but not the only factor determining the peck order within a group of cows. the rate of growth during the rearing period influences

milk yield; the level of nutrition at around the time of calving. Immediately after calving a heifer is maternally oriented and service affects pregnancy rates and the management of the heifer at calving the incidence of dystokia and calf

difficult calving. She is, therefore, least able to establish her mortality. It must be emphasized that attention to detail

place in the peck order. Bullying following introduction and good management throughout the rearing period to the herd can be reduced by ensuring that the heifer has regained her strength after calving and by profitability.

allowing her to join the herd in the late afternoon or early evening when there appears to be less aggressive behaviour. Competition for food can be reduced by providing access to 'easy'-feed silage in self-fed systems. It is as well to remember that heifers calving at

References

two years of age are changing their teeth at this time

and find difficulty in extracting silage from well-

Drew, S.B. (1988) The influence of management factors during

consolidated clamps. The problem is obviously most

rearing on the subsequent performance of Friesian heifers.

British Cattle Breeders Digest, **43**, 41–8.

acute on farms where the width of the silage face is

Drew, S.B. & Altman, J.F.B. (1982) The effect of weight at first inadequate or where true 24-hour access is not pro-insemination on the subsequent performance of Friesian

vided. Silage or other forage should be fed in mangers

dairy heifers. *Animal Production*, **34**, 371.

or cut from clamps and fed behind an easy-feed barrier.

Drew, S.B. & Pointer, C.G. (1977) The effect of level of nutri-The newly introduced heifers should be observed care-

tion on fertility in Friesian heifers in autumn and early

fully to ensure that they are maximizing forage and con-

winter. EAAP 28th Annual Meeting, Brussels, 22–27 August

centrate intake. Sufficient loafing areas and loose

1977. Commission on Animal Health and Production Paper

housing or cubicles should be provided for all the cows

77/8, pp. 1–3.

in the herd. There is a tendency for farmers to provide

Foldager, J. & Sejrsen, K. (1982) Nutrition of replacement 5 per cent fewer cubicles than cows in the group on the

heifers affects mammary development and their ability to

assumption that not all the animals will wish to lie down

produce milk. World Congress on Diseases of Cattle, The

Netherlands, vol. I, p. 45.

at the same time. Heifers will not always lie in empty

Furniss, S.J., Kirby, S.P.J. & Smith, G. (1988) The effect of cubicles between older cows and therefore it is recom-dam's parity on the performance of daughters. *British Cattle* mended that one cubicle is provided for each cow and

Breeders Conference Digest, **43**, 49–50.

heifer in the group.

Furniss, S.J., Stroud, A., Barrington, H., Kirby, S.P.J., Wray, J.P.

& Dakin, P. (1986) The effect of dams' parity on first

lactation performance of dairy heifers. *Animal Production*, **42**, 463.

Hafs, H.D., Manns, J.G. & Drew, S.B. (1975) The onset of

oestrus and fertility of dairy heifers and suckled beef cows

Table 5.12

Mean first lactation yields (kg) as percentage of

treated with prostaglandin. *Animal Production*, **21**, 13–28.

mature yields in different management systems.

Keown, J.F. & Everett, R.W. (1986) Effect of days carried calf, days dry and weight of first calf heifers on yield. *Journal of Group*

Mean

Mean

Yield

Heifer yield as

Dairy Science, **69**, 1891–6.

heifer

cow difference

percentage

of

Kossaibati, M.A. & Esslemont, R.J. (1996) *Understanding the yield*

yield

cow yield

Rearing of Dairy Heifers – A Stockman’s Guide. BCVA,

NMR, DAISY, Forte Dodge.

1

5200

6265

-1065

83

Little, W. & Kay, R.M. (1979) The effects of rapid rearing 2

4504

5630

-1126

80

and early calving on the subsequent performance of dairy

3

4274

5479

-1205

78

heifers. *Animal Production*, **29**, 131–42.

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Pirlo, G., Miglior, F. & Speroni, M. (2000) Effect of age at first calving on production traits and on difference between milk
Sejrsen, K. & Foldager, J. (1992) Mammary growth and milk

calving on production traits and on difference between milk

production capacity of replacement heifers in relation to

yield returns and rearing costs in Italian Holsteins. *Journal of Dairy Science*, **73**, 603–8.
concentration and plasma hormone levels . *Acta*

of Dairy Science, **83**, 603–8.

Agricultura Scandinavica Section A. Animal Science, **42**, Sejrsen, K. (1994)

Relationships between nutrition, puberty

99–105.

and mammary development in cattle. *Proceedings of the*

Nutrition Society, **53**, 103–11.

Chapter 6

Tropical Cattle Management

R.D. Fielding and R.W. Matthewman

Introduction

68

the absence of very low temperatures; grass species

Statistics

68

capable of greater energy capture and dry matter yields

The productivity of tropical cattle

68

than temperate grasses; vast land areas un-utilized or

Production strategy

69

underutilized; labour availability, much with strong

Calf management

69

animal keeping traditions; and many locally adapted

Prewaning management

70

breeds that have been selected for production in

Weaning

71

adverse environments.

Post weaning management

71

Onset of puberty

72

Counteracting these advantages are several con-

Monitoring growth

72

straints, which include: high temperatures which

Adult production

72

depress production and food intake; long periods

Grass production

73

without rain when grass growth is impossible; animal

Systems of production

74

diseases, such as trypanosomiasis and those transmitted

Management of dairy cows

77

by ticks; communal grazing systems that inhibit invest-

Management of beef production

78

ment and the use of improved management techniques;

and a lack of effective local demand and infrastructure

to stimulate and support production. These advantages

Introduction

and constraints are discussed in greater detail in the

subsequent sections.

In the following sections an overview is given of tro-

pical cattle management and the tropical production

The productivity of tropical cattle

systems in which cattle are found. Attention is paid to

the main differences between tropical and temperate

The productivity of tropical cattle is sometimes said to

production systems in order to introduce them to the

be low. This is often untrue when total output is considered. The majority of tropical cattle are kept in extensive subsistence systems that differ fundamentally low compared with temperate cattle, but capital inputs are also usually low. Productivity levels, in terms of use of capital as opposed to production levels, may be specialized single product units whilst in the tropics therefore be relatively high.

livestock units are usually multi-purpose with

The multiple outputs of tropical cattle systems include important social as well as commercial objectives.

milk, meat, dung, hides and draught power (Fig. 6.1).

Additional functions include acting as a saving mechanism, as a means of realizing emergency cash, as a means

Statistics

of fulfilling social obligations and as a symbol of wealth

Asia, Africa and South America contain approximately and status within the community. These outputs are not

74 per cent of the world's cattle (Table 6.1) and if other reflected in commonly used measurements of performance such as offtake, which refer only to products and America then over 78 per cent are found in the tropics animals that are sold. Social offtake, barter offtake, sub- and subtropics. The same tropics produce only 59 per cent of the world's beef and veal and 55 per cent of the animals for draught power are not reflected in normal world's milk (Tables 6.2 and 6.3).

offtake measurements. The difference between the This discrepancy represents a challenge for those number of animals sold each year from subsistence involved in tropical cattle management, since the systems and from commercial ranches of 8–10 per cent tropics have many advantages for bovine production. and 18–20 per cent respectively may be explained largely These include: a potential year-round growing season in by these differences in objectives and products.



Tropical Cattle Management • 69

Production strategy

support this view. Intensification and specialization are certainly inhibited by the need for multi-purpose pro-
Some people have argued that the tropics are suited to
duction. However, as development evolves it will prob-
multipurpose low input/low output systems. The envi-
ably promote the type of specialization that has taken
ronmental constraints on high producing animals and
place in temperate areas. The speed of this evolution
the lower quality forages and byproducts of the tropics
will be determined by our ability to counteract the
direct and indirect negative environmental effects on
the production systems and to capitalize on the positive

Table 6.1

World cattle numbers by continent (1000 head).

ones.

Source: Food and Agriculture Organization (1998)

1989–91

%

1998

%

World

1 294 020

1 318 386 (+1.9)

Calf management

Africa

187 534

14.5

217 388 16.5

North and

160 074

12.4

158 195 12.0

In traditional cattle systems, such as pastoralism and

Central America

settled extensive systems, reproductive cycles follow

South America

272 829

21.1

299 947 22.8

seasonal climatic variation. The peak of calving often

Asia

400 563 (FMR)

31.0

450 389 34.2

occurs in the early wet season. In Sudan, in southern

Europe

123 383 (FMR)

9.5

156 212

11.8

Darfur, most calvings occur in April, May and June.

Oceania

31 759

2.5

36 254

2.7

South of the Equator in Zambia the corresponding

USSR

117 877

9.0

—

—

peak occurs in October and November. In humid areas

FMR: formerly; prior to break-up of USSR.

where there is less seasonal nutritional stress, calvings

Number in brackets is the percentage change 1989–91 to 1998.

occur throughout the year.

Table 6.3

World milk production from cows, by continent

(1000 t, whole, fresh). Source: Food and Agriculture Organization **Table 6.2**

World beef and veal production by continent

(1998)

(1000 t). Source: Food and Agriculture Organization (1998)

1989–91

%

1998

%

1989–91

%

1998

%

World

475 154

466 347 (-1.9)

World

52 718

53 695

(+1.9)

Africa

15 221

3.2

17 913

3.8

Africa

3 319

6.3

3 805

7.1

North and

84 146

17.7

91 385

19.6

North and

13 217

25.1

14 710

27.4

Central America

Central America

South America

32 013

6.7

45 325

9.7

South America

9 044

17.2

9 886

18.4

Asia

56 674 (FMR)

11.9

82 871

17.8

Asia

5 269 (FMR)

10.0

10 023

18.7

Europe

167 194 (FMR)

35.2

207 768

44.6

Europe

11 056 (FMR)

21.0

12 726

23.7

Oceania

14 126

3.0

21 086

4.5

Oceania

2 188

4.1

2 546

4.7

USSR

105 779

22.3

—

—

USSR

8 625

16.3

—

—

FMR: formerly; prior to break-up of USSR.

FMR: formerly; prior to break-up of USSR.

Number in brackets is the percentage change 1989–91 to 1998.

Number in brackets is the percentage change 1989–91 to 1998.

Fig. 6.1

Draught cows in Guadaloup.



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Prewaning management

For the first two months the calf should ideally be kept in an individual stall measuring about 2 × 1.5 m. In In pastoral systems, young calves are separated from warmer areas, portable calf pens may be used. The most the grazing herd during the day and kept in special calf

practical indoor system is a shed sited in a gravel or concrete areas. After evening suckling cows and calves may again be separated. In Nigeria, Fulani pastoralists allow calves to suckle briefly in the morning to induce milk. Calves warm in cool weather and cool in warm weather. It may be no more than a simple roof over a concrete base. Slatted-floor houses have advantages, but are more expensive (Fig. 6.2). No walls are necessary in the humid tropics, but adequate overhangs are required in overmilking for human consumption. This is typical of high rainfall areas.

pastoralism in Africa and the Middle East.

Housed calves, if properly managed, tend to perform

Calf management inputs in these systems are

better than calves on pasture. A typical home mixed

minimal and this is reflected in the levels of production.

ration to accompany whole milk is a mixture of 50 per

In arid areas gains may be as low as 200 g/day with

cent ground guinea corn and 50 per cent groundnut

liveweights of 60 kg and 150 kg at 6 and 18 months

cake from two to three weeks of age.

respectively. Calf mortality rates of 15–20 per cent in

The calf cannot utilize average quality tropical forage

pastoral herds and of up to 40 per cent in sedentary

successfully until it is four or five months old. Conse-

herds are normally encountered.

quently, it is desirable to suckle or bucket feed for as

Environmental stress, principally due to high ambient

many months as possible and to feed concentrates

temperatures, is best minimized through correct time of

and high-quality roughage. In intensive systems, the

grazing, use or planting of shade trees and provision of

provision of some good quality forage is beneficial to

simple open houses that offer protection from the

stimulate rumen development.

direct rays of the sun, but ensure maximum air flow.

Major calf diseases in intensive systems include

As production systems improve indoor rearing

scouring (gastroenteritis) (see Chapter 14), pneu-

systems may be adopted. Semi-intensive and intensive

monia (see Chapter 17) and worms (roundworms,

calf rearing systems found in the tropics include the

following.

- Single suckling for beef calves where the objective is to achieve greatest calf growth rates and conversion of milk to meat.

- Dairy ranching where calves are separated at night and the cows are milked once in the morning and limited or restricted suckling is allowed throughout the day. If management will allow evening milking, suckling all night and separating the cow and calf during the day is a better option, as carried out by some pastoralists.

In the tropics calves may be reared inside, outside or a combination of both. Calves can be reared outside all year, though a number of disadvantages are associated with outside rearing on grass paddocks.

- It is difficult to keep grass sufficiently young and nutritious for calves.
- It is difficult to keep calves free from internal parasites if they are grazing in small enclosed areas.
- Environmental stress (temperature and radiation) can be high, even if good shading is available.

The combination of poor nutrition, parasites and climatic stress is a major constraint to calf rearing. Adequate nutrition is the key to success and to enabling a stable host–parasite relationship to be established.

Fig. 6.2

Australian dairy Zebu calves in Malaysia.

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thread-worms, hookworms, lungworms and tapeworms) rainy season, but weight loss frequently occurs in the (see Chapter 19). Suckling systems tend to have lower dry season caused by combined energy and protein mortality rates than bucket feeding systems. The advantages. Of the two, protein is usually the more important. Levels of nitrogen in dry tropical grasses

plete sterilization of buckets and equipment, and commonly fall to around 0.5 per cent, equivalent to 3–4 suckled calves may receive more milk at more frequent per cent digestible crude protein (DCP) in the dry intervals. In herds with many calves to be fed twice a matter. At this level of protein, grass digestibility is low day, maintenance of sanitation is difficult and the incidence of gastrointestinal disorders and indigestion in balance as metabolic faecal nitrogen loss exceeds bucket-fed calves is often high (Chapter 16).

nitrogen intake. Young cattle allowed only poor

Restricted suckling can overcome many problems quality roughages thus tend to develop a ‘bloated’ and is the system commonly practised in pastoral and appearance.

semi-intensive systems. It is also widely used in the Breeds differ in their ability to recover from weight dairy ranching systems of South America. Calves are loss. These differences include the ability to reduce housed separately, either at night or during the day, and

metabolic rate during feed shortage and the efficiency are then allowed to suckle for one minute to achieve of water conservation in periods of water shortage. The let-down before the cows are milked in the morning or latter may lead to increases in feed digestibility due to evening. The cows are fully milked-out and the calves slower rates of passage. The risk with major weight loss, then run with the cows for the rest of the day or night. in excess of 15–20 per cent, is permanent stunting and When properly managed, restricted suckling systems increased disease susceptibility.

have several advantages including better calf growth During ‘normal’ dry seasons deaths are often rare, and health, higher milk yields including more saleable but once the rains begin, losses may occur due to the milk and are associated with reduced mastitis in the stresses of wetting, lower temperatures and highly cows.

succulent grasses causing digestive upsets.

Provided that the dry season weight loss is within the range of tolerance of the breed type, then rearing cattle

Weaning

usually show compensatory growth during the subse-

In many systems calves are naturally weaned when the
quent rainy season and make rapid gains (Hogg, 1991).

milk supply from the cows ceases due to the poorer

Tropical cattle have been naturally selected to cope

nutrition of the dry season. This leaves calves at varying

with recurring periods of undernutrition and then to

liveweights and different degrees of readiness for sur-

compensate effectively. This is a characteristic of great

vitality during the dry period. Where cows continue to

importance which may be threatened by the increased

produce milk, the chances of reconception are reduced,

introduction of exotic genes. The physiological mecha-

often to such an extent that a calving pattern of a calf

nisms that allow compensation involve reduced

every two years develops.

metabolic rate, induced by a period of undernutrition,

In some systems weaning is effected earlier by tradi-

and increased voluntary food intake during realimen-

tional methods. These may include thorns tied to the

tation. The period of realimentation and compensation calf's head or sacks tied around the cows' udders. Dairy is determined by the length of the rainy season. This is heifers in less extensive systems are best weaned at four often short and animals again enter a period of under- to five months of age, depending on the availability of nutrition resulting in further weight loss. A 'zig-zag' alternative feeds of adequate quality. Beef calves pattern of animal growth thus characterizes many tropical should have maximum benefit from the cows' milk and ical rearing systems. Whilst dry season losses can be hence should be weaned as late as seven months. Heifer avoided by supplementation this is rarely justified calves should be weaned early enough to prevent them economically.

conceiving to the bull running with the cows. Avoiding An understanding of compensatory growth is important early conceptions is difficult in many extensive traditional for reasons of rearing economy and in the managerial systems, though in many zebu breeds maturity agement of feeding trials involving measurement of

does not occur until a greater age (i.e. 18 months plus) liveweight gain. Animals that are in a compensation phase when animals have reached approximately 66 per cent of their mature weight.

animals not in a compensation phase.

The consequences of the weight gain/weight loss pattern of growing tropical cattle are several and

Post weaning management

include delayed oestrus, reduced lifetime performance, Once weaned, calves usually encounter a period of sub-optimal nutrition. Reasonable gains can be made in the reduced selection opportunities and also increased susceptibility to disease.

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When rearing stock are housed or confined they may still suffer extended periods of poor nutrition when Castration is carried out in many traditional cattle systems, but is usually left until the animal's potential they are fed adult diets, such as sugar-cane tops or has been assessed. Inferior animals are castrated in

other low-quality byproduct feeds. In pastoral systems order to stop them mating. However, sometimes the greater care is normally given to female stock because 'biggest and best' are castrated to produce draught of their importance in producing calves and milk. Sub-oxen.

optimal management of rearing stock is common in all ruminant systems, tropical or temperate. It is unfortunate that the consequences cannot be better communi-

Monitoring growth

cated to the livestock owners and herders so as to motivate them to improve the management of their In traditional systems, monitoring of liveweight is by rearing stock.

eye, but in more controlled systems regular weighing is desirable. If weighing facilities are not available on the farm, heart-girth measurements can be taken using a

Onset of puberty

weigh-band. Attention should be paid to the breed for

Oestrus

which the weigh-bands were initially designed, and if

possible the accuracy should be checked against actual

In heifers, the period between weaning and calving can

weighings and the bands calibrated appropriately.

be divided into two phases: weaning to first service and

Direct weighing and heart-girths do not always fully

first service to calving. The aim is to achieve optimum

define the state of the animal and condition scoring may

growth with the earliest maturity at the lowest cost. To

be appropriate. Systems of condition scoring for *B.*

achieve this small quantities of concentrate or supple-

taurus cattle (pp. 10, 11) may not be appropriate for *B.*

ment can be a great benefit. A check in growth occurs

indicus breeds, and scoring systems designed for tropi-at weaning, but can be compensated for by the addition

cal cattle should be used (Pullen, 1978; Nicholson and

of concentrates.

Butterworth, 1985). Pullen suggested a score range

The onset of oestrus occurs at a particular weight

of 0–5 from emaciated to fattest while Nicholson and

according to breed. Under good conditions regular

Butterworth suggested a nine-point scoring range.

cycling may be achieved at 13–14 months. In the tropics low nutritive quality of grasses, disease and environmental stresses usually result in delayed oestrus compared with temperate conditions. Whereas *Bos taurus*

Adult production

reach puberty at 30–40 per cent of mature weight, *B.*

indicus reach puberty at around 60 per cent of mature weight. The different tropical cattle production systems are the

weight (Macfarlane and Worrall, 1970).

result of differing levels of dry matter production per

The weight of the young heifer at first service and its

unit area available for grazing. This varies from very

rate of growth up to calving have an important effect

low levels in the arid areas (<1000 kg/ha per annum) to

on milk yield in later lactations. If early calving is com-

high levels (>10 000 kg/ha per annum) in the humid

bined with underfeeding in the rearing stage, heifers

areas, reducing again to low levels where intensive crop

may be permanently stunted and milk production

production limits grazing to roadside verges and

reduced.

intercrop areas.

Once heifers are cycling normally and have achieved
Grazing utilization is often complicated by land
an adequate body weight, mating or service should be
ownership, which includes 'ownership' by nation, tribe,
timed to ensure calving at the optimum time of the year.
group or individual. Most grazing is communal and
This is often assumed to be the beginning of the rains,
herders have the right to graze as many animals as they
but it may be better to aim for calving just before the
wish. Individual ownership and/or leasing of grazing
rainy season. This will allow the calves to start grazing
land are increasing and this may stimulate intensifica-
during the rainy season before the quality of the
tion and improvement. Much depends on the availabil-
herbage falls and as the dry season approaches.

ity of employment opportunities for those who are
unsuccessful when land rights are allocated. If such
opportunities are not available enforcement of land

Sperm production

ownership is difficult and social pressure tends to result

The onset of sperm production is not as critical as the
in the continuation of communal grazing.

onset of oestrus. Only where conditions are very poor

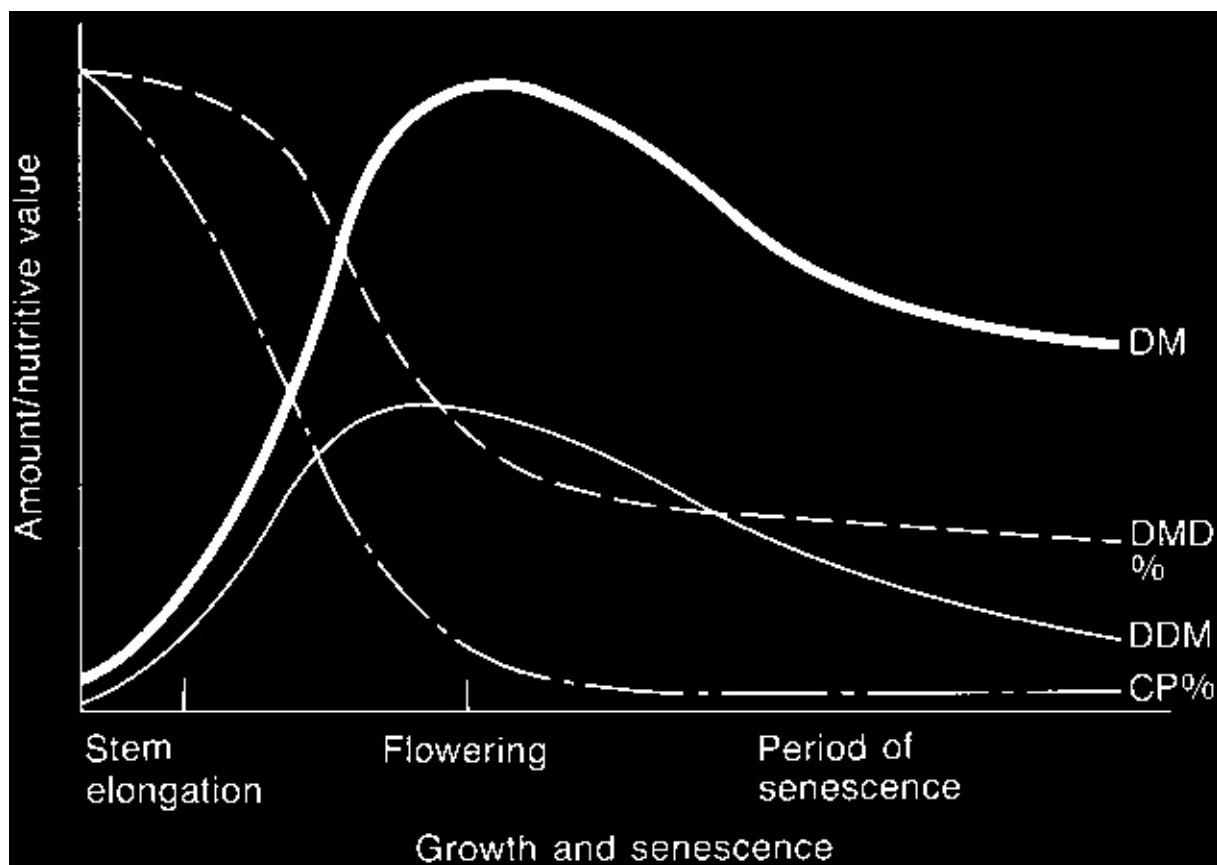
Since grazing is crucial in cattle nutrition, the follow-

and liveweights low is the onset of sperm production

ing sections highlight important aspects of grassland

delayed.

production.



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Grass production

Grass represents the major link between the sun as the ultimate source of energy and the grazing animal. For the grass, however, the grazing animal can be regarded as a parasite. It is thus important that this relationship is not a destructive one for the grass. Animal productivity depends on a vigorous grass sward and management must safeguard the viability of the herbage above all else.

The factors that determine grassland dry matter productions are amount of light, leaf area, efficiency of species, amount of carbon dioxide, temperature, water and nutrients.

In the short term the manager can determine leaf

Fig. 6.3

Generalized changes in the amount and nutritive value area and nutrient level and in the longer term the of herbage during growth and senescence of the herbage. DM, species and possibly water level, through run-off dry matter available for grazing – determined by species and control. Many tropical grasses are physiologically dif- available nutrients; DMD, dry matter digestibility – falls from over ferent from

temperate grasses. They fix carbon as C₄

65% to around 40% largely as a result of changes in the crude

rather than C₃ compounds and are regarded as biolog-

protein level; DDM, available digestible dry matter – the result-ically more efficient than temperate grasses. This is

ant of DM and DMD; CP, crude protein – falls from over 12% to

partly the result of the structure of tropical grasses,

around 2%.

which are taller and more effective in sunlight

interception.

Tropical grasses have evolved to survive short rainy

seasons. They grow quickly, reproduce and senesce.

that can be visited annually to assess the degree of soil

Whilst grass breeders have extended the growing

erosion and ground cover. The botanical species are

period of many species, the short life cycle of grasses is

identified and the increase or decrease in particular

still a problem when grazing animals require dry matter

species is recorded. Botanical trend monitoring is

throughout the year.

appropriate for stable ecosystems where changes occur

Figure 6.3 illustrates the changes in the nutrient only slowly. In arid and semi-arid areas with variable composition of grasses with time.

rainfall changes can occur relatively quickly. When The fall-off in crude protein (CP) percentages rains are adequate and grazing pressure reduced areas reduces the rate of degradation of dry matter and rate that appeared seriously overgrazed can recover dra- of passage and thereby dry matter intake. While matically through the germination of seeds of annual growing conditions are good in the wet season, CP grasses (Behnke *et al.* , 1993).

levels can be as high as 12–14 per cent, but can be as low as 2–3 per cent in the dry season. The aim of the *Management to maximize voluntary food intake* manager is to counteract low dry matter intake by appropriate management measures as discussed below. Maximization of voluntary food intake is the underlying objective in ruminant feeding management.

Range monitoring

Tropical grasses are less digestible at the same stage of

growth than temperate grasses, due to greater lignifica-

The interaction between animals and range is con-
tion. The lower digestibility reduces rate of passage and
stantly changing and the result may be a long-term
hence reduces food intake. Some breeds of tropical
improvement or deterioration. A knowledge of the
cattle may have evolved to cope with poor quality feeds
direction of this trend is very important and regular
through a greater dry matter intake capacity per unit
observation (monitoring) should be carried out using
body weight than temperate cattle.

the following methods.

Management strategies to maximize voluntary intake
involve maximizing food production, avoiding food

Remote sensing: This involves the use of satellites or losses and stimulating
intake as follows.

aircraft to determine land use, land potential and
changes in herbage production over time.

(1)

Provision of adequate water and water harvest-
ing through the use of pounds and micro terraces.

Soil and botanical trend monitoring: This involves

(2)

Control of grazing time, intensity and frequency
establishing specific points or transects on the range
to maximize grass production, i.e. maintenance of



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a sustainable carrying capacity according to the

Systems of production

manager's objectives.

Management and production levels are determined by

(3)

Inclusion of legumes in the sward or as browse.

factors such as the herder's objectives, climate and

(4)

Fire control to prevent destruction of accumu-

land tenure. In communally owned dry areas that are

lated standing hay in the dry season and in

marginal or exclude crops pastoral livestock systems

ranching systems to control bush encroachment.

are found with cattle (Fig. 6.4), sheep, goats and camels.

(5)

Exclusion of wildlife and non-authorized grazers.

In wetter areas small-scale mixed farming systems

(6)

Provision of minerals and, in the long term,

have developed where sheep and goats are important

correction of soil mineral deficiencies.

and where cattle and buffaloes may have a role for

(7)

Simple rotation and grazing by appropriate milk, meat, dung and draught power. In tropical high-herding, or use of fencing under intensive land areas where the climate is less severe milk production systems.

duction is often based on exotic cross cattle (Fig. 6.5).

(8)

Division of the herd into units of need, i.e. young

Where land is privately owned, particularly in South stock, dry cows, lactating cows.

America, more intensive dairy farming methods

(9)

Good herding practices, including choice of area occur in combination with large-scale dairy and beef to be grazed and duration of grazing.

ranching.

(10)

Routine health maintenance.

Fig. 6.4

Fulani cattle in Nigeria.

Fig. 6.5

Sahiwal ¥ Friesian cattle in

Malaysia.

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Pastoralism and settled extensive systems

Milk production

of production

Milk is produced under a variety of systems, ranging from one-cow zero-grazed units in Mauritius where Low and unpredictable rainfall coupled with communal cows are fed on sugar-cane tops and roadside grasses, land-use rights have led to migratory and seasonal patterns of grazing. Such systems are found throughout the South America, intensive large-scale milk cooperatives Sahel from Mauritania to Sudan and through Ethiopia, in India, and very large, zero-grazed, environmentally Somalia, Kenya, Uganda and Tanzania. Cattle owners controlled exotic cow units in Saudi Arabia.

include the Fulani (Fulbe), Tuareg, Dinka, Borana,

Consumers in the tropics accord a high value to milk

Karamajong and Masai. Pastoralists keep cattle to meet

as a human food. The rate of increase in milk production has been considerably greater in tropical countries than in traditional milk producing and exporting areas. Due to recurring droughts and increased population pressure resulting in loss of traditional grazing areas, these patterns are changing in many areas.

over the same period (FAO, 1998). Milk production can be an important enterprise in rural development for pastoralists are being forced to settle and engage in agriculture or some other activity.

several reasons:

Settled production occurs where cattle are combined with crop production. In Nigeria the term 'mixed

- it provides a year-round source of income for the farming' has been used to denote the use of cattle as

farmer;

draught animals for cultivation. Draught animals are

- it contributes to family nutrition;

common throughout the tropics and because of their

- it can utilize crop byproducts;

vital role in the production of subsistence crops such as

- it serves to transfer money from urban to rural

maize, sorghum, millet or rice their management is

areas; and

often better than that of cattle which are not used for

- it creates rural employment opportunities.

draught.

These advantages have led to many development ini-

Most settled farming is subsistence in nature and

tatives involving local/exotic crossbreds and sometimes

except where used for draught, livestock have a sec-

pure exotics. There have been many disasters and fail-

ondary role to crop farming. Management inputs are

ures, but also some notable successes as in India with

often relatively low. Land is seldom individually owned

Operation Flood, and in Kenya and Uganda with

and communal grazing is a major method of food smallholder milk production schemes.

supply. The management and improvement of commu-

The major challenges for milk producers in the

nally owned land are limited by the difficulties associ- tropics include:

ated with getting people to cooperate.

Food inputs are natural pasture, browse, crop

- achieving a high intake of good quality food

residues and supplements. Crop residue grazing is sea- throughout the year;

sonal, but important for extensively managed cattle.

- coping with disease challenges;

Millet and sorghum residues provide early dry-season

- storage and transporting a bulky and perishable

food and may be stored in stacks for use in the dry commodity.

season, although they are often grazed off quickly in a

‘free for all’.

Extensive systems: dairy ranching: Dairy ranching is an One technology that has been proposed for improv-extensive system of milk and calf production particu-

ing the system as a whole is that of fodder banks.

larly associated with South America, and countries such

Fodder banks are fenced areas of high-quality forage

as Bolivia and Columbia. It is a flexible, dual purpose,

that are made available for grazing for only a few hours

low input low output system that depends for its success

per day and for only the most needy stock. The estab-

on the plentiful availability of low cost land and labour.

lishment of fodder banks has been described by Otsyina

Dairy ranching occurs particularly in those areas where

et al. (1987) using Stylosanthes species such as S. guia-conditions are hot and humid. There is enough mois-

nensis cv. Cook and S. hamata cv. Verano. Fodder banks ture to produce sufficient food throughout the year but

have had limited success as has the oversowing of

the temperature is too high to permit specialized high

rangeland with legumes. The latter depends for success

output milk production.

on the control of grazing, which with extensive grazing

A wide range of genotypes are used in dairy ranch-

is virtually impossible.

ing. Crossbreeds between Bos indicus and Bos taurus are

commonly encountered. In the more favourable environments high grade cattle are used whilst in the more difficult areas use is made of pure *Bos indicus* types. (Waters-Bayer, 1995). Urban livestock farming, and dairying in particular, has largely developed without outside involvement in response to people's needs for income. Some farmers milk their cattle twice a day whilst others milk their cattle only once a day. Some depend entirely on grazing whilst others use concentrates. In most cases they are undertakings that have usually been ignored by traditional ministries and organizations. However, if the presence of the calf is used to stimulate milk let-down and one or more quarters are left for suckling by the calf after milking. The calves are allowed only policies to limit and perhaps relocate urban dairying

limited access to the cows. However, much depends on and livestock production are urgently needed.

the relative prices of meat and milk. If meat is in high demand then the calf may be allowed more milk in

Semi-intensive and intensive dairy production: In South order to promote growth. If the price of fresh milk is

America, exotic breeds are used intensively in highland high then the amount allowed for the calf may be areas. Highland areas are found throughout South severely restricted, which sometimes leads to increased America and also in Africa on the Jos Plateau in calf mortality.

Nigeria, the Kenyan and Ethiopian Highlands and in The total milk production from dairy ranching in parts of Tanzania such as Arusha.

South America is considerable and may represent half of the total milk consumed in a country such as Colom-

Kenya Highlands: The smallholder dairy industry in bia. One of the great advantages of dairy ranching is Kenya has grown impressively over the last 25 years. its flexibility and the regular income from milk sales

There are now over 400 000 smallholder dairy farmers that it offers. It has been adopted as a system by producing 70 per cent of milk sold. It is estimated that many farmers throughout South America and clearly there are over 3 million dairy cattle in Kenya, concentrated for the most part in peri-urban areas. The majority include some amount of Friesian in their genetic

Smallholder dairying: the Indian model: India is famous make-up, but other breeds including Sahiwal, Brown

for its Anand pattern of dairy development. It is the Swiss, Ayrshire and Channel Islands have a significant fourth highest milk producer in the world after role (Fig. 6.6). The pure exotics are found in the high Europe, the Russian Federation and the USA and since potential areas in the highlands where lower temperatures and more dependable rains and food supplies involved the setting up of dairy cooperative societies make them a sustainable option. Dairy crosses with which all milk producers are eligible to join. Farmers

local Zebus are found in the harsher lower potential are usually paid for their milk within 12 hours of delivery environments.

ery to a collection point. Payment is based on quality

In the most fertile highland areas the production and quantity.

system is based on zero-grazing of Napier grass (Pen-

The societies are formed into milk unions, which

nisetum pupureum) together with the use of roadside

provide collection, processing and marketing ser-

grasses and available byproducts such as maize stover,

vices, and may market livestock feed and provide

banana stems and sweet potato vines. The use of forage

artificial insemination (AI), veterinary and other

and tree legumes is being encouraged. So intensive is

services.

the system that 29 per cent of smallholders buy forage

to feed their cows at some time during the year. Com-

Urban dairying in Africa: Recent years have seen a

mercial concentrates are widely used. Grazing systems

huge increase in urban dairying in and around cities and

of varying intensity are encountered in less fertile areas. major towns throughout the tropics. Whilst such developments provide a regular income for producers and grass (*Pennisetum clandestinum*) and Rhodes grass readily available fresh milk for the consumers they (*Chloris gayana*).

are also associated with a number of problems. These Yields of around 2500 kg per 10-month lactation include urban pollution due to waste accumulation and period are commonly obtained. The median calving undesirable smells and noises. Disposal of the carcasses interval amongst the better smallholder herds has been of dead animals is a further problem as are the various estimated at 426 days. Fifty per cent of herds use artificial threats to human health. The latter arise directly from cial insemination with an approximate success rate of the consumption of milk and meat containing organ- 50 per cent. Zero-grazed systems do not lend themselves that cause diseases, such as tuberculosis and brucellosis to easy heat detection and this is an area for

cellosis, or indirectly from the increased activities of continuing extension inputs and training.



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Fig. 6.6

Sahiwal and Sahiwal × Friesian cattle in Kenya.

East coast fever (pp. 750–53), transmitted by ticks, is Beef may remain a byproduct of milk and draught the biggest threat to exotic breeds, although zero-power in Africa and Asia for the foreseeable future. It grazing represents a significant tick avoidance strategy. is appropriate therefore to focus research on ways of Smallholders are increasingly adopting strategic hand-feeding and managing culled cows and oxen so as to spraying methods of tick control rather than using gov-

maximize their value as meat animals.

ernment dips.

In South America the situation is different and spe-

Many smallholder households in Kenya have bene-

cialized high quality ranch beef from extensive systems

fited both nutritionally and financially from their dairy-

has found a ready market in America, Europe and the

ing enterprises. Perhaps regrettably, intensification and

Far East for many years.

commercialization of milk production tend to be asso-

There are indications that the demand for meat is

ciated with the control of income from milk moving

growing rapidly in urban areas of the tropics. This rep-

from the women to the men. Nevertheless, it is hoped

resents a tremendous opportunity for beef producers,

that the accumulated expertise and improved tech-

large and small, in tropical areas (Delgado et al. , 1999).

nologies will allow the continuing expansion of small-

holder dairying into the low potential areas of the

Management of dairy cows

country (Reynolds et al. , 1996).

The general objectives of management can be assumed to be broadly the same for all types of dairy cattle.

Beef production

Ideally, dairy cows should produce a calf every year, In Africa and Asia beef has traditionally been a byproduct since milk production is usually maximized with a 305-day lactation and draught power production. Governments have often been disappointed with the level of calving intervals which vary between 275 and 287 days, it is necessary for the beef production from their traditional herds. This has led to the mounting of many projects aimed at increasing service should be at about 50 days after calving. This has led to the mounting of many projects aimed at increasing beef output. These have included group ranching which is seldom achieved in tropical systems, and calving schemes as in Kenya and parastatal ranching initiatives with calving intervals of up to 500 days occur. The main reason is as in Uganda and several other countries. Most of these delayed return to oestrus due to poor nutrition, suckling calves, and other factors. Such projects have ultimately proved uneconomic, although

ling and other stresses including those of climate and private commercial ranches have usually been more disease.

successful.

Artificial insemination is not widespread in the

Increased beef production from medium- to large-

tropics, but in some countries, such as Kenya and

scale intensive feedlots has also been attempted with

Pakistan, insemination services have been operational

the aim of drawing immature stock from the rural areas

for many years. The benefits of genetic improvement

for finishing to produce improved quality beef. These

from AI are constrained by the problems of detecting

initiatives have not usually been successful and small-

oestrus, which are especially acute in smallholder one

scale finishing schemes are proving a more successful

cow units. The achievement of optimum breeding

form of intensification.

patterns and milk production also depends on the

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provision of suitable environmental conditions and

Table 6.4

Typical rations for dairy cows using combinations of proper feeding.

feedstuffs. BF = butterfat.

A major objective of the management of dairy cows in the tropics should be the reduction or the avoidance

Feedstuff

Diet (kg fresh weight)

of environmental heat load. This can be achieved in a

360 kg cow

540 kg cow

number of ways:

- *Where possible animals should be grazed at night.*

Diet 1

Diet 2

- *Concentrates should be fed early in the morning*

Legume hay (average)

1

1.6

and late in the afternoon.

Maize silage (average)

11

16

Grass hay (mature)

2

3.9

- *Adequate water should be provided.*
- *Natural and constructed shade should be provided.*

Diet 3

Diet 4

- *Buildings and shelters should be made of appropri-*

Legume hay (average)

4

4

ate materials.

Maize silage (average)

18

27

- *Houses and yards should be sited so as to take full*

Maintenance + 4 litres

(4.5% BF)

(3.5% BF)

advantage of prevailing winds.

- *Calving should occur in the coolest season, if nutri-*

Diet 5

Diet 6

tion allows.

Green lucerne (early flower)

11

3

- *Animals should be managed indoors wherever*

Grass hay (average)

3.5

18

possible, so as to reduce muscular work.

Maize silage (average)

9

4.5

- *Animals should be sprayed with water during the*

Maintenance + 4 litres

(4.5% BF)

(3.5% BF)

hottest periods to facilitate cooling.

The effect of adverse tropical environmental conditions can be overcome to a large extent if cost is not a constraint. In the Middle East, exotic breeds of dairy cattle are maintained in intensive systems and can throughout lactation and optimum levels of fertility so achieve yields in excess of 8000 l/cow per year (Chapter 8, 71).

tion it is necessary to rear a healthy calf to weaning and to first calving. Attention must be given to a number of Feeding (see also Chapter 9) disease and general health problems.

Dairy cows require higher levels of feeding than Tick-borne infections, trypanosomiasis (Chapter 45), growing or working animals. Energy inputs for lactation helminths and liver fluke (Chapter 19) are major problems in many areas, as is streptothricosis (see p. 886) though in the tropics requirements for lactation will in parts of Africa. Exotic animals are particularly not usually exceed maintenance. For a 300 kg cow pro-

susceptible to many of the endemic diseases.

ducing 1000 l in a 300-day lactation, average daily

Adequate attention to disease prevention is of

metabolisable energy (ME) requirements will be

primary importance in the tropics, where veterinary

approximately 30 MJ for maintenance and 15–20 MJ

services are often unavailable (Chapter 69).

for lactation depending on the butterfat (BF) content

In hot climates it is better to milk at the cooler times

of the milk. Such levels of milk production can be

of day. Milk cooling can present problems where con-

achieved by a number of rations and feeds can be com-

stant electricity supplies are not present. Water supplies

bined according to their degradability, ME content and

to local dairies are often poor and hygiene may be

their CP content. Some typical dairy rations used in

neglected.

southern Africa are shown in Table 6.4.

Other rations can be devised for different tropical

regions based on a knowledge of locally available foods

such as groundnut cake, wheat bran, maize, oats/barley,

Management of beef production

green grain, tapioca, rice bran, urea, molasses and cottonseed.

Ranching

In commercial ranching systems both land and animals

Health and hygiene

are under the control of the same active management.

The two prime considerations of dairy cow health are

This contrasts with pastoral systems where land is

the need to maintain optimum milk production

communally owned but rarely communally managed.

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Table 6.5

Example of routine management programme for an East African cattle ranch.

Month

Approximate monthly

Operation

rainfall (mm)

December/January/February

110

Calving; ear tagging,

weighing and recording

February

100

*Check breeding bulls and
cows; cull and replace*

March

160

*Improved nutrition for
breeding bulls and
cows (bulls throughout
mating season)*

March/April/May

140

*Brand, castrate, dehorn
last year's calves*

April/May/June

110

*Join breeding bulls to
herd of breeding cows*

*Sell marketable stock;
steers, surplus heifers,*

culls

May/June

60

Supplementation of

lactating cows

Conserve forage

July

10

Remove bulls from

breeding herd

July/August/September

10

Wean

August

10

Pregnancy diagnosis;

remove non-pregnant

stock

Under some circumstances a system with two 70-day breeding seasons at four month intervals can be applied and may improve annual calving percentage and economic returns.

Animals are owned by individuals or groups of indi-

important husbandry tasks are designated to be carried out by individuals who try to maximize rather than optimize their output at particular times during the year. All concerned with the use of the communal land.

then know what should be done and when it should be done. Ranching can operate throughout a range of intensities, from extensive to intensive. Table 6.5 shows an example of a routine system of activities. Extensive systems may involve a one-herd programme for a cattle ranch in East Africa. The programme of activities is determined by seasonal feed availability in the cattle. Intensification may involve one or more of the following stages.

Government and parastatal companies have often involved themselves in ranching to intensify cattle production away from subsistence systems. The attempts

- *Water development, e.g. boreholes or small dams.*

have rarely been successful. The reasons have usually

- *Routine health care.*

have rarely been successful. The reasons have usually

- *Mineral supplementation.*

been economic, arising from indifferent managements,

- *Construction of handling and dipping facilities.*

low meat prices and the high costs of intensification,

- *Correct use of fire, e.g. creation of firebreaks.*

which have rarely been justified. Most successful

- *Multiple herd systems, e.g. young stock, steer herds,*
ranching is in the purely commercial sector.

etc.

- *Protein supplementation.*
- *Fencing, e.g. perimeter and paddock.*
- *Forage conservation.*

Supplementation

An important aspect of ranch management is

*Tropical cattle commonly suffer a period of weight
organizing routines. A routine approach means that the
loss during the dry season. Compensatory growth*

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can overcome the worst effects, but feed supple-

*Drought: Drought conditions represent a special case
mentation may be feasible where it can be controlled.*

for supplementation where survival is the objective. An

The practical approach should include the use of early difficulty is establishing that drought, as opposed sustainable stocking rates, fire control, deferred to a long dry season, exists. When the fact is established grazing, seasonal control of breeding and tactical a number of options are available: (i) do nothing; marketing. Once supplementation is accepted as (ii) sell some stock and buy feed; (iii) sell all stock and a workable option two considerations arise: the restock later; or (iv) move animals to an area with nature of the supplement and the stock to be grazing.

supplemented.

Where supplementation is adopted it is important not to start supplementing too soon, but to allow animals to lose some weight to reduce their maintenance requirements. Bovines have adaptive mechanisms for dealing with drought, one of which involves a as a substitute for a part of that diet. The effective uti-

reduction in metabolic rate. Such mechanisms should
lization of nitrogen supplementation in the form of urea
be activated prior to survival supplementation.

mixed in a carrier such as molasses depends upon there

Animals at greatest risk and females required to

being a deficiency of degradable protein in the rumen.

continue the herd after the drought should be given

This is not always the case and the response to urea sup-

priority for drought feeding. Once a decision has

plementation has not always been as predictable as

been taken to provide survival feeding the feedstuff

anticipated.

is usually determined by cost and availability.

Other forms of background supplement such as min-

Infrequent feeding of large amounts is probably pre-

erals are important over the long term, but have no

ferred so as to ensure every targeted animal gets some

effect on reducing dry-season weight losses. The pos-

feed.

sible exception is salt, which may serve to stimulate the

intake of poor-quality roughage. In Nigeria, a naturally

occurring and renewable mineral supplement known as

Kanwa is fed to both settled and transhumant cattle

Feedlots

owned by Fulani and agropastoralists. Kanwa contains

Feedlots have been set up in different forms and for

1.5 per cent Na, 4.7 per cent K, 23.7 per cent Ca, 0.6 per

a wide variety of reasons; as large- or small-scale

cent P and trace amounts of Mg, Fe, Mn, Cu, Co and

units, as commercial or government sponsored ven-

Zn.

tures to produce export quality carcasses and as a

Increased attention is being paid to the potential of

means of trying to reduce overgrazing of com-

small amounts of highly digestible cellulose as a sup-

munal areas. Initial developments tended to follow

plement in the form of legume or browse material. Such

the large-scale pattern of North American feedlots,

material can stimulate cellulolytic bacteria and enable

but it is now recognized that small-scale projects

an improved utilization of a low quality diet. Browse

that demand more labour than capital are more

usually contains over 10 per cent CP/unit DM and sustainable.

can often have as high as 25 per cent CP/unit DM.

Certain circumstances are required before intensifi-

*It thus forms a good supplement to poor-quality
cation becomes an economic strategy, as follows.
roughage.*

Other forms of supplement in Nigeria include cot-

- Beef is one of the most expensive meats within the
tonseed cake, groundnut tops, cow pea stalks, ground-
country.*

nut cake and also salt, chaff, bran, cut branches and cut

- Animals are economically available for feeding on
grass. In Sudan some livestock owners in the Gezira
a continuing basis.*

area provide daily supplement mixtures containing

- Above average quality feeds are economically
cottonseed cake, groundnut cake, bran and molasses.
available on a continuing basis.*

Where breweries are nearby, brewers' grains may also

- Adequate initial funding is available.*

be available.

- *Infrastructural factors such as transport, communications, husbandry and health services are available*

Animals to supplement: Not all animals have the same and adequate.

need for supplementation and optimum use of

- *There is an absence of restrictive factors, e.g. resources can be achieved by identifying an order of butcher cartels or legal restrictions on export.*

priority, e.g. weaned calves, late pregnancy/early lactation cows, breeding heifers. Lowest priority will

A failure to satisfy these requirements has led to the normally be given to growing steers.

limited success of many feedlots.



Fig. 6.7

*Stall-fed fattening of Fulani cattle
in Nigeria.*

Smallholder fattening

*success in Kano State where work bulls after 2–3 years
are stall-fattened for 6–8 months and sold to butchers.*

*In Malawi a smallholder fattening project was started
as long ago as 1957 and has now expanded to other
parts of the country. It has proved to have many bene-
fits, as follows.*

References

- *Home-grown beef for the local market saving
foreign exchange.*

Behnke, R.E., Scoone, I. & Kerven, C. (eds) (1993) Range

- *A means of saving for farmers, which can be used
ecology at disequilibrium: new models of natural variability
to invest in crop production.*

*and pastoral adaptation in African savannas. Overseas
Development Institute (ODI), London.*

- *The provision of manure for the maintenance of soil*

Delgado, C., Rosegrant, M., Steinfeld, H., Ehui, S. & Courbois, fertility.

C. (1999) Livestock to 2020: the next food revolution. Brief

- *Stabilization of the farming system by providing an*

61. International Food Policy Research Institute (IFPRI),

employment opportunity.

2033 K Street, Washington.

The participating farmer must build a suitable stall

Food and Agricultural Organization (1998) Production Year-

book 52, pp. 186–8, 195–7, 212–14.

for one or two animals. Several farmers commonly

Hogg, B.W. (1991) Compensatory growth in ruminants. In

build their stalls together. Farmers receive a credit steer

Growth Regulation in Farm Animals (ed. by A.M. Pearson of 260–280 kg with the assistance of the extension serv-

& T.R. Dutson), pp. 103–34. Elsevier Applied Science,

ices. This is fed on maize stover, groundnut haulms and

London.

maize bran during the dry season and cut-and-carry

Macfarlane, J.S. & Worrall, K. (1970) Observations on the

*Napier grass (*Pennisetum purpureum*) and/or Rhodes*

*occurrence of puberty in *Bos indicus* heifers. East African grass (*Chloris gayana*) during the rainy season. Average Agricultural and Forestry Journal, 35,*

409–10.

daily gain is about 0.5 kg and with approximately 200

Nicholson, M.J. & Butterworth, M.H. (1985) *A Guide to*

days feeding total gains of 100 kg are achieved pro-

Condition Scoring of Zebu Cattle, International Livestock ducing a final weight of 360–380 kg.

Centre for Africa, Addis Ababa, Ethiopia, pp. 3–30.

In Nigeria, stall feeding and fattening is also carried

Otsyina, R.M., von Kaufman, R.R., Mohamed Saleem, M.A.

& Suleiman, H. (1987) Manual on Fodder Bank Establish-

out and is a common method of cattle production in the

ment and Managment, International Livestock Centre for subhumid zone and northern savannah areas (Fig. 6.7).

Africa, Addis Ababa, Ethiopia, pp. 8–12.

Farmers often keep up to six animals, usually young

Pullen, N.B. (1978) Condition scoring of White Fulani Cattle.

bulls, for 18–24 months before selling them to urban

Tropical Animal Health and Production, 10, 118–20.

traders. This method of production has been the subject

Reynolds, L., Metz, T. & Kiptarus, J. (1996) Smallholder

of a major extension exercise by the National Livestock

dairy production in Kenya. World Animal Review 87, Projects Department in

Nigeria, and has been of some

66–73.

82 • Chapter 6

Waters-Bayer, A. (1995) Living with livestock in town: urban

(ed. by K.H. Zessin), pp. 121–32. Deutsche Stiftung für

animal husbandry and human welfare. In Livestock Pro-

Internationale Entwicklung, Zentralstelle für Ernährung

duction and Diseases in the Tropics: Livestock Production

und Landwirtschaft.

and Human Welfare: Proceedings of the VIII International

Wilson, R.T. (1995) Livestock Production Systems, pp. 1–141.

*Conference of Institutions of Tropical Veterinary Medicine MacMillan
Education, London.*

Chapter 7

Ethnoveterinary Medicine

in the Tropics

R.D. Fielding

Introduction

83

McCorkle, 1989; Bizimana, 1994; IIRR, 1994), including

Definition and examples of nature of EVM

83

various internet sites. Some examples of EVM from

Why ethnoveterinary medicine?

83

these and other sources are presented below.

Advantages and disadvantages of EVM

84

The majority of ethnoveterinary practices are aimed

The way forward – validation?

84

at relatively chronic conditions such as internal and

Discussion

85

external parasites, digestive disorders such as inap-

Conclusions

86

petance, constipation, diarrhoea and bloat, wounds,

lameness, non-specific ‘fevers’ and ‘coughs and colds’.

In Ethiopia goatkeepers boil the leaves of the castor-

Introduction

oil plant (Ricinus communis) to provide a viscous liquid which they use to

control mange in their goats. The

Over the last 30 to 40 years most 'top-down' rural development projects in the tropics have continued to have provides an example of an EVM agent that must be disappointing results. In response to this, and prompted handled with care (Peacock, 1996).

largely by social scientists, there has been a growing In many Mediterranean countries honey is used on awareness that an appreciation of rural people's local wounds to promote healing (personal observation). The knowledge should be the starting point for discussing mode of action is believed to be partly achieved development interventions of any kind. This participatory 'bottom-up' approach is now being applied across wound. This fluid serves to 'flush out' dirt and other all areas of natural resource use in the tropics. As contaminants and thus promotes healing.

regards the maintenance of health amongst the live- There are many plants which appear to have some

stock resource it is Constance McCorkle, an anthropol-

anthelmintic effect: *Artemisia maritima*, *Caesalpinia ogist*, and Evelyn Mathius-Mundy, a veterinarian, who

crista,

Melia azedarach,

Mallotus philippinensis,

have done most in the last 15 years or so to bring atten-

Chrysanthemum spp., *Matteuccia orientalis*, *Carica* tion to that local knowledge in the tropics which deals

papaya, *Heracleum spp.*, *Hedysarum coronarium*, Aloe with animal disease treatment. This body of knowledge

barteri, *Terminalia avicennioides* and *Diospyros mollis* is now commonly referred to as ethnoveterinary medi-

(Hammond et al., 1997). Firing and bloodletting

cine (EVM). The objective of this chapter is to highlight

are widely used practices to treat problems such as

the key issues and developments in EVM in the tropics.

lameness and inflammation. These practices are most

commonly used by owners of horses and donkeys.

Definition and examples of

nature of EVM

Why ethnoveterinary medicine?

For the purposes of this chapter ethnoveterinary med-

In addition to the increasing appreciation of local medicine is defined simply as the traditional treatments and knowledge as explained above there are several other practices that livestock keepers are using now, other reasons which have contributed to the recent growth in than modern synthetic drugs. The majority of the treatment interest in EVM at farm, local, national and international levels. Some EVM involves magico-

religious practices. Although these latter practices can Antibiotics, acaricides and anthelmintics can be be very important to those who use them, they are not dramatically effective when affordable and correctly discussed here.

used. However, the rising cost of these medicines and There is an increasing amount of literature listing its consequences is now a major issue. If the cost of and describing EVM practices (Mathius-Mundy & a treatment is a significant proportion of the value

of the animal that is being treated then one or more per cent fewer Haemonchus contortus in the treated things may happen:

group as compared to the control ($p < 0.05$).

- It is freely available or at a cost in proportion to the*
- The animal is left untreated or a low cost EVM value of the animal.*

method is used.

- It is easily administered, usually topically or*
- The seller of the chemical dilutes it to make orally.*

it cheaper and so that it will 'go further'. Monteiro

- EVM relieves the worry and concern of owners et al. (1998) in Kenya found that of seven of sick animals in that it makes it possible to do anthelmintics marketed as containing levamisole, something, effective or not, for their animals.*

an effective anthelmintic agent, two contained

However, EVM is not without disadvantages and none, whilst two others had levels of levamisole of limitations:

11.8 per cent and 78.7 per cent of the amount stated on the label.

- *Particular methods are often geographically local-*
- *Livestock owners who buy costly drugs may also try*
ized and the scope for their further dissemination is
to make them 'go further' by diluting them, by
limited.

underdosing or by not completing the full recom-

- *Cures are variable in their effectiveness according*
mended course of treatment. All abuses of drugs
to season, method of preparation, etc., and few if
are associated with incomplete cures and with the
any have been validated in the same way in which
promotion of organism resistance.

synthetic drugs must be validated.

- *From a technical standpoint some are totally*
Even when properly administered the long-term
ineffective.

regular use of drugs and chemicals such as

- *EVM has little to offer against the acute viral*
anthelmintics and acaricides leads to the loss of an

diseases of animals, other than treatments for the animal's natural resistance. If for whatever reason the signs.

anthelmintics/acaricides are suddenly unavailable the

- EVM is not always practical on a large scale. A animal is totally exposed to the worst effects of the particular EVM method may require considerable asites and organisms which these drugs are keeping amounts of leaves, seeds or even roots. Identifying under control. For this reason the routine use of acaricides, for example, has been questioned for some time destruction. In Nepal people have been excluded (Norval, 1983).

from some forests because of the excessive

Farmers in the tropics rarely have any awareness of collection of medicinal plants, albeit they were for the environmental damage and pollution that may be use in human medicine.

caused by the incorrect disposal of chemicals and/or the

In the new millenium the major arguments for giving

associated packaging.

greater attention to EVM include the following:

Globally there is a growing interest in ‘alternative’ and ‘sustainable’ approaches to disease treatment. In

- It is what livestock keepers use now and, by definition therefore, should be the starting point for any such as herbalism, acupuncture and homeopathy, are animal health intervention.*

now taken seriously. Veterinary medicine is currently

- It would be unwise to rely on only one strategy evolving, in a similar way (see Chapter 65).*

for disease control, i.e. modern drugs, when the strategy has limitations, some of which have been highlighted above.

- In the long term the ‘official’ recognition of EVM*

Advantages and disadvantages

empowers those who practise it, with potential

of EVM

benefits for increased participation in other areas of social and economic development.

The advantages of EVM are as follows:

- *Livestock keepers are already familiar with it, it is*

The way forward – validation?

what they use now.

- *A significant part of it appears to ‘work’. For*

Over the last 15 years or so there have been many example, Bennet-Jenkins and Bryant (1996) tested publications listing EVM medicines and practices, as the anthelmintic effect of Eucalyptus grandis leaves indicated above. However, there have been very few with feral goats. On autopsy these authors found 91 trials and validations of EVM that stand up to rigorous Ethnoveterinary Medicine in the Tropics • 85 scientific scrutiny. There may be a bias in favour of (6)

The remedy has been scientifically validated as identifying treatment success with EVM, as has been effective to treat the problem in the livestock observed in human ethnomedicine (Roscoe, 1991). species in question.

Thus it is now timely to bring to EVM the rigour of

Of some 330 treatments listed by IIRR (1994) only five objective assessment.

are designated as '6', i.e. as being scientifically validated.

Hammond et al. (1997) have outlined one approach

Of these five, one is for a pig sedative using the leaves

for the identification and validation of anthelmintic

of Mimosa pudica, the other four are for upper respi-

plants used in EVM. In brief this methodology involves

ratory problems in poultry (leaves of Heliotropium

the following stages:

indicum, Spondias pinnata and garlic, Allium sativum)

- *A wide-ranging survey to identify all relevant and for internal parasites (garlic and the latex of EVMs and the people involved.*

papaya, Carica papaya).

- *Selection of the 'best bet' anthelmintic plants being*

Is validation of EVM important for the livestock

used against the most economically important

owners who use it? Possibly 'no' if they are using practices that have been used 'successfully' for generations.

- *Study of selected plants in terms of their use in*

But possibly 'yes' if they have seen the effectiveness of human medicine, given that many plants are used modern drugs as indicated above. Are users of EVM in both human and animal medicine.

experimenting with/validating EVM medicines and

- *Preparation of the test medicines in the ways used methods now in any way at all? Until relatively recently by the livestock owners/healers.*

the answer given by outsiders would have been 'no'.

- *Testing for activity against indicator helminths.*

But following on from the work of van Veldhuizen et al.

- *Testing for toxic effects, probably on-station.*

(1997) and others, it is now clear that much experi-

- *Validation on-farm with livestock owners.*

mentation is done by ordinary farmers in the tropics although its extent remains to be fully documented.

If the results of the above are positive it is logical to

One example of an on-farm ethnoveterinary validation proceed to more detailed identification of the active with farmers has been provided in Peru within the

*principles and of how their effects might be extended,
Project for the Validation of Technologies in Commu-
for example through improved storage.*

*nities (McCorkle & Bazalar, 1996). In this instance
It is relatively easy to list EVM practices and to
the validation exercise proved to be very positive and
review the literature in human and animal medicine.
beneficial for the community concerned.*

*However, as soon as any attempt is made to test EVM
If EVM is locally specific then validation probably
in a quantitative way major problems arise. The
has to be locally specific. In the past validation work has
ultimate one is cost, but this arises from the fact that
been something usually done on-station and in labora-
so many EVM practices are locally specific and highly
tories with the use of specialized equipment. The need
variable in their application and use.*

*for these has not gone away, but there are a number of
One response to the above problem is a qualitative
tools such as weigh-bands and body condition scoring
method of validation based on 'confidently used' EVM.*

systems which serve to make validation on-farm more
IIRR (1994) produced a collection of EVM practices in
feasible. Van Wyk et al. (1997) have reported an eyelid Asia using a workshop
brainstorming technique. The
colour system for measuring anaemia in sheep and
authors attempted to 'validate' practices on a scale of
relating this to worm burdens. This system could be
1–6 as follows:

used to help organize balanced groups of animals with
similar worm burdens for the purposes of testing the

(1)

Workshop participants agreed that the treatment
efficacy of, for example, anthelmintic plants. The
would be useful.

McMaster method for counting parasite eggs in animal

(2)

Treatment is widely used in a region or a country.

faeces is one that can be learnt relatively easily and

(Some remedies were also validated against prac-

used at farmer group level. Although facilitated farmer
tices from outside Asia.)

validation may seem ambitious it seems unlikely that

(3)

*Workshop participants had first-hand knowledge
extensive validation of EVM in the tropics will be
of the remedy's use on-farm.
achieved in any other way.*

(4)

Traditional healers are known to use the remedy.

(5)

*The remedy is cited in the literature in one of two
ways: either it is used to treat the same problem*

Discussion

*in humans or another animal species or this plant
has proven pharmacological activity to treat the*

*If it is assumed that validation trials can be organized
problem in question (laboratory validation).*

and carried out, there are two possible outcomes. One

86 • Chapter 7

is that the plant has no statistically significant effect on

Bizimana, N. (1994) Traditional Veterinary Practice in Africa.

the targeted organism. For example, on the basis of a

Deutsche Gesellschaft für Technische Zusammenarbeit

literature review, Hammond et al. (1997) suggested that (GTZ) GmbH, Eschborn.

Mallotus philippensis, widely used in EVM in Asia,

Hammond, J.A., Fielding, D. & Bishop, S.C. (1997) Prospects could be effective as a broad spectrum anthelmintic.

for plant anthelmintics in tropical veterinary medicine.

Veterinary Research Communications, 21, 213–28.

Following an actual validation of the dried powdered

IIRR (1994) Ethnoveterinary Medicine in Asia: An Informa-

fruit of this plant, termed kamala, Jost et al. (1996) con-tion Kit on Traditional Animal Health Care Practices. Inter-cluded that a particular sample of the dried fruit was

national Institute of Rural Reconstruction, Silang, Cavite,

ineffective against particular gastrointestinal nema-

Philippines.

todes in certain goats indigenous to Balochistan,

Jost, C.C., Sherman, D.M., Thomson, E.F. & Hesselton, R.M.

Pakistan. If a medicinal plant is found to be ineffective

(1996) Kamala (Mallotus philippinensis) fruit is ineffective in a validation it will be difficult to communicate the

as an anthelmintic against gastro-intestinal nematodes in

result to many livestock owners. There is unlikely to be

goats indigenous to Balochistan, Pakistan . Small Ruminant

any campaign, or funding, to stop livestock owners

Research, **20**, 147–53.

using it. Furthermore, livestock owners may not believe

McCorkle, C.M. & Bazalar, H. (1996) Field trials in ethno-

veterinary research and development: lessons from the

the negative result, and in any case there may be some

Andes. In *Ethnoveterinary Research and Development*

medicinal effect albeit in different circumstances and at

(ed. by C.M. McCorkle, E. Mathias & T.W. Schillhorn van

a biological but statistically insignificant level.

Veen), pp. 264–82. Intermediate Technology Publications,

If a validation proves positive, what are the options?

London.

One obvious option is to encourage greater use of the

Mathius-Mundy, E. & McCorkle, C.M. (1989) *Ethnoveterinary* medicine in
question through extension services and

Medicine: An Annotated Bibliography. Bibliographies in publications. Even so it
will be difficult to make uni-Technology and Social Change, No 6. Technology
and Social

versal recommendations as is possible with a synthetic

Change Program, Iowa State University, Ames, Iowa.

drug, because of the underlying variability in EVM as

Monteiro, A.M., Wanyangu, S.W., Kariuki, D.P., Bain, R.,

arising, for example, from species, seasonal and method

Jackson, F. & McKellar, Q.A. (1998) Pharmaceutical quality of preparation effects.

of anthelmintics sold in Kenya. *Veterinary Record*, **142**, 396–8.

Thus, whether or not validation is carried out and

Nichter, M. (1992) (ed.) *Anthropological Approaches to the* whether or not it is positive seems unlikely to have

Study of Ethnomedicine. Gordon and Breach Sciences, San much effect on the short-term use of ethnoveterinary

Antonio.

medicines by livestock owners outwith the specific

Norval, R.A.I. (1983) Arguments against intensive dipping.

locality in question, although this is not necessarily the

Zimbabwe Veterinary Journal, **14**, 19–25.

case for the long term.

Peacock, C.P. (1996) *Improving Goat Production in the Tropics*

– *A Manual for Development Workers*. Oxfam, Oxford.

Roscoe, P. (1991) *The Perils of ‘Positivism’: A Critique*

Conclusions

of the Image of ‘Positivism’ in Cultural Anthropology.

Unpublished manuscript, as cited by Nichter (1992).

The key issues highlighted above are as follows:

van Veldhuizen, L., Waters-Bayer, A., Ramirez, R., Johnson,

A. & Thompson, J. (eds) (1997) *Farmers' Research in*

- If poverty and/or drug costs continue to increase

Practice: Lessons from the Field. Intermediate Technology then so will the use of EVM.

Publications, London.

- Some EVM 'works' and is worth investigating/

van Wyk, J.A., Malan, F.S. & Bath, G.F. (1997) Rampant

validating objectively.

anthelmintic resistance in sheep in South Africa – what are

- The evolution of evidence-based EVM will be slow

the options? In *Managing Anthelmintic Resistance in*

Endoparasites (ed. by J. van Wyk & P.C. van Schalkwyk), and may require a new participative approach to

pp. 51–63. Workshop held at the 116th International Con-

validation.

ference of the World Association for the Advancement of

From the above points it can be further concluded that

Veterinary Parasitology, August 1997, Sun City, South

there is likely to be a continuing place for EVM in the

Africa.

tropics alongside modern methods of disease treatment and according to circumstances.

Further reading

References

Ibrahim, M.A., Nwude, N., Ogunsusi, R.A. & Aliu, Y.O. (1984) Screening of West African plants for anthelmintic activity .

Bennet-Jenkins, E. & Bryant, C. (1996) Novel anthelmintics.

International Livestock Centre for Africa Bulletin, **17**, 19–

International Journal of Parasitology, **26**, 937–47.

23.

Ethnoveterinary Medicine in the Tropics • 87

ITDG & IIRR (1996) *Ethnoveterinary Medicine in Kenya: A Relevant internet*: <http://www.rbgekew.org.uk/peopleplants/>

Field Manual of Traditional Animal Health Care Practices.

(17.11.99).

Intermediate Technology Development Group and Inter-

Wanyama, J. (1997) *Confidently used Ethnoveterinary Knowl-*

national Institute of Rural Reconstruction, Nairobi.

edge among Pastoralists of Samburu, Kenya. Intermediate Relevant internet site:

<http://pc4.sisc.ucl.ac.be/prelude/>

Technology, Nairobi.

[prelude_Homepage.html](#) (17.11.99).

Chapter 8

Heat Stress in Dairy Cattle

J.K. Shearer

Introduction

88

ondary concern and almost always suffers. The specific

Thermoregulation and the thermal comfort zone

88

thermoregulatory responses that impact performance

Body heat production

89

are a reduction in feed intake and nutrient absorption,

Heat gain from the environment

89

and a redirection of blood flow from internal organs to

Body heat loss

89

peripheral tissues. During the prepartum period, this

Effects of heat stress on performance

89

results in lower calf birthweight and reduced milk yield

Prepartum heat stress

89

in the subsequent lactation. Postpartum, hyperthermia

Peripartum heat stress

90

Postpartum heat stress

90

depresses dry matter intake, reduces milk yield and

Methods to reduce heat stress

90

decreases reproductive performance.

Natural shade

90

Methods to relieve heat stress include the provision

Artificial shade

90

of shade, forced air movement as with fans, and water

Cooling by reducing ambient air temperature

91

in the form of a fog, mist or sprinkle droplet. In some

Evaporative cooling pads and fans

92

areas of the world cooling of cows may be accomplished

High pressure foggers

92

by permitting access to ponds which are managed

Misters

92

for such purposes. Depending upon specific climatic

Enhancing the cow's natural mechanisms of heat loss

92

conditions (i.e. hot dry or arid verses hot and humid)

Sprinklers and fans

92

these can be used in combination to assist cows in the

Sprayers in parlour exit lanes

93

achievement of acceptable heat balance.

Cooling ponds

93

Introduction

Thermoregulation and the thermal

comfort zone

Cattle, like all mammals and birds, are homeotherms.

Despite wide fluctuations in environmental tem-

For lactating dairy cattle, the most comfortable

perature they are capable of maintaining a relatively

environmental temperature range is between 5 and

constant body temperature. This ability to regulate or

25°C (41 and 77°F), otherwise known as the thermal

stabilize body temperature is essential to preserve the

comfort zone. Within this range of temperatures

multitude of biochemical reactions and physiological

optimal animal performance can be expected. Temper-

processes that occur with normal metabolism. As envi-

atures that range outside this zone may require an alter-

ronmental temperatures rise a series of thermoregula-

tion in the basal metabolic rate for the animal to

tory responses designed to stabilize body temperature

maintain normal body temperature. The lower critical

are initiated. These include physiological, anatomical

temperature is the point at which an animal will begin

and behavioural changes which exhibit themselves as

to feel cold and must increase body heat production.

reduced feed intake, decreased activity, shade or wind

It varies with age, physiological status (lactating or

seeking, increased peripheral blood flow, sweating and

non-lactating), degree of insulation, level of milk pro-

panting.

duction and acclimatization. For example, the lower

During periods when ambient air temperature and

critical temperature for neonatal calves is reported to

humidity are particularly high, thermoregulatory

be 12.8°C (55°F). On the other hand, a mature cow in

activities may not be sufficient to maintain normal body

peak lactation may be comfortable at a temperature of

temperature. The result is a rise in body temperature

-25°C (-13°F). The upper critical temperature is the

and the induction of a series of thermoregulatory

point above which an animal begins to feel warm and

events whose objective is survival from a potentially

must begin to compensate. Unlike the variability

life-threatening crisis. Performance is naturally a sec-

described for lower critical temperature, the upper

Heat Stress in Dairy Cattle • 89

critical temperature limit remains constant at about mechanisms. Between the temperatures of -17.8 and 25°C (77°F) regardless of age or physiological status. 10°C (0–50°F), 75 per cent or more of heat loss from the body occurs by non-evaporative cooling (i.e. conduction, convection and radiation). However, at tem-

Body heat production

peratures of 29.5°C (85°F) and above, nearly 80 per cent of heat is dissipated by evaporative cooling (i.e. the Total body heat load is a combination of heat derived evaporation of water from the skin and respiratory from metabolism and that obtained from environmental sources. In general, whenever temperatures exceed 21°C (70°F), evaporative cooling becomes the predominant basal body functions (digestion and cellular biochemical mechanism of heat loss in cattle.

cal reactions) accounts for 35–70 per cent of total daily

Bos indicus (Zebu) cattle have larger and a greater

heat production. In ruminants there is greater heat gain number of sweat glands than *Bos taurus* cattle, but associated with the digestion of roughages as compared actual sweating rates are only slightly higher. The evaporation of water from the cow's skin is a very effective include that associated with daily physical activity and cooling mechanism. It is enhanced by conditions which increased metabolic activity associated with lactational provide air movement which moves water vapour away performance. Finally, it is significant to note that the from the skin thereby increasing the vapour-pressure increased respiratory rates and panting associated gradient in the immediate air space surrounding the with evaporative cooling, while necessary to cool the animal.

cow, also account for an increase in daily maintenance The primary obstacle to evaporative cooling is requirements by 7–25 per cent.

high relative humidity. Humid air is more saturated with water vapour. Thus, the vapour-pressure gradient

is reduced. In some environments this is exacerbated

Heat gain from the environment

still further by limited air movement. This results in

very low water evaporation rates and consequently

The primary sources of heat gain from the environment

minimal cooling. In hot and humid environments this

are solar radiation and high ambient air temperature.

condition may be overcome by a combination of sprin-

The amount of heat absorbed by an animal exposed to

kling the skin with water and forced air movement with

direct sunlight is related to coat colour. Black cows

fans. Fanning cools wetted cows by accelerating the

absorb over twice as much heat from the sun as white

water vaporization rate.

cows. This is complicated by virtue of the fact that

the flow of heat away from the cow's body is restricted

by high ambient air temperature which narrows the

Effects of heat stress on

thermal gradient between the cow's body and the

performance

surrounding air. Thus, solar radiation together with

high ambient air temperature are important sources of

Prepartum heat stress

heat gain from the environment. Since little can be

done to reduce air temperature, efforts to provide

Elevated environmental temperatures during the last

shade should be a high priority.

trimester of gestation alter blood flow to the uterus and

maternal–fetal hormone concentrations. The result

is lower calf birthweight and reduced milk yield in

Body heat loss

the subsequent lactation. Approximately 60 per cent of

fetal growth occurs during the last 90 days of gestation.

The release of heat from any object to the environment

Chronic heat stress during late gestation leads to

is proportional to its exposed surface area. Further,

reduced blood flow to the uterus which retards placen-

the ratio of surface area to body mass decreases as

tal and fetal growth. As a consequence, placental mass

overall size increases. This means that large animals

is reduced and calf birthweights are lowered by as much

such as cows are at a disadvantage in losing excess

as 6 to 8 per cent. Studies have also shown that milk body heat. They are also at greater risk of becoming yield is related to calf birthweight and lower calf birth-overheated. Calves, on the other hand, have a greater weights are associated with reduced milk production. amount of surface area relative to body mass and, Although the precise reasons for reduced milk yield are therefore, are much better at dissipating heat than a unknown, most researchers believe that hyperthermia mature cow.

in late gestation interferes with normal hormonal Avenues for the dissipation of heat in cows include regulation of mammary development, lactogenesis and both non-evaporative and evaporative cooling milk yield.

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Peripartum heat stress

Heat stress has a major impact on reproductive performance. It lowers conception rates, reduces the Cows calving during daylight hours in hot climates length of the oestrous period and oestrous intensity,

with direct exposure to solar radiation are particularly modifies endocrine function, alters the oviductal and subject to hyperthermia and heat stroke (p. 935). This uterine environment and increases early embryonic is particularly true for cows experiencing hypocalcaemia at calving, when control of body temperature seasonal depression in reproductive performance that by natural mechanisms is greatly diminished. Therefore results is one of the most serious problems for the dairy close monitoring of calving cows is essential in hot and livestock industry of subtropical and tropical climates.

regions throughout the entire world.

Heat stress at parturition also creates significant consequences for calves. Once delivered, calves born in stressful conditions are weaker and slower to suck colostrum. Indeed, passive transfer of immunoglobulins

Methods to reduce heat stress (p. 975)

has a seasonal pattern in calves. Calves born during the hotter summer months have higher rates of failure

Successful abatement of heat stress generally of passive transfer whereas calves born during the requires environmental modification. Critical components more moderate times of the year tend to have better include shade, water in the form of a fog, mist or rates of passive transfer. At least one study suggests sprinkling and air flow – either natural or by forced air that an increase in serum corticosteroids in heat movement with fans. The primary objectives are to stressed neonates reduces permeability of the intestine reduce direct solar radiation, lower air temperature, to immunoglobulin absorption. Thus, it would appear improve or assist air movement or in some cases increase that both physical and physiological mechanisms are the natural evaporative cooling from skin surfaces. responsible for high rates of failure of passive transfer in calves.

Natural shade

Trees are an excellent source of shade. They are

Postpartum heat stress

not only effective blockers of solar radiation but the

Reduced feed intake is a primary strategy for lowering evaporation of moisture from leaf surfaces cools the body heat production. The consequence is a reduction surrounding air without appreciably interfering with air in milk production of as much as 10 to 20 per cent circulation. In addition, animals acquire very little or more. Similar effects on constituent (milk fat and radiant heat load from the shade of a tree compared protein) yield are observed. Milk quality parameters with a metal roof. Therefore, trees are a highly desirable natural resource in the environment of the dairy counts tend to increase during periods of hot and humid weather. Although specific data are limited, where they are not protected. In fact, most last only the observed increase in somatic cell counts suggests about one to two summers after the onset of cow exposure a greater susceptibility to infection due to decreased host resistance or increased exposure to pathogens

to seek protection from the summer sun they quickly created by an environment more favourable for their develop mud holes at the base of trees. This soon leads propagation.

to death of the tree and loss of this natural shade source.

Improper ration formulation, intermittent feeding

In order to take advantage of natural shade, some effort behaviour, a lack of cud chewing, elevated respiratory must be made to ensure that trees are protected from rates, excessive losses of saliva from drooling and an damage by cows.

overall reduction in the buffering capacity may interfere with the normal buffering of rumen contents. This

Artificial shade

is believed to be a significant contributor to rumen acidosis (see p. 829), laminitis (see p. 420) and other

Solar radiation is a major factor in heat stress and lameness conditions (see Chapter 31) that seem to be increases heat gain by direct as well as indirect means. particularly prevalent during periods of hot weather.

Blocking its effects through the use of properly

Feeding the lactating cow is particularly challenging constructed shade structures alone increased milk during periods of intense heat. The objective is to production by 10–19 per cent in studies conducted in maintain some level of performance and homeostasis Florida. Options include permanent or portable shade yet not add to the internal heat load or tendency toward structures. In the following sections consideration rumen acidosis.

will be given to design and maintenance factors.

Heat Stress in Dairy Cattle • 91

Permanent shade structures

Structures wider than 12 m (40 feet) should have eave heights of at least 5 m (16 feet) or more.

Major design parameters for permanent shade struc-

- There should be at least 15m (50 feet) of clearance

tures include:

between adjacent buildings or other obstructions.

- Orientation
- Gable roofs should have at least a 4:12 slope (6:12
- Floor space

is acceptable but difficult to work on) and a

- Height

continuous open ridge. Ridge caps if desired should

- Ventilation

have a minimum of 0.3 m (1 foot) of clearance

- Roof construction

between the cap and the roof peak.

- Feeding and water facilities

- Ridge openings should be a minimum of 0.3m (1

- Waste management system.

foot) wide plus 5 cm (2 inches) for each 3 m (10 feet)

of structure width over 6 m (20 feet).

The preferred orientation of a shade structure

- Painting metal roofs white and adding insulation

depends upon whether or not cows are confined to the

directly beneath the roofing will reflect and insu-

structure. Alignment of the long axis in an east–west

late from effects of solar radiation and will reduce

direction achieves the maximum amount of shade

thermal radiation on cows.

under the structure and is therefore the preferred

orientation for confined animals. On the other hand, Thermal radiation from the roof of shade structures where cows are free to move with the shadow of the can add significant heat load to cattle, particularly structure a north–south orientation is better because in low structures without a ridge opening. In these this orientation will allow sunlight to dry out as much types of structures thermal radiation can be reduced by as 35–50 per cent of the area beneath the shade cooling the roof with water, adding insulation or paint-structure during both the morning and afternoon hours. ing the roof with a reflective type of paint. However, it This is particularly important for shade structures with should be remembered that these additions to the earthen floors.

structure do not cool air, reduce humidity or augment Some prefer concrete slab floors. A reinforced concrete slab at least 10 cm (4 inches) thick, with a smooth beneath the shade structure. Furthermore, proper finish and grooved for good footing, is recommended.

design of the shade structure (adequate eave height and If a flush system is to be used the floor should be sloped an open ridge) will naturally limit thermal radiation 1.5–2 per cent. Water availability, space and environmental effects. When faced with the need to retrofit cooling mental concerns are currently of interest in floor scraping into an ill-designed existing structure, the priorities ing and removal of manure solids from the premises. should be directed to cooling the cows rather than the Various other waste handling facilities incorporate roof. Roof cooling (beyond painting with a reflective settling basins, liquid/solid separators, pumping and paint), while beneficial, is a secondary consideration. gravity-flow systems.

Guidelines regarding the size of shade structures

Portable shade structures

vary according to climatic conditions. Some recommend 1.75–2.5 m² (19–27 square feet) of floor space per Portable shades offer some advantages over permanent cow. However, for hot and humid environments, some structures in their ability to be moved as required to

would recommend a floor space equivalent to 5.5–
cleaner and drier locations. However, protection from
6.0 m² (60–65 square feet) per cow. Space requirements
solar radiation is less than that achieved in permanent
are doubled for hot and humid climates to provide an
structures. Shade cloth patterns come in various weaves
additional open area for improved air movement.
providing 30–90 per cent shade. One of the more com-
Natural air movement under a shade structure is
mon types is a woven polypropylene fabric which
affected by its height and width, the slope of the roof
provides 80 per cent shade. While longevity is consid-
and the presence of, or size of, the ridge opening.
erably less than that expected of permanent structures,
Air movement may occur naturally as breezes through
shade cloth if properly maintained (kept tight) can last
the open sides of structures or by thermal buoyancy,
5 years or longer.
in which air warmed by the presence of animals
and thermal radiation through the roof creates air
flow toward the ridge opening. A steady flow of air

Cooling by reducing ambient air

through a shade structure requires the following design

temperature

specifications:

- Shade structures of 12m (40 feet) or less in width

As temperatures rise above the upper critical temperature threshold of 25.5°C (78°F) the dairy cow begins to require a minimum eave height of 3.7 m (12 feet).

temperature threshold of 25.5°C (78°F) the dairy cow begins

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to increase heat loss via the respiratory tract and skin

litres (30–50 gallons)/cow per day). The primary

surface. However, despite the remarkable efficiency of

disadvantage is that they require more maintenance.

these thermoregulatory responses to dissipate heat, as

In-line water filters must be cleaned or checked daily

temperatures continue to rise these natural mechanisms

to prevent clogging of fogger nozzles.

are overwhelmed, leading to hyperthermia and reduced

performance. In these circumstances efforts to mini-

mize additional heat gain and supplement cooling be-

Misters

come necessary. Water and air movement become the

A mist droplet is larger than a fog droplet but cools air

agents by which the micro-environment is cooled and

by the same principle. These systems do not work

evaporative cooling by the cow is augmented.

well in windy conditions or in combination with fans in

humid environments. In warm humid environments

Evaporative cooling pads and fans

mist droplets are too large to evaporate fully before

settling to the ground. The consequence is wet bed-

Air temperatures can be lowered by air conditioning or

ding and wet feed. A further complication with misters

refrigeration, but the expense of such types of mechan-

is the formation of an insulating layer of air between

ical air cooling makes these impractical for cooling

the droplets of water on hair shafts and the cow's

dairy cows. A more economically feasible method to

skin. When this occurs it impedes natural evaporative

cool the micro-environment is the evaporative cooling

heat loss from the skin and can result in body heat

pad (corrugated cardboard or similar material) and fan

build-up.

system which uses the energy from air to evaporate water. This process cools the air and raises its relative humidity. Although these systems are most effective in

Enhancing the cow's natural

arid climates, they have been observed to reduce air temperature in humid climates as well.

mechanisms of heat loss

Protecting the cow from solar radiation with shades and

High pressure foggers (see also Chapter 71)

reducing ambient air temperatures through the pro-

In recent years interest in high pressure foggers has
cess of water vaporization and controlled ventilation
grown. These systems offer effective cooling but with
are important considerations in cooling dairy cattle.

lower water use. Foggers disperse very fine droplets of
Various combinations of these techniques have proven
water which quickly evaporate, cooling the surrounding
to be particularly useful in arid climates. Cooling in hot
air and raising the relative humidity in the process. The
and humid climates, on the other hand, can be more

typical design incorporates a ring of fogger nozzles challenging. Instead of trying to lower ambient air attached to the exhaust side of a fan. As fog droplets temperature, another technique is to provide shade, wet are emitted (200 psi) they are immediately dispersed the skin and move air to enhance the cow's primary into the fan's air stream where they soon evaporate. The mechanism for the dissipation of heat – evaporative temperature of the animals is reduced as they inspire cooling from the skin.

the cooled air and it is blown over their bodies. Fogger systems are most effective in areas of low humidity.

Sprinklers and fans

However, even where humidity is normally quite high, daytime humidity is still low enough to allow effective Sprinkling systems utilize a larger size water droplet cooling with fogger systems. In areas where relative that is able to wet the hair coat to the skin. Cooling humidity increases to nearly 90 or 100 per cent in the is accomplished as water evaporates from the hair overnight hours foggers must be turned off. Once the

and skin. In combination with forced air, sprinkling air is saturated with moisture evaporation is reduced substantially increases the loss of body heat over and cooling stops. High pressure foggers should that possible by sweating alone. Several studies have be designed to operate during the less humid hours of demonstrated upper body sprinkling followed the day. Finally, they should be used only in open- by forced-air ventilation to be an effective means sided, ridge-vented, tall (greater than 3.7 m (12 feet)) to reduce body temperature, increase feed intake and barns. Low barns with side walls restrict air flow and boost milk yield. This combination has been applied to fog droplet evaporation. This reduces cooling and holding areas outside milking areas, shade structures, makes for excessively wet conditions in the barn. feed barns and free-stall barns with a high degree of High pressure foggers are advantageous in the fact success.

that they use far less water (13.5–23 litres (3–5 gallons)/

Sprinkler and fan systems require a properly sloped

cow per day) compared with sprinkler systems (135–230

concrete floor with facilities to handle water run-off at

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rates of somewhere between 230 and 450 litres (50 and

good reason. Experience has shown that free access

100 gallons) of water per animal per day depending

to streams and ponds may predispose to a number of

upon sprinkling rates. In early Florida studies water use

infectious diseases and some toxicities in dairy cattle.

amounted to 455 litres (120 gallons)/cow per day (sprin-

Most notable of these are leptospirosis (see pp. 734–7)

kling for 30 seconds every 5 minutes when ambient air

and mastitis (see Chapter 23) caused by a variety of

temperatures exceeded 26.5°C (80°F)). Later work has

organisms, particularly *Protheca* species (achlorophyllic shown that rates of 230
litres (50 gallons)/cow per day

algae). As a result, most advise that cows be fenced

or less will provide effective cooling. Consequently, in

away from streams and ponds. Cooling ponds, there-

addition to plans for water run-off and containment,

fore, represent a controversial method for the manage-

some determination of the water supply is advised.

ment of heat stress. However, studies in the USA have

Sprinklers should be located above the cows, with

found that cooling ponds not only effectively reduce

nozzles directed such that they wet the cows but not

body temperature, but have no apparent adverse effect

the feed. The type of nozzle chosen depends upon the

on udder health.

volume of water and sprinkling rate desired. Generally,

The primary mode of heat loss in cooling ponds is

low pressure (10 psi), 180° spray nozzles, capable of

conduction, with a small amount lost by evaporative

delivering the equivalent of 1.25 mm (0.05 inches) of

cooling during the 5–10 minutes after exiting the pond.

rainfall per sprinkling cycle, are used. This sprinkling

Water temperature of the cooling ponds studied in the

rate assures that the cows will be wetted to the skin.

USA generally ranged from 24 to 30°C (75 to 86°F),

Nozzles are spaced approximately every 2.5 m (8 feet)

or occasionally higher. At this temperature there was

or as far apart as necessary to provide overlapping

a favourable heat transfer gradient between the cow's coverage.

body and the pond water.

Fans (0.5–1.0 hp) capable of air flow rates of 11 000

Major questions remain as to how cooling ponds

cfm or greater are recommended. Ninety centimetre

should be designed or maintained. Some operations

(36 inch) fans rated as such can be hung above the

that rely on ponds for cooling cows maintain them by

sprinklers every 9 m (30 feet) (every 1.2 m (40 feet) for

providing a constant inflow of water, with an overflow

120 cm (48 inch) fans). They should be tilted downward

at one end of the pond. They also drain, dredge and fill

at a 20–30° angle (from vertical) to direct the flow

them with new sand every 1 to 2 years. Although total

of air on to the cows. An air velocity of 120–185 m

bacterial content does not appear to be appreciably

(400–600 feet) per minute over the cow is desired. The

affected there is less build-up of organic material. There

system combines fans and sprinkling, with cows being

is some evidence that allowing cows access to stagnant

sprinkled for 1–2 minutes at 15-minute intervals. Fans or natural ponds may negatively affect milk quality and should be run continuously. The entire system should the incidence of mastitis. Cows from herds with man-made ponds, which are maintained regularly, produce when ambient air temperatures reach or exceed 26.5°C milk with lower bacteria and somatic cell counts (80°F).

compared with cows from herds which have no ponds or natural ponds. Thus, one would conclude that the use of ponds for heat stress management should be accom-

Sprayers in parlour exit lanes

panied by plans for pond maintenance.

Exit lane sprayers are available commercially and designed to automatically spray water on to cows as they pass through. Fan nozzles and timing of the spray

Further reading

are designed to spray only on to the cow's back and sides. Fan spray nozzles must have a flow rate of at least Beede, D.K. & Shearer, J.K. (1991) Heat stress, part IV:

36 litres (8 gallons) per minute at 40 psi. These systems
nutritional management of dairy cattle during hot weather.

would seem to have greatest appeal in operations

Agri-Practice, **12**, 5–13.

where cows travel some distance from the milking

Beede, D.K., Bray, D.R., Bucklin, R.A., Elvinger, F. & Shearer, parlour to feed
and in loafing areas. A less complex

J.K. (1987) Integration of cooling methods for environmen-

system can be made by simply locating an ordinary

tal management systems in hot humid environments.

Proceedings of the 24th Annual Florida Dairy Production

shower nozzle above cows in the parlour exit lane.

Conference, pp. 68–93.

Cows can be showered as they leave the parlour.

Bray, D.R. (1986) Housing systems for dairy cattle in Florida.

Southeastern Dairy Review, **25**, 12–13.

Cooling ponds

Bray, D.R., Beede, D.K. & DeLorenzo, M.A. *et al.* (1991) Environmental
modification update. *Proceedings of the 28th Tradition* has held that it is better
to limit or exclude

Annual Florida Dairy Production Conference, pp. 134–

access of cows to streams and farm ponds, and with

40.

94 • Chapter 8

Bray, D.R., DeLorenzo, M.A. & Elvinger, F.C. *et al.* (1989) Holstein cows in a subtropical climate. *Journal of Dairy*

Cooling ponds and milk quality. *Proceedings of the 26th*

Science, **57**, 476–81.

Annual Florida Dairy Production Conference, pp. 63–72.

Roman-Ponce, H., Thatcher, W.W., Buffington, D.E., Wilcox,

Bray, D.R. & Shearer, J.K. (1988) Environmental modifica-

C.J.

& Van Horn, H.H.

(1977) Physiological and

tions on Florida dairies. *Proceedings of the 25th Annual*

production responses of dairy cattle to a shade structure

Florida Dairy Production Conference, pp. 52–9.

in a subtropical environment. *Journal of Dairy Science*, Bucklin, R.A., Bray, D.R. & Beede, D.K. (1988) Methods to

60, 424–30.

relieve heat stress for Florida dairies. *Florida Cooperative* Schneider, P.L., Beede, D.K., Wilcox, C.J. & Collier, R.J. (1984) *Extension Service Circular* 782.

Influence of dietary sodium and potassium bicarbonate

Bucklin, R.A., Turner, L.W., Beede, D.K., Bray, D.R. &

and total potassium on heat stressed lactating dairy cows.

Heurleen, R.W. (1991) Methods to relieve heat stress for

Journal of Dairy Science, **67**, 2546–53.

dairy cows in hot, humid climates. *Applied Engineering in*

Shearer, J.K. & Beede, D.K. (1990a) Heat stress, part I:

Agriculture, **7**, 241–7.

thermoregulation and physiological responses of dairy

Buffington, D.E., Collier, R.J. & Canton, R.H. (1983) Shade cattle in hot weather. *Agri-Practice*, **11**, 5–18.

management systems to reduce heat stress for dairy cows.

Shearer, J.K. & Beede, D.K. (1990b) Heat stress, part II: effects *Transactions of the ASAE*, **26**, 1798–802.

of high environmental temperature on production, repro-

Collier, R.J. & Buffington, D.E. (1979) Common Florida shade duction, and health of dairy cattle. *Agri-Practice*, **11**, 6–18.

management systems to reduce heat stress. *Proceedings of*

Shearer, J.K., Beede, D.K., Bucklin, R.A. & Bray, D.R. (1991) *the 16th Annual Florida Dairy Production Conference*, pp.

Heat stress, part III: environmental modifications to reduce

36–45.

heat stress in dairy cattle. *Agri-Practice*, **12**, 7–18.

Collier, R.J., Doelger, S.G., Head, H.H., Thatcher, W.W. & Shearer, J.K., Bray, D.R., Elvinger, F.C. & Reed, P.A. (1987) Wilcox, C.J. (1982) Effects of heat stress during pregnancy

The incidence of clinical mastitis in cows exposed to cooling on maternal hormone concentration, calf birth weight, and ponds for heat stress management. *Proceedings of the 26th postpartum milk yield of Holstein cows. Journal of Animal Annual Meeting, National Mastitis Council*, pp. 66–70. *Science*, **54**, 309–19.

Strickland, J.T., Bucklin, R.A., Nordstedt, R.A., Beede, D.K.

Collier, R.J., Eley, R.M., Sharma, A.K., Pereira, R.M. &

& Bray, D.R. (1989) Sprinkler and fan cooling system for

Buffington, D.E. (1981) Shade management in a subtropical

dairy cows in hot, humid climates. *Applied Engineering in*

environment for milk yield and composition in Holstein and

Agriculture, **5**, 231–6.

Jersey cows. *Journal of Dairy Science*, **64**, 844–9.

Taylor, S.E. (1985) *Evaluating the potential benefits of using an* Collier, R.J.,

Simerl, N.A. & Wilcox, C.J. (1980) Effect

evaporative cooling system for Florida dairy cattle. Masters of month of calving on birth weight, milk yield, and

thesis, University of Florida.

birth weight–milk yield interrelationships. *Journal of Dairy* Thatcher, W.W. &

Collier, R.J. (1985) Effects of climate on *Science*, **63**(Suppl 1), 90.

bovine reproduction. *Current Therapy in Theriogenology*, Erdman, R.A. (1988)

Dietary buffering requirements of the

pp. 301–9.

lactating dairy cow: a review. *Journal of Dairy Science*, **71**, Thatcher, W.W., Gwazdavskas, F.C. & Wilcox, C.J. *et al.* (1974) 3246–66.

Milking performance and reproductive efficiency of dairy

Flamenbaum, I.D., Wolfenson, D., Mamen, M. & Berman, A.

cows in an environmentally controlled structure. *Journal of* (1986) Cooling dairy cattle by a combination of sprinkling

Dairy Science, **57**, 304–7.

and forced ventilation and its implementation in the shelter

Turner, L.W., Chastain, J.P., Hemken, R.W., Gates, R.S. &

system. *Journal of Dairy Science*, **69**, 3140–7.

Crist, W.L. (1989) Reducing heat stress in dairy cows

Hahn, G.L. (1985) Management and housing of farm animals

through sprinkler and fan cooling. *Transactions of the*

in hot environments. In *Stress Physiology in Livestock*,

American Society of Agricultural Engineers, Paper No 89-

Volume II, Ungulates (ed. by M.K. Yousef), pp. 151–74. CRC

4025. St Joseph, MI.

Press, Boca Raton.

Wiersma, F. & Armstrong, D.V. (1988) Evaporative cooling

Igono, M.O., Johnson, H.D., Steevens, B.J., Krause, G.F. & dry cows for improved performance. *Transactions of the*

Shanklin, M.D. (1987) Physiological productive and

American Society of Agricultural Engineers, Paper No 88-economic benefits of shade, spray and fan systems versus

4053. St Joseph, MI.

shade for Holstein cows during summer heat. *Journal of*

Wolfenson, D., Flamenbaum, I. & Berman, A. (1988) Dry

Dairy Science, **70**, 1069–79.

period heat stress relief effects on prepartum progesterone,

Ingraham, R.G., Gillette, D.D. & Wagner, W.D. (1974) Rela-

calf birth weight, and milk production. *Journal of Dairy*

tionship of temperature and humidity to conception rate of

Science, **71**, 809–18.

Chapter 9

Nutrition

J.M. Wilkinson

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The bovine animal, along with the other ruminants,

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focused on the rumen as the most important part of the

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bovine digestive system. The fermentation in the rumen

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is dominated by the degradation of plant cell walls, of

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which the most abundant constituent is cellulose. This

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103

adaptation to plant cell wall digestion, in addition to cell

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content degradation places the bovine in an important

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Van Soest (1994) classified cattle feeding habit as

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being one of grazing fresh grass, rather than browsing

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trees and shrubs. Cattle have a greater need for water

Metabolizable protein

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than other ruminants, possibly because they retain fibre

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water content, typically around 90 per cent, is essential

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In temperate regions of the world, a constant supply

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of feed throughout the year is achieved by preserving

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excess herbage growth as hay or as silage. This activity,

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coupled with the production of root crops specifically

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totally dependent on grass growth to one which is not.

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Thus beef cattle, for example, no longer lose weight in

Out-of-parlour feeders

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the winter when grass is scarce, but are able to main-

Flat-rate feeding

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tain or even increase in weight on a winter diet of hay,

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silage or stored root crops. The development of the crops by ensilage. Essentially, dietary carbohydrates, human food and drink industry has released a wide proteins and some fats are reduced to short-chain fatty range of by-products, such as bran from the milling of acids with the production of carbon dioxide, methane grain for flour, and spent grains from the brewing of and ATP.

beer and the production of spirits. These by-products,

The short-chain fatty acids produced in the digestive

which are not edible by the human population, play a tract are principally acetic, propionic and butyric acids, vital role in the nutrition of the bovine when grazed although occasionally lactic acid is also produced. pasture grass is in short supply.

The most important bacteria involved in fermentation in the rumen are the cell-wall digesting or cellulolytic micro-organisms. A remaining challenge in bovine nutrition is to achieve a similar constancy of feed supply to that of the temperate regions of the world, to bovine populations in rumen fluid is about 10^{10} to 10^{11} bacteria/ml, with in regions where drought is common, and where the most being attached to particles of food. The predominant species of bacteria varies with the type of fermentation is less well developed.

tation, which depends on the principal substrates in the diet. Thus the major species of bacteria in the rumen In this chapter, the fermentation in the rumen is described, with particular emphasis on the kinetics of

of animals given a diet of grass are those which digest the process and its interaction with feed intake.

cellulose and hemicellulose, such as *Ruminococcus*

Requirements for energy and protein are then consid-

albus, *Ruminococcus flavefaciens* and *Bacteriodes* ered. The composition of feeds is outlined, together

succinogenes (see Table 9.1).

with technologies for feed preservation and feed pro-

Other species, such as *Streptococcus bovis*, ferment

cessing. Finally, the management of the feeding of

starch to acetic acid and ethanol. This species, along

the bovine is outlined in relation to maintaining stabil-

with other streptococci, can produce lactic acid and are

ity in the rumen, maximizing intake and meeting

more tolerant of acid conditions than other species

requirements for high levels of productivity.

of rumen bacteria. Acidosis can occur if the diet of

the animal is changed abruptly from cellulose to starch

or sucrose. The population of bacteria in the rumen

Fermentation in the rumen

changes from cellulolytic to amylolytic as the pH falls.

Lactic acid accumulates and accelerates the fall in pH.

The rumen accounts for more than half the total volume

Even a small fall in the pH of the rumen from pH 7 to

of the digestive tract of cattle. The animal relies very

pH 6 is reflected in a reduction in cellulose digestion

heavily on the reactions that occur in the rumen for its

and a change in the population of the bacteria towards

supply of major nutrients. Thus on a sole diet of grazed

the more acid tolerant species.

grass some 90 per cent of the animal's total energy

Proteins are degraded to a varying extent during the

and protein supply is derived directly from the

fermentation to their constituent amino acids. Some

rumen micro-organisms and the end-products of their

amino acids are used directly by bacteria and protozoa,

metabolism. Therefore, the importance of main-

but most are used as a source of energy and are broken

taining optimal conditions for fermentation cannot be

down further to ammonia and volatile fatty acids.

overstressed.

Ammonia is used as a substrate for the production of

The symbiotic relationship between the micro-microbial protein, with the excess being absorbed into organisms of the rumen and the host animal has been the animal's portal blood and converted to urea in the crucial to the survival of the bovine, since the animal itself liver. The extent to which proteins are degraded during does not produce the enzymes to degrade the cellulose the fermentation in the rumen depends on their and hemicellulose in plant cell wall material. This task is solubility, which is generally relatively high. However, undertaken by the microbial population of the rumen. solubility is lower in feeds which have been subjected In the wild, surrounded by foliage and limited sup-to heat treatment during processing. Thus the degrada-plies of feeds containing starch or sugar, the bovine's tion of protein in brewer's grains is only about 0.6, com-fermentation of plant cell wall material secured not pared to 0.9 for fresh herbage. Protein degradation also only the supply of energy, but also a vital supply of depends on the time the material spends in the rumen, microbial protein. The ruminant has evolved a nutri-

and is lower for diets which pass rapidly through the rumen (concentrates and feeds of small particle size) sources of amino acids and B vitamins – a considerable advantage when grass is the only feed available.

Protozoa and fungi are also involved in the fermentation process. These organisms can digest cellulose, starch, sugars and fats to produce acetic acid, butyric acid, lactic acid, hydrogen and carbon dioxide.

Fermentation in the rumen is the anaerobic process of microbial activity, which also occurs in the lower digestive tract of animals and in the preservation of

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Table 9.1

Common types of rumen micro-organisms and their action.

Species

Energy source

Fermentation

Requirements

products

Bacteria

Bacteroides

amylophilus

Starch

F, A, S

CO₂, NH₃, BrVFA

succinogenes

Cellulose

F, A, S

CO₂, NH₃, BrVFA,

SVFA, Vit.

Ruminococcus

albus

Cellulose, xylan

F, A, E, H₂, CO₂

BrVFA, CO₂, NH₃,

Vit.(A)

flavefaciens

Cellulose, xylan

F, A, S, H₂

Butyrivibrio

fibrosolvans

Xylan, starch

F, A, B, L, H₂, CO₂

BrVFA, A, CO₂,

NH₃, Vit.(A)

Lachnospira

multiparus

Pectin

F, A, L, E, H₂, CO₂, A, Vit.

Selenomonas

ruminantium

Lactate, starch

A, P, L, CO₂, H₂

A(CO₂)

Methanobacterium

ruminantium

Formate, H₂

Methane

A, BrVFA, Haem,

CO₂, NH₃

Protozoa

Holotrichs

Isotricha

Starch and sugars

A, B, L, H₂

Dasytricha

Starch and sugars

A, B, L, H₂

Entodiniomorphs

Entodinia

Starch

F, A, P, B, (L)

Epidinium

Starch, hemicell.

A, B, H₂, (F, P, L)

Ophryoscolex

Starch

A, B, H₂, (P)

Diplodinium

Eudiplodinium

H₂, fatty acids

Polyplastron

Symbols: F, formate; A, acetate; P, propionate; B, butyrate; BrVFA, branched-chain VFA; E, ethanol; L, lactate; S, succinate; SVFA, straight-chain VFA; Vit., B vitamins.

The predominance of the weak acids acetic, propionic and butyric in the end-products of fermentations in the rumen remains relatively constant. However, digestive disorders can arise to disrupt the equilibrium. Toxins, produced by undesirable bacteria and from moulds present in feeds, can damage the sensitive lining of the wall of the rumen and reduce the absorption of nutrients. Sudden changes in diet can change the microbial population and during eating and rumination is therefore crucial to the result in the production of lactic acid in the rumen, as in neutralization of the fermentation acids (see below).

the fermentation of crop material in the silo, with a con-

Long fibre is often included as a supplement to diets high

sequent reduction in rumen pH. If the pH of the rumen in concentrates to stimulate chewing and rumination. falls below pH 5 and remains at a low level, there is a risk The constant flow of saliva (p. 98) into and outflow of of rumen stasis which can result in bloat because the digesta from the rumen, and the absorption of digested animal can no longer eructate the gases produced by the nutrients into the portal blood, ensure that in most nutri-fermentation.

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Methane loss from the rumen is estimated to account cellulose and cellulose to glucose and fructose. Starch for about 8 per cent of the total gross energy eaten by and pectins are also similarly degraded (Fig. 9.1). In the the animal. Apart from the rumen fermentation, case of plant cell wall material, this erosion continues methane is also lost to the environment from hind gut until lignified tissue is encountered. Lignin, which is fermentations and together these fermentations con-cross-linked to hemicellulose and cellulose, provides stitute a significant contribution of methane to the

structural strength to the plant and is also very resistant to bacterial enzymic attack.

include chemical food additives (ionophores), and The protozoa in the rumen mainly ferment starch and changing the pattern of fermentation to increase the sugar, but they also consume bacteria. The protozoa proportion of propionate and to reduce the proportion are thought to be active in assisting the bacterial of acetate.

population in adapting to new feeds.

The end-products of bacterial digestion are short-chain acids acetic, propionic and butyric (the volatile

Saliva

fatty acids, VFA), microbial cells (and their constituent protein), and the gases methane and carbon dioxide.

Feeds are chewed during eating and also regurgitated

Methane comprises the major gaseous energy loss as during rumination to allow very thorough mastication.

a result of fermentation. Gas is lost by eructation

Throughout these activities large quantities of saliva are

whilst the VFA are mainly absorbed through the rumen mixed with food. The effect of saliva is to buffer the wall.

acids that are produced in the rumen as a result of the Some common types of rumen micro-organisms and fermentation. This buffering is vital to the maintenance their actions are summarized in Table 9.1.

of the correct type of fermentation, and can even prevent the collapse of normal rumen function in extreme situations.

The principal buffering constituent of saliva is bicar-

Fermentation of different types of feed

bonate. It has been estimated that a dairy cow may

The fermentation of plant cell walls is optimal at produce up to 3.5 kg of bicarbonate per day. Clearly, the relatively high rumen pH (around pH 7.0) because the need for adequate buffering is more important if the bacteria responsible are sensitive to excess acidity.

diet is rapidly fermented than if it is only slowly fer-

Their growth is depressed if rumen pH falls below mented. Further, if the diet is acidic, as in the case of

about pH 6.2. The principal end-product of cellulose silage, it is essential that salivary secretion is sufficient fermentation is acetate, an important precursor of to prevent a build-up of excessive acidity in the rumen milk fat.

or in blood.

Starch and sugar are fermented to give propionic and It follows that feeds which stimulate rumination are butyric acids as the main end-products. The micro-more useful to the maintenance of optimal conditions organisms responsible for their fermentation are more for fermentation in the rumen than those which do not. tolerant of acidity than those which ferment cell wall.

Unfortunately many constituents of high-energy con-

Some species of starch-digesting bacteria (e.g. *Strepto-* concentrates, such as molasses and ground cereal grains, are

coccus bovis, *Selenomonas ruminantium*) produce lactic fermented rapidly in the rumen and do not stimulate

acid, a stronger acid than the VFA. Large amounts of rumination. At the other extreme, hay and straw are lactic acid can predispose the animal to rumen stasis

chewed extensively to break down the fibre and their and to acidosis (see p. 829).

rate of digestion is also relatively slow. Hence a com- Protein is fermented to yield ammonia and VFA from promise with respect to saliva production is required; the carbon skeletons of amino acids. The ammonia may fibre is required together with concentrated feed be used by bacteria to synthesize new protein in their sources in order that saliva output is maintained.

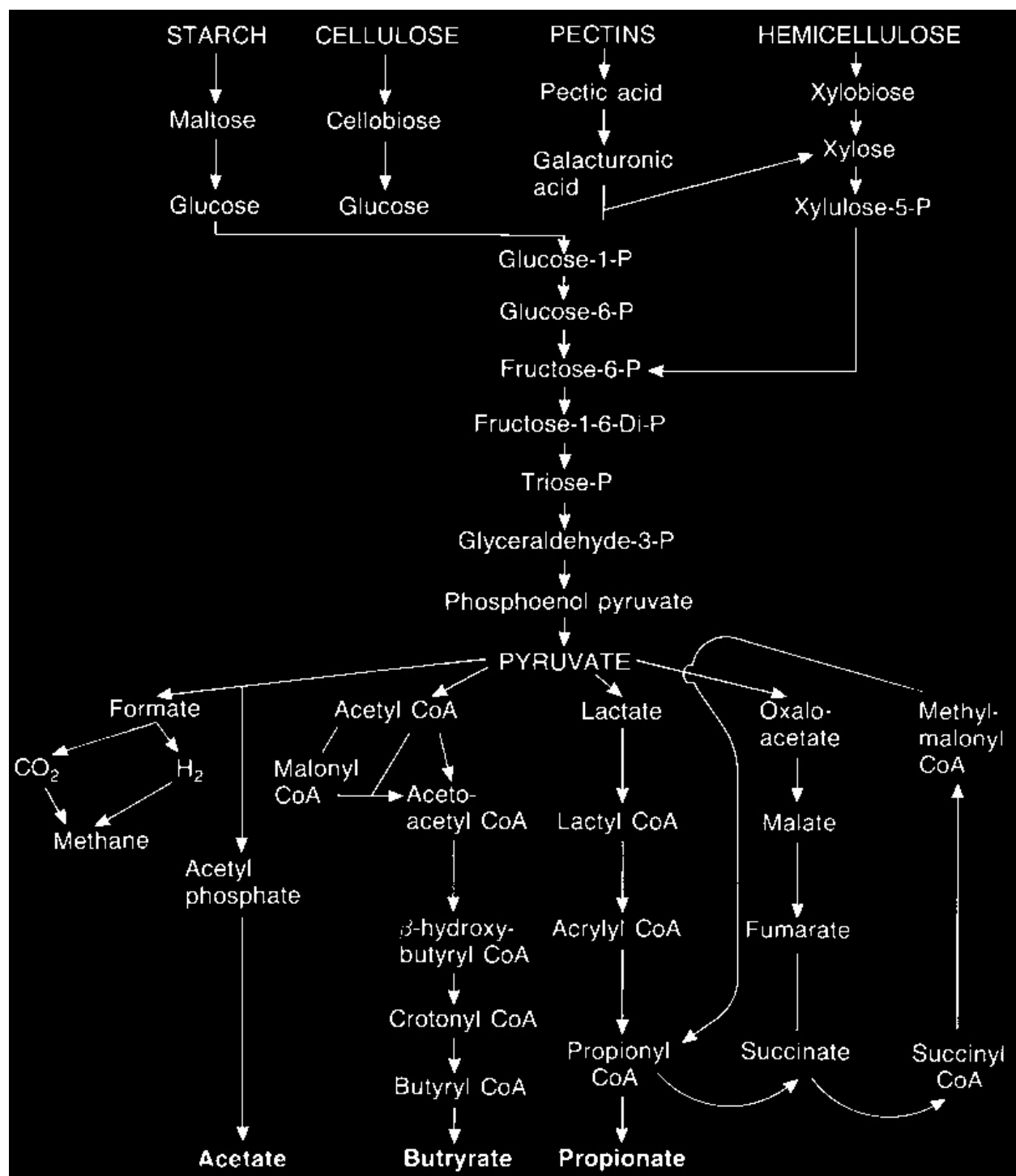
cells, but since bacterial growth is generally limited by the energy available from carbohydrate digestion, rather than from protein digestion, ammonia in excess

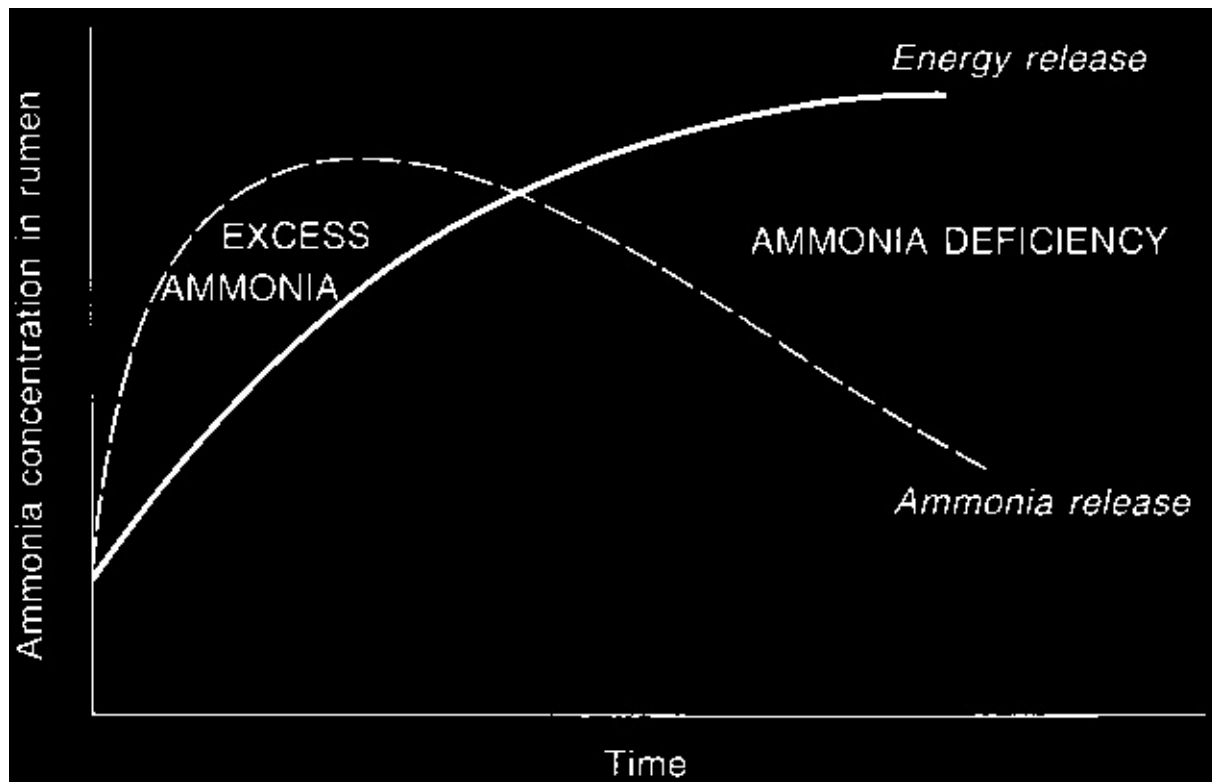
The actions of micro-organisms

of microbial requirements can easily be produced, The major organisms responsible for digestion in the especially on high-protein diets. Excess ammonia is rumen are anaerobic bacteria and protozoa, although converted, at an energy cost, to urea in the liver and anaerobic fungi are thought to be responsible for much excreted in urine. A deficit of ammonia in the rumen of the initial colonization of feed particles in the rumen.

slows down bacterial growth, reduces rate of digestion

The cellulolytic bacteria adhere to particles of fibrous and depresses feed intake. Thus the rate of release of feeds and secrete enzymes that gradually erode out the ammonia should match as closely as possible the digestible material. Their enzymes break down hemi-release of energy (see Fig. 9.2).





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Fig. 9.1

Chemistry of rumen fermentation.

liable to pass on down the tract and out in the faeces.

The rate of passage out of the rumen can influence the extent to which fibrous particles are actually digested.

The faster the rate of passage, the lower the actual, relative to the potential, digestibility.

The same concept applies to protein, especially that fraction which is available for microbial fermentation in the rumen. A high-yielding dairy cow, given a high-

quality diet with a fast rate of passage of feed through the rumen, will have a relatively lower fibre digestibility than a dry cow given less of the same diet, or a diet of lower energy content. However, the protein in the diet of the high-yielding cow will have a lower digestibility in the

Fig. 9.2

Typical patterns for the rates of release in the rumen of rumen, yield less ammonia and provide more unde-

ammonia from the breakdown of protein, and of energy, following graded protein to the abomasum than that in the dry

a meal. The rate of ammonia release should match as closely as cow's diet (see Table 9.13). Paradoxically, the lower the

possible that of energy, either by reducing the amount of quickly actual, relative to potential, digestibility in the rumen,

degraded protein in the diet, or by increasing the quantity of the worse off the animal is with respect to energy, but the

readily-fermentable energy (e.g. starch or sugar) in the diet.

better off it is likely to be with respect to protein.

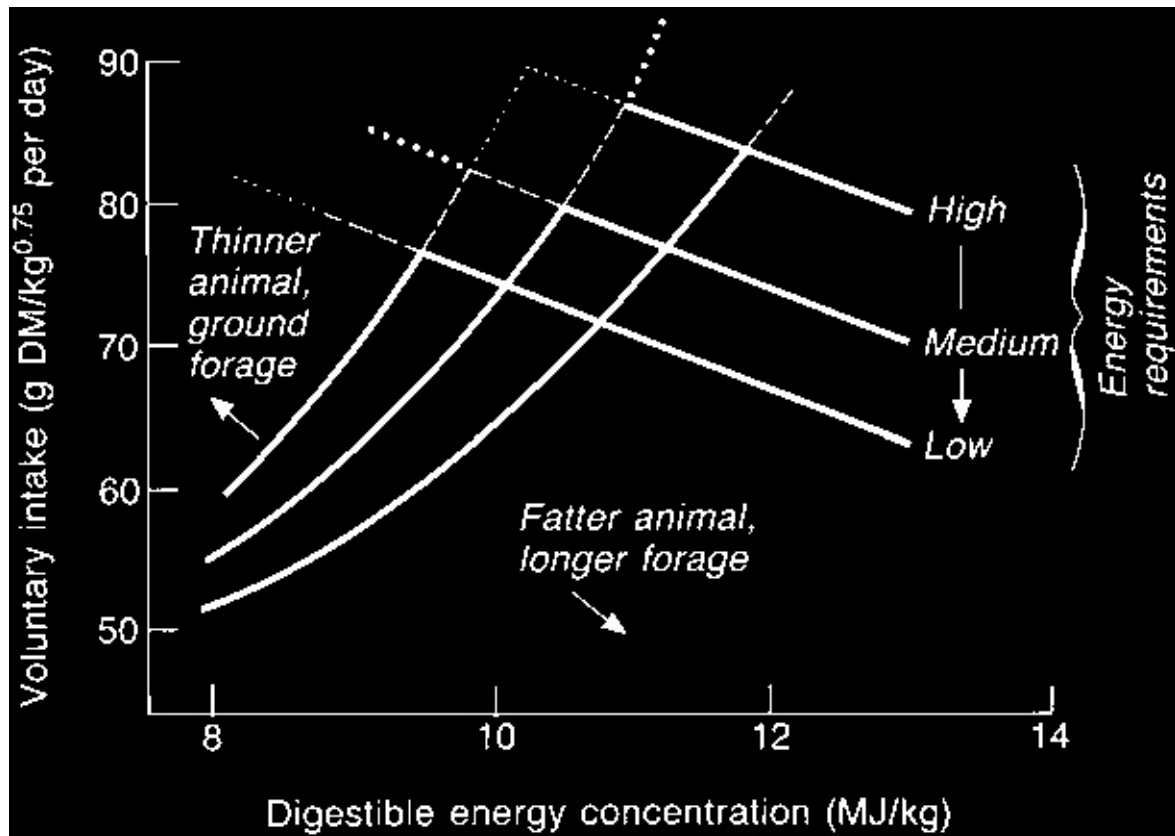
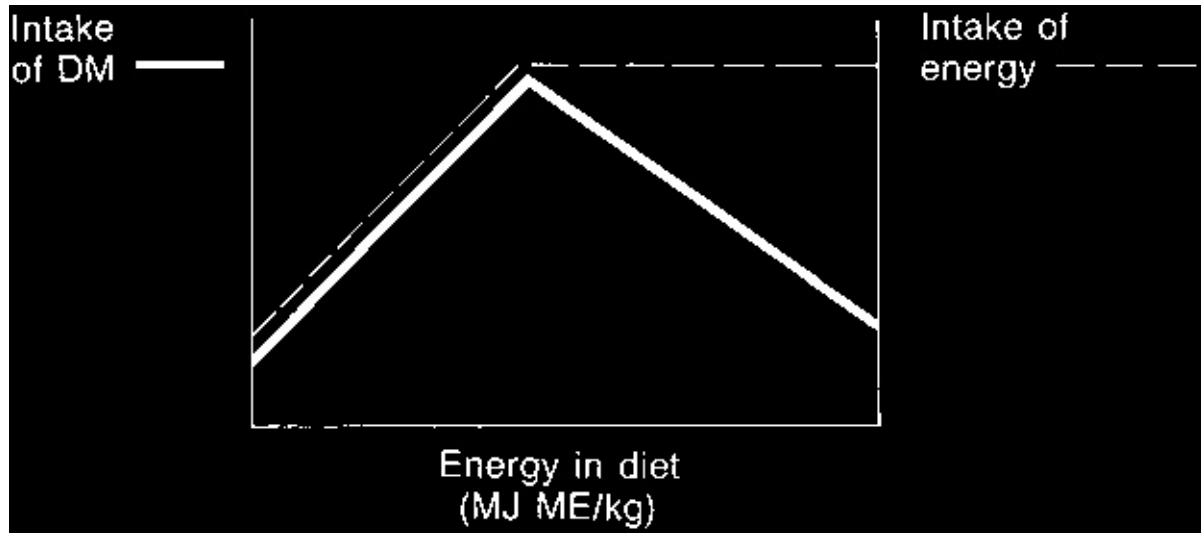
Actual and potential digestion

Optimizing digestion in the rumen

Fibre that has been reduced to a small enough particle

It follows from the above discussion that the following size at which it can pass out of the rumen, and which

principles need to be adopted in order to optimize the
has also resisted being digested by microbial activity, is
fermentation in the rumen:



- Cell wall is the principal source of digestible energy in the diet of the bovine, and conditions in the rumen should be optimal for its digestion.
- Sufficient saliva must be produced to maintain rumen pH above pH 6.5, otherwise cell wall digestion will be reduced.
- Adequate degradable protein must be supplied to meet the requirements for microbial protein synthesis (see section on protein requirements, p. 106).
- Supplementary energy in the form of starch or

Fig. 9.3

Simplified relationship between the energy content of sugar must not interfere with the maintenance of the diet and feed intake. As the energy content of the diet the above conditions in the rumen (see section on increases, dry matter intake will increase until it reaches a feeding management, p. 118).

maximum. If energy content increases further, dry matter intake is reduced because metabolic factors, rather than the bulk, or When cell wall digestion is optimized, then feed intake

‘fill’, of the diet now control intake.

is likely to be maximal, at least with respect to fibrous feeds (see below).

Feed intake

Most diets for cattle are offered in excess of the amount the animal actually consumes, although there are situations where the amount of feed offered each day is restricted intentionally. Two such situations are readily apparent: the dry cow and the suckler cow in late lactation. Both situations require that the animal does not overeat and become excessively fat.

The concept of voluntary feed intake is discussed in this section, that is, the amount of feed the animal will eat when offered an excess supply so that about 10 to 15 per cent of the daily amount offered is refused.

Fig. 9.4

Composite diagram of the relationships between intake and animal and feed factors in ruminants. At any given energy content, intake is higher for cows that have a greater

Is feed intake under control?

requirement for energy (or potential milk production). Also, intake of dry matter is greater the thinner the cow, and the smaller the The fact that cattle can become overfat suggests that

particle size of the feed. Source: Forbes (1983), reproduced with feed intake is under relatively imprecise control.

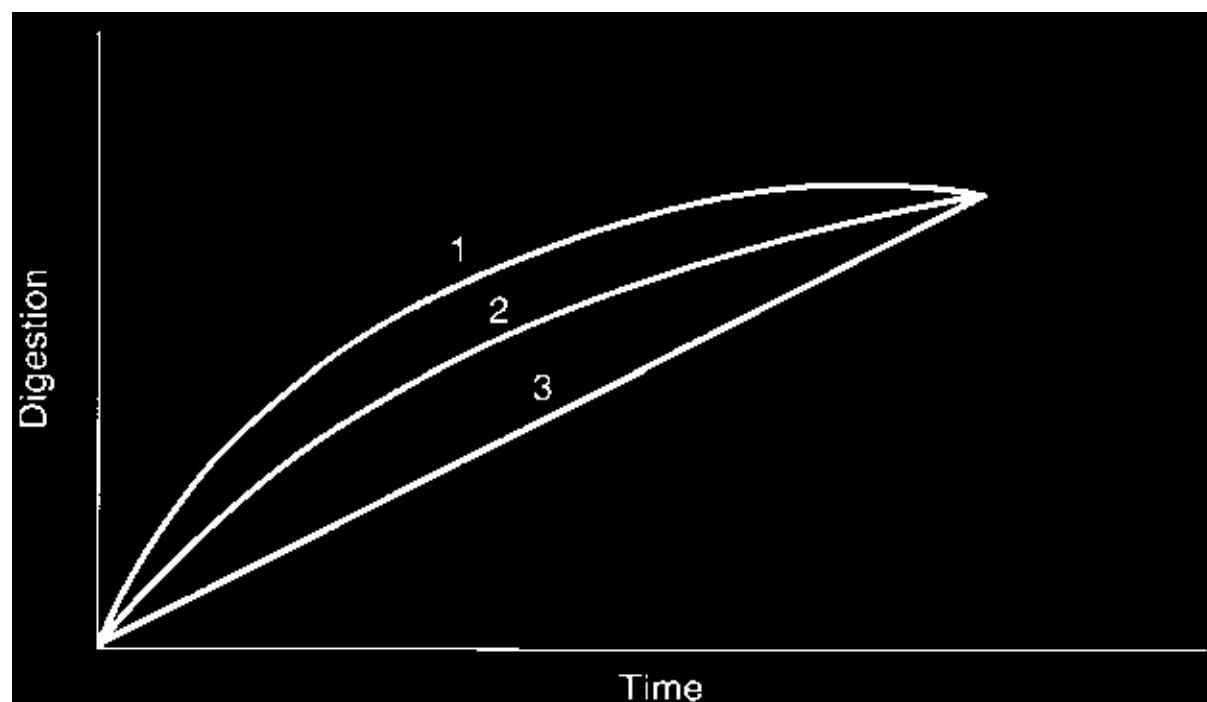
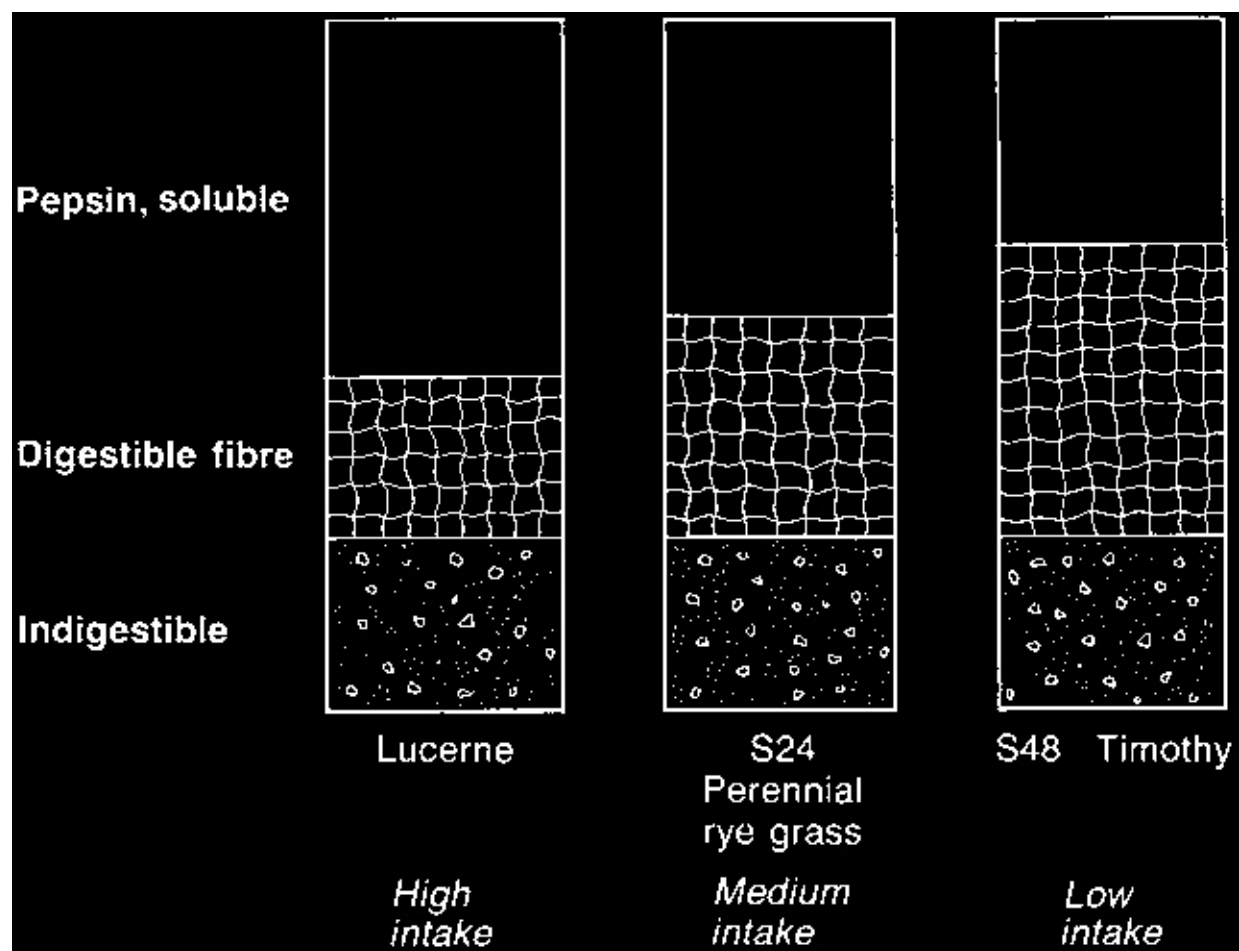
permission of CABI International.

Equally, sparse availability of range pastures or the provision of very low quality roughages as the sole feeds can lead to inadequate levels of feed intake and chronic undernutrition. The animal can suffer from (e.g. hypokalaemia), but the signals to cease deprivation because it is unable to ingest, or digest, eating may be physical (bulkiness of the food or restriction of rumen capacity by the fatness of the animal) or metabolic (concentrations of absorbed nutrients in blood), depending on the type of food. These concepts to maintain weight and produce tissue growth or milk. are discussed in more detail below.

The generalized relationships between the energy content of the diet and dry matter intake (Figs 9.3 and

Concentrates

9.4) suggest that signals received by the brain when the animal's energy needs are met in turn elicit the response to cease eating. Perhaps cattle eat as much as capacity of the rumen is reached. Intake is determined by the possible whilst at the same time attempting to minimize animal's capacity to metabolize the nutrients absorbed the total discomfort which may be caused physically or following digestion. Enhanced rate of metabolism, metabolically. Signals to commence eating may be for example following the administration of somatotrophic (for example, the concentration of a somatotrophin, is reflected in increased feed consumption of



high-energy diets where physical limitations do not apply (see below).

Roughages

With roughage feeds the volume of the rumen usually restricts intake. Thus intake is proportional to the volume of the rumen – the larger the rumen, the more feed is consumed. Larger animals eat more than smaller animals because their rumen volumes are greater.

Rumen capacity

Animals with large rumen capacities relative to their total body weight eat more roughage than those with relatively smaller rumen volumes. The implications are

Fig. 9.5

Variations in the ratio of material soluble in acid pepsin

that calves should be reared on diets that encourage

(cell contents) to digestible fibre (cell wall) in three forages of the rumen development, and cattle should be selected for

same digestibility, but differing in intake. Source: Osbourn (1967), large rumen capacities. Channel Island cattle (Jersey,

reproduced with permission of the British Grassland Society.

Guernsey) typically eat more food relative to their

body weight than Holstein cattle because, although they weigh much less, their rumen capacities are relatively greater. Thinner cattle tend to eat relatively more than fatter cattle (Fig. 9.4).

Digestibility of forages

Digestibility or energy concentration of forage feeds exerts a large influence on feed intake (Fig. 9.5).

However,

the relationship between intake and digestibility is much less evident for silages than for dried forages, where the pattern of fermentation in the silo, particularly the presence or absence of residual

Fig. 9.6

Three feeds with the same potential digestibility but different sugar and the pH value of the silage, can have an overriding influence on intake. The animal will eat most of feed 1 and least of feed 3. Reproduced from Orskov (1998) with permission of Chalcombe Publications.

sugar concentrations tend to be eaten in greater

amounts than more extensively fermented silages, possibly because they provide more readily available form of cell contents, principally sugars and proteins, nutrients to the rumen microbial population.

and in this regard it is nearer to a concentrate in terms of its speed of digestion in the rumen.

Cell walls are generally fermented at a slower rate

Speed of digestion

than cell contents. The actual rate of cell wall digestion

Speed of digestion also has an important influence on depends on initial particle size, the extent to which it is intake, since it determines the length of time the feed

broken down by rumination, and the extent to which it remains in the rumen. Legumes are digested at a faster

may be lignified. Three feeds with the same potential rate than grasses of the same overall digestibility, partly digestibility but different speeds of digestion are repre-

because they contain less cell wall than grasses (Fig.

sented in Fig. 9.6. The feed with the fastest speed of

9.5). Also, the structure of the cell walls of legumes

digestion will be eaten in the greatest amount by the

enables bacteria to gain access more rapidly than with animal.

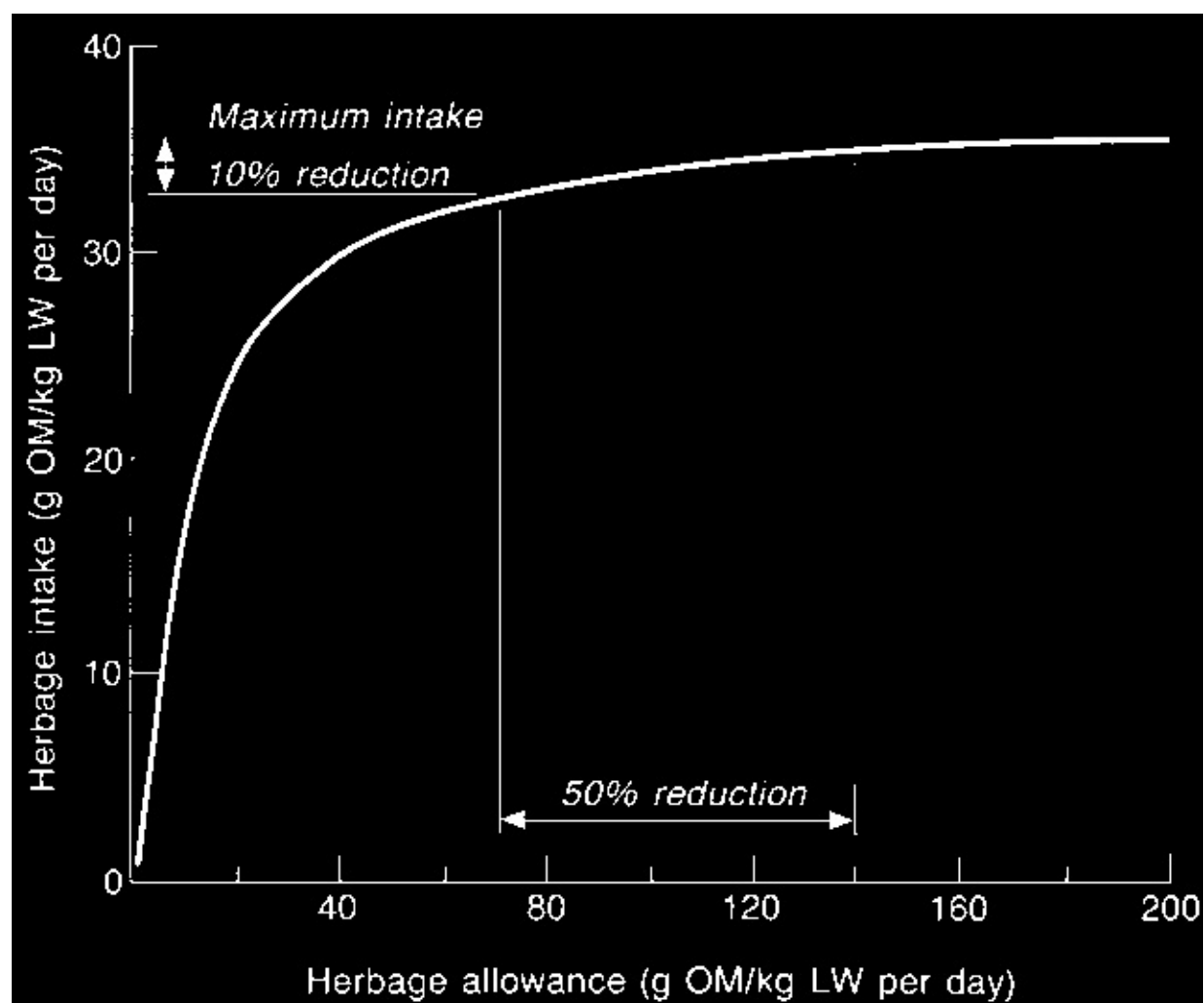
grasses.

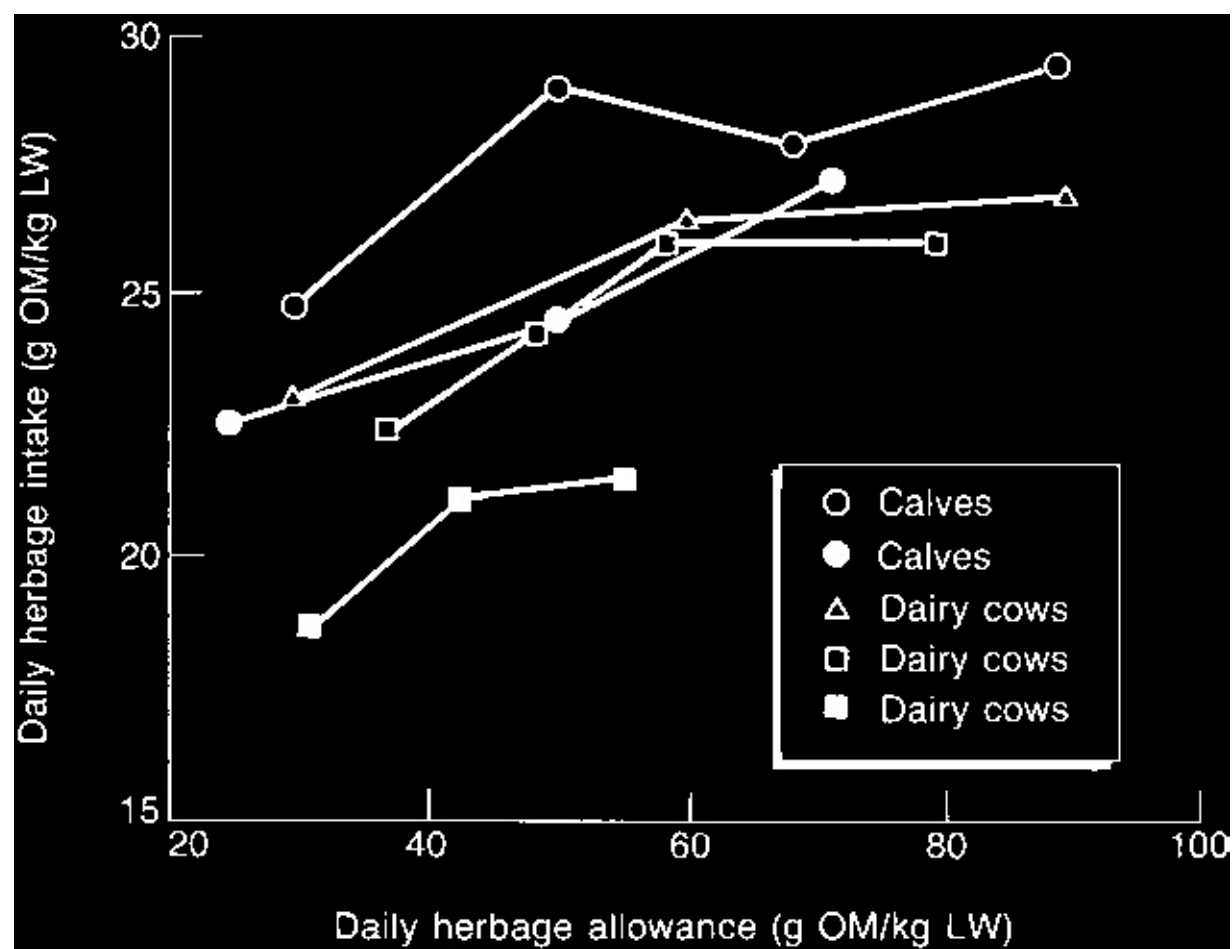
The cell contents of forages are fermented very

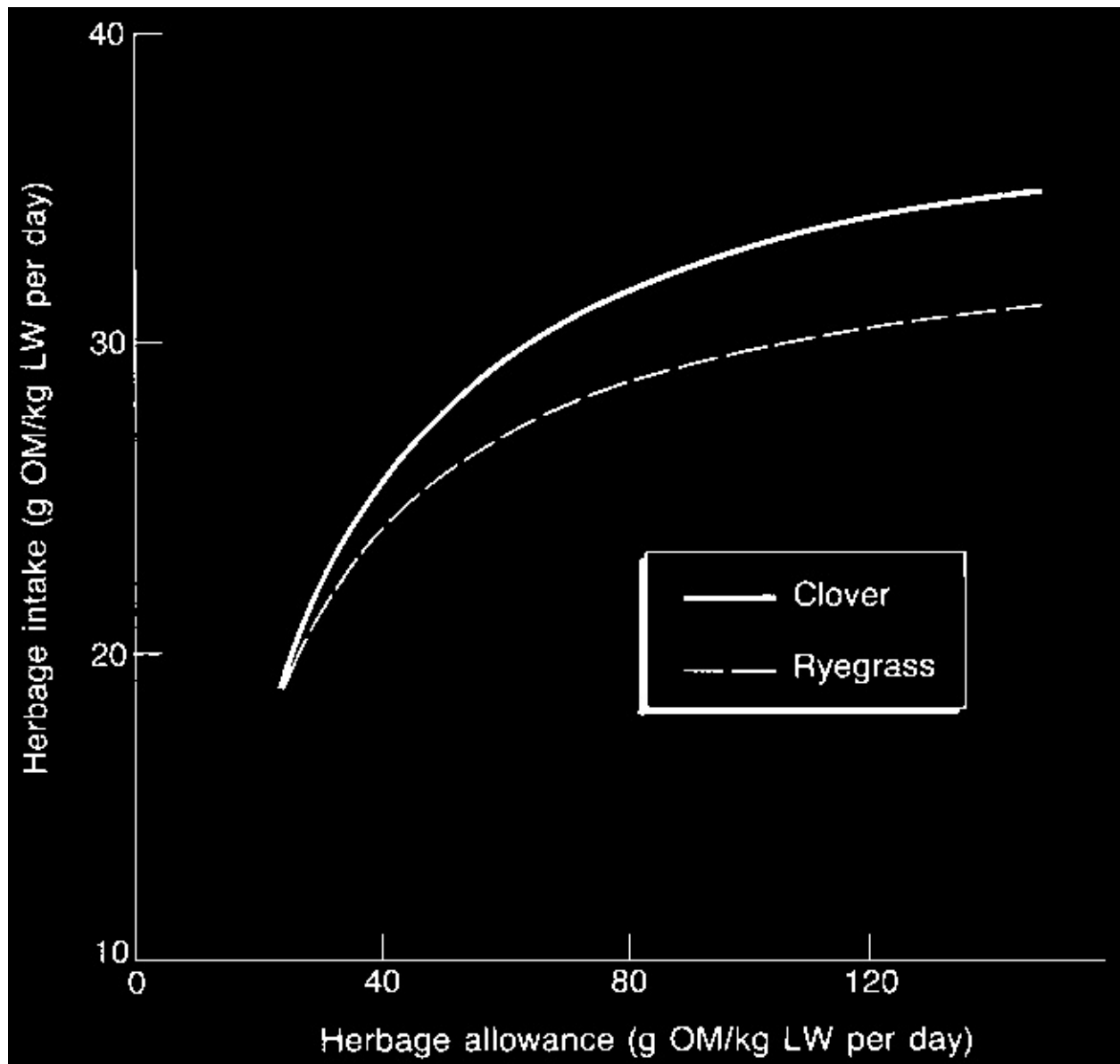
Grazed pasture

quickly in the rumen, provided they are released by eating and by rumination. For example, young grass

Unlike indoor feeding, grazing animals select what they may contain up to 40 per cent of its dry matter in the eat to a considerable extent, particularly when the







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Fig. 9.7

Relationship between daily herbage allowance and herbage intake in grazing lambs. Source: Hodgson (1975), reproduced with permission of the British Grassland Society.

Fig. 9.9

Herbage intake of lambs grazing perennial ryegrass

and red clover. Source: Gibb & Treacher (1978), reproduced with permission of Cambridge University Press.

be expressed per kilogramme liveweight rather than per hectare. It follows that stocking rate, a commonly used but empirical and inflexible way of describing herbage allowance, should not be expressed as number of animals per hectare, but as kilogrammes of liveweight per hectare (see section on feeding management, p. 118).

The relationship between herbage allowance and intake holds for animals in different physiological states, and for legumes as well as grasses (Fig. 9.9).

Studies of grazing behaviour and herbage intake by animals grazed on adjacent monocultures of grass and clover demonstrated that the grazing bovine has a

Fig. 9.8

Relationship between daily herbage allowance and strong preference for clover. Clover comprised 70% of

herbage intake in cattle. (Data from different studies.) Source: total dry matter intake, and this preference was greater

Hodgson (1975), reproduced with permission of the British

earlier in the day than during evening grazing, suggest that some discomfort may have been induced following large meals of clover which the animal herbage on offer is heterogeneous and is offered in attempted to attenuate by preferentially eating grass in excess of consumption. Thus a measure of the digestibility of the herbage on offer is of little use as an indicator of intake, since the animals are usually able to select

Probable levels of intake

material of higher digestibility than the average of that on offer.

Estimated levels of voluntary dry matter intake for Herbage intake under grazing is predominantly growing and lactating cattle are shown in Tables 9.2 and influenced by the amount on offer. To achieve 9.3. The relative intake of cows given the same diet maximum intake, the amount on offer should exceed throughout lactation, expressed as a percentage of the that actually consumed by three to four times (Figs 9.7

mean for the whole lactation, is shown in Table 9.4. The and 9.8).

important feature in Table 9.4 is the relatively low Intake is usually expressed relative to liveweight, and intake in the first month of lactation, when the demand it follows that the amount of herbage on offer should for nutrients for milk production is at its highest, and

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Table 9.2

Estimated feed intake of growing cattle (kg DM/day). Reproduced from Allen (2001) with permission of Chalcombe Publications.

Well-preserved

Poorly-preserved

Big bale

Maize

Hay

Barley

Concentrate

grass silage

grass silage

silage

silage

straw

Metabolizable

11.0

10.0

10.5

11.0

9.0

6.5

12.5

energy

(MJ/kg DM)

Liveweight

Forage intake (kg DM/day)

(kg)

200

4.3

3.6

3.9

4.5

3.6

2.4

6.0

300

5.8

4.8

5.2

6.1

4.8

3.2

8.0

400

7.2

6.0

6.5

7.6

6.0

4.0

9.0

500

8.5

7.1

7.7

9.0

7.1

4.8

9.5

600

9.7

8.1

8.8

10.3

8.1

5.5

10.0

Reduction of forage DM intake/kg concentrate DM fed

0.5

0.4

0.5

0.6

0.3

0.2

—

Table 9.3

Probable dry matter intake of cows in mid and late lactation (kg/day). From Ministry of Agriculture, Fisheries and Food (MAFF, 1984).

Liveweight

Milk yield (Y) (kg/day)

(W) (kg)

5

10

15

20

25

30

35

40

350

9.3

9.8

10.3

10.8

11.3

11.8

400

10.5

11.0

11.5

12.0

12.5

13.0

450

11.8

12.3

12.8

13.3

13.8

14.3

14.8

500

13.0

13.5

14.0

14.5

15.0

15.5

16.0

550

14.3

14.8

15.3

15.8

16.3

16.8

17.3

17.8

600

15.5

16.0

16.5

17.0

17.5

18.0

18.5

19.0

650

16.8

17.3

17.8

18.3

18.8

19.3

19.8

20.3

700

18.0

18.5

19.0

19.5

20.0

20.5

21.0

21.5

Note: In the first 6 weeks of lactation, reduce these values by 2–3 kg DMI/day.

Based on $\text{DMI (kg/day)} = 0.025 W + 0.1 Y$.

the consequent deficiency in intake of nutrients which

Table 9.4

Relative intake of dairy cows fed on the same diet
results in the mobilization of nutrients from body
throughout lactation (daily intake per cent of mean intake for tissues.
complete lactation). From ARC (1980), reproduced with permis-
sion of CABI International.

Energy requirements

Month

Relative

Month

Relative

intake

intake

Requirements and allowances

1

81

6

108

The notion of requirements takes no account of the

2

98

7

101

variation between animals when kept in groups. Hence,

3

107

8

99

if a group is given enough feed to meet the mean

4

108

9

97

requirement for a given performance, a proportion of

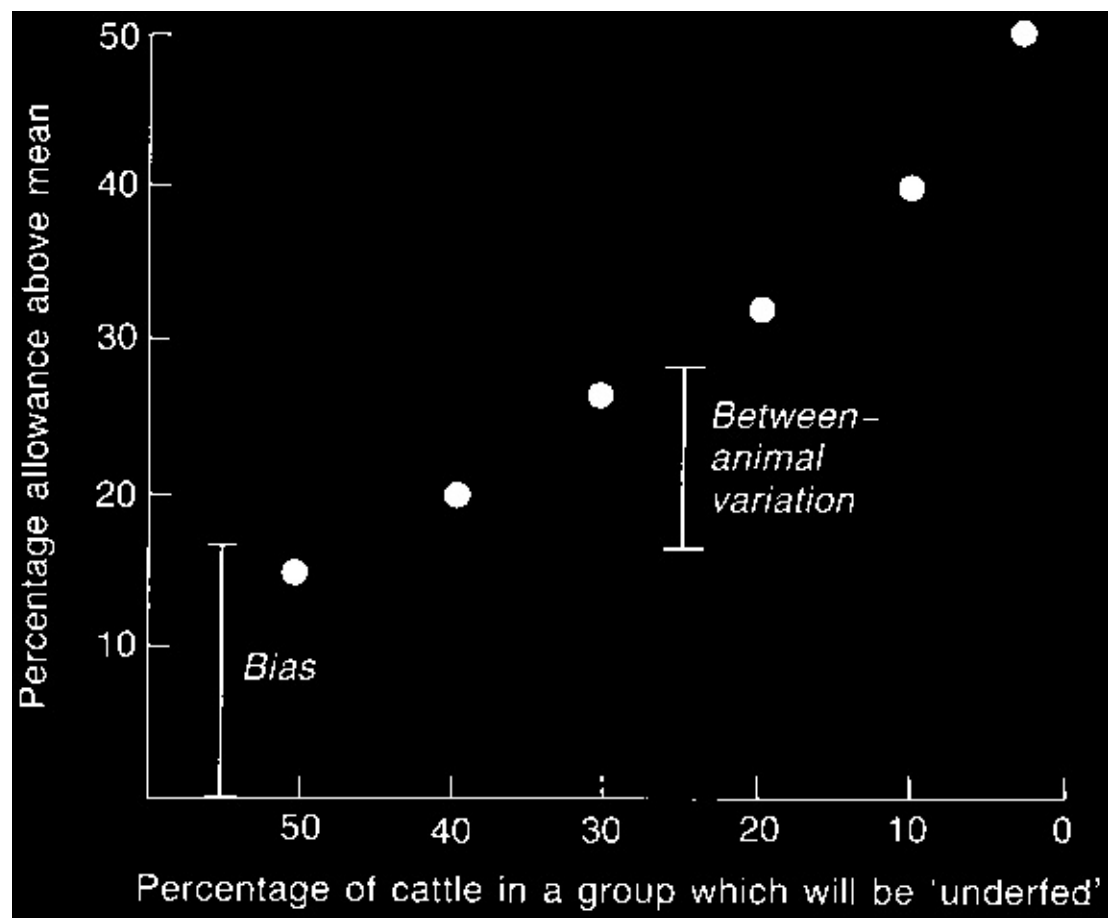
5

109

10

93

the group will be underfed and a proportion will be



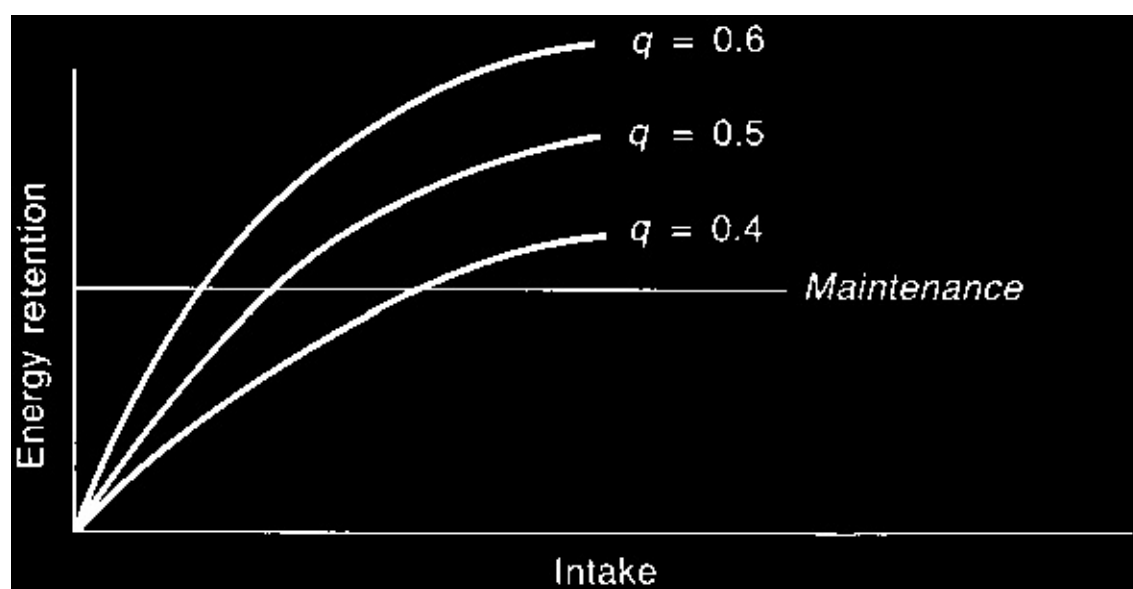
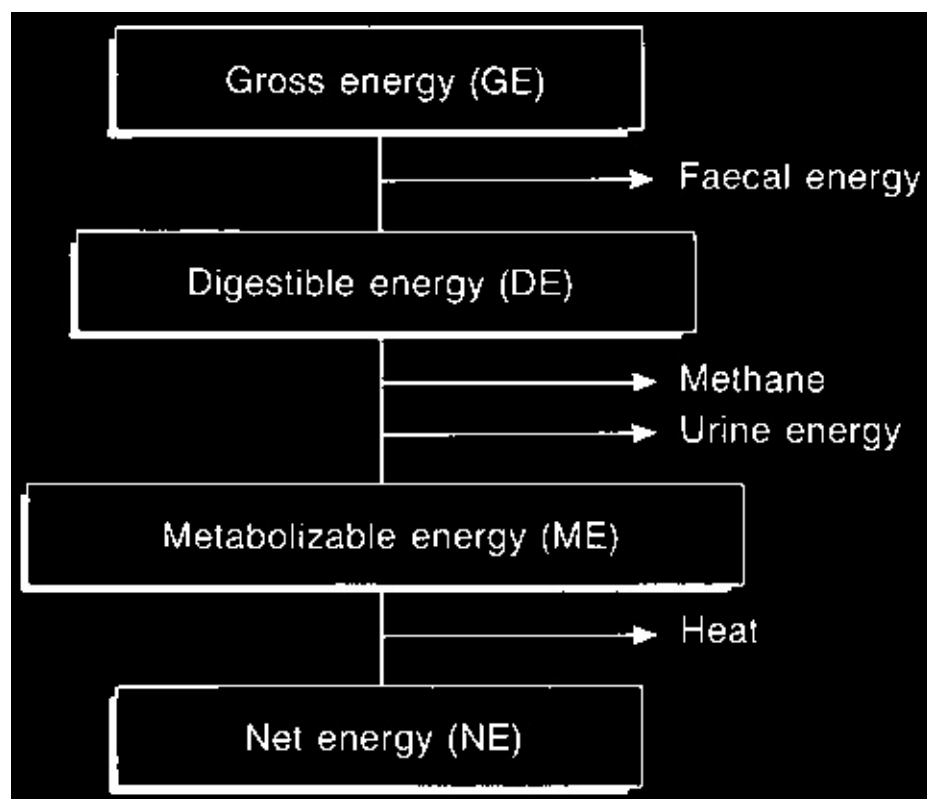


Fig. 9.11

Partition of energy.

Fig. 9.10

Estimated allowances above mean requirements

(ARC, 1980) required to meet the ME needs of a defined pro-

portion of beef cattle in a group. Additional allowance due to bias in the estimate of requirements is shown separately from that

due to estimated between-animal variation. Source: *Energy*

Group Report (1988) Inter-Departmental Working Party.

overfed. Since underfeeding is considered to be the

more serious failure of the system, allowances are made

so that only a small proportion of the group remains

Fig. 9.12

Efficiency of use of ME for maintenance (k_m) is rela-

underfed. If, as is the case with beef cattle, the metab-

tically high at 0.7. At twice the maintenance energy intake

(common level for growing animals) efficiency of use of ME for olizable energy system of assessing requirement overgrowth (k

predicts performance, an additional increment (15 per

$g) = 0.3$ to 0.5 for diets ranging from $q = 0.4$ to $q = 0.6$; k_g declines further at higher levels of energy intake and correc-cent) is added on to account for this inaccuracy. The

tion must be made for the curvilinear decline.

result (Fig. 9.10) is that allowances may exceed require-

ments by some 30 per cent. 'Requirements' in practical

feeding systems include a safety margin, although in most cases this margin is only 5 per cent and is lowest for feeds of low digestibility, such as straws, and arbitrary.

highest for concentrates.

Partition of feed energy

Efficiency of use of metabolizable energy

The proportion of the gross energy (GE) of a feed that

The proportion of ME used for maintenance and pro-

is absorbed by the animal depends on its digestibility.

ductive functions, such as growth or lactation, depends

The amount remaining for metabolism (metabolizable

on the efficiency with which it is utilized by the animal

energy, ME) is the digested energy less energy lost as

(k). This depends on q , the level of feeding above main-methane or in urine.

Further heat losses occur as a

tenance and on the productive purpose for which the

result of metabolism and the remaining energy (the

energy is to be used. Thus for growing cattle, k for

net energy) is that which is available to the animal for

maintenance (k_m) varies as shown in Fig. 9.12.

maintenance of body weight, for weight gain or for milk production. The partitioning is shown diagrammatically

Energy requirements of growing cattle

in Fig. 9.11.

In calculating energy requirements, the term ‘metab-

For growing cattle, a variable net energy system has

olizability’ (q) is used. This is an expression of ME/GE, been adopted to take account of the fact that k varies where GE is usually about 18.4 MJ/kg dry matter (DM)

with q . The system involves assessing the animal pro-

for conventional feeds. The greater the work of diges-

duction level (APL), which depends on the weight of

tion, the higher the heat lost as a result of this work and

the animal and the desired level of weight gain, the net

the lower the q value of a feed. Thus q is generally energy allowance for the particular animal and level of

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Table 9.5

Net energy allowances for maintenance and produc-

Table 9.7

Net energy values of feeds for maintenance and

tion NEm_p in growing beef cattle (MJ/day). From MAFF (1975).

production in growing beef cattle. From MAFF (1975).

Liveweight

Liveweight gain (kg/day)

APL

Energy concentration in feed (MJ ME/kg DM)

(kg)

0.25

0.50

0.75

1.00

1.25

1.50

8

9

10

11

12

13

14

100

14.7

17.4

20.7

24.6

Net energy value (NE/kg DM)

200

21.6

25.0

29.0

33.9

39.9

300

28.6

32.6

37.3

43.1

50.2

1.00

5.8

6.5

7.2

7.9

8.6

9.4

10.1

400

35.5

40.1

45.6

52.3

65.8

77.0

1.10

5.2

6.0

6.8

7.6

8.3

9.1

9.9

500

42.4

47.7

53.9

61.5

70.9

82.9

1.15

5.1

5.8

6.6

7.4

8.2

9.0

9.8

1.20

4.9

5.7

6.5

7.3

8.1

8.9

9.8

1.30

4.6

5.4

6.3

7.1

7.9

8.8

9.7

1.40

4.4

5.2

6.1

6.9

7.9

8.8

9.7

Table 9.6

Values for animal production level (APL) in growing

1.50

4.2

5.1

5.9

6.8

7.7

8.6

9.5

beef cattle. From MAFF (1975).

1.75

3.9

4.8

5.6

6.5

7.4

8.4

9.3

2.00

3.8

4.6

5.4

6.3

7.3

8.2

9.2

Liveweight

Liveweight gain (kg/day)

2.25

3.6

4.4

5.3

6.2

7.1

8.1

9.1

(kg)

APL

0.25

0.50

0.75

1.00

1.25

1.50

Table 9.8

Example of feed requirements for a 300 kg steer

growing at 0.75 kg/day on silage (ME 10) and barley (ME 13).

100

1.19

1.40

1.66

1.98

200

1.15

1.33

1.54

1.79

2.11

1

Dry matter intake (DMI) (simplified), $0.02 W = 6 \text{ kg/day}$ 300

1.13

1.29

1.47

1.70

1.97

2.33

2

Net energy for M + LWG (NEmp) (from Table 9.5),

400

1.12

1.26

1.43

1.64

1.90

2.22

37.3 MJ/day

500

1.11

1.25

1.41

1.60

1.84

2.15

3

Animal production level (from Tables 9.6), 1.46

NE + NE

4

Net energy values of feeds (from

m

p or

MEmp

gain, and the net energy value of a feed, which depends

Table 9.7)

on its ME content and the particular APL. The relevant

Silage ME 10 = 5.9 MJ/kg

Barley ME 13 = 8.6 MJ/kg

information is shown in Tables 9.5, 9.6 and 9.7. For two

5

Energy concentration in ration,

ingredient rations, a Pearson square is used to solve the

37 3

.

simultaneous equations to derive the amount of each

NE

DMI =

= 6 2

. MJ kg

mp

6

feed required in the diet (see example in Table 9.8).

6

Pearson Square to calculate ration:

Barley 8.6

0.3

0.3

.2

/ 7

. $\frac{6}{7}$ = 0.6

. 7 kg barley DM

Energy requirements of lactating cattle

6.2

As with growing cattle, the main determinant of the ME

Silage 5.9

2.4

.

2.4

.2

/ 7

. $\frac{6}{7}$ = 5.3

. 3 kg silage DM

requirement for maintenance is liveweight, but the ME

.

system for lactating cattle also recognizes that k_m varies 7

Divide by DM, $0.67/0.85 = 0.8$ kg fresh barley

with q (Fig. 9.13). A further important consideration is $5.33/0.25 = 21.3$ kg fresh silage

the composition of the milk produced by the cow. The higher the fat and protein concentration, the higher the milk energy concentration and the greater the requirement for ME for milk production at any given dietary

The requirement for ME to support the growth of the

q value. However, in most practical feeding situations fetus during pregnancy increases progressively with

the q of diets for lactating dairy cows is unlikely to vary the duration of gestation. For the first half of gestation

significantly from about 0.7, because of the need to

the requirement is negligible, and in practice the energy

include high-energy forages and concentrates to meet

requirement of the fetus only assumes significance in

the total energy requirement. The values for ME

the last two months of pregnancy, i.e. in the dry period.

required for milk production in Table 9.9 relate to a q

The requirement for ME for pregnancy (Fig. 9.14)

value of 0.7 for simplicity. If the diet has a q value lower assumes a 40 kg calf at term. The requirement for

than 0.7, then the requirement should be increased

heavier calves, such as Charolais ¥ Holstein, is increased

accordingly (Table 9.9).

by direct linear scaling.

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75

73

70

69

69

65

65

65

66

61

62

62

60

57

58

59

55

54

Ration quality

55

$q = 0.50$

50

52

$q = 0.60$

$q = 0.70$

Maintenance requirements (MJ ME/day)

45

40

Fig. 9.13

Metabolizable energy require-

500

550

600

650

700

ment for maintenance in lactating cattle.

Liveweight (kg)

Source: AFRC (1992).

Table 9.9

Metabolizable energy requirement (MJ/litre) for milk

world the energy expended in activity is considered

production. Reproduced from Chamberlain & Wilkinson, 1996

to be moderate and the above requirements relate to

with permission of Chalcombe Publications.

that situation. However, cattle are sometimes subjected

to extensive foraging under range and semi-arid

Milk fat (%)

Milk protein (% per litre)

conditions, and account should be taken of the

per litre)

increased energy required for activity when assessing

3.00

3.20

3.40

3.60

requirements.

The total ME required is calculated as the sum of the

$$q = 0.6$$

3.00

4.39

4.47

4.54

4.62

above factors, but the overall efficiency of energy uti-

3.50

4.71

4.79

4.86

4.94

lization of the lactating animal declines with increasing

4.00

5.03

5.11

5.18

5.26

level of production, partly because of the faster rate of

4.50

5.35

5.43

5.50

5.58

passage of food through the digestive tract at higher

5.00

5.67

5.75

5.82

5.90

levels of voluntary intake and partly because the

processes of absorption and metabolism are less effi-

$q = 0.7$

cient at higher levels of output. The correction for ME

3.00

4.16

4.23

4.30

4.37

required depends on the animal production level

3.50

4.47

4.54

4.61

4.68

(APL), defined here as the total ME required divided

4.00

4.77

4.84

4.91

4.98

by the ME required for maintenance. Correction

4.50

5.07

5.14

5.21

5.28

factors for level of production are in Table 9.11.

5.00

5.38

5.45

5.52

5.59

q = gross energy/metabolizable energy.

Protein requirements

The net energy value of weight change is assumed to

Traditionally, protein requirements were expressed as

be relatively constant in the case of adult cattle, but the

digestible crude protein (DCP), that is, the proportion

impact of weight loss on the ME requirement varies

of the crude protein (CP or total nitrogen multiplied by

according to the presumed utilization of the body tissue

6.25) that is apparently digestible and therefore avail-

catabolized – milk production in early lactation and

able to the animal. It is now recognized that DCP is

growth of the fetus in the dry period. The ME value of

totally inadequate as a system for assessing the protein

weight change also varies with the q value of the diet requirements of ruminants.

(Table 9.10).

The concept of digestible crude protein ignored the

The energy requirement of the cow is also influenced

fact that a proportion of the digestible protein is

by the amount of activity, especially walking, that the degraded to ammonia by the action of the rumen micro-animal undertakes daily. In most temperate areas of the organisms. Some of this ammonia is synthesized into

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50

45

44

40

35

30

25

22

20

15

11

10

5.5

Metabolizable energy requirement (MJ/day)

5

2.8

0.3

0.7

1.4

0

0

5

10

15

20

25

30

35

40

Fig. 9.14

Metabolizable energy require-

Week of pregnancy

ment for pregnancy. Source: AFRC (1992).

Table 9.10

Metabolizable energy value (MJ ME) of weight

Table 9.11

Correction factors for metabolizable energy require-

changes in dairy cows given diets of different qualities (q).

ment according to the animal production level (APL). Repro-

Reproduced from Chamberlain & Wilkinson, 1996 with permis-

duced from Chamberlain & Wilkinson, 1996 with permission of sion of
Chalcombe Publications.

Chalcombe Publications.

Live weight change (kg/day)

APL

Correction factor

q

-1.00

-0.50

0.50

1.00

0.7

0.99

1.0

1.00

Lactating cows

2.0

1.02

0.5

-23.3

-11.6

19.4

38.8

3.0

1.04

0.6

-22.0

-11.0

18.3

36.7

4.0

1.05

0.7

-20.8

-10.4

17.4

34.7

5.0

1.07

Dry, pregnant cows

0.4

-24.8

-12.4

34.5

69.0

APL = total ME/ME_{maint}

0.5

-24.8

-12.4

27.7

55.4

the animal's requirement (Table 9.12). Most classes

q = gross energy/metabolizable energy.

of ruminant livestock can fulfil their total requirement

for metabolizable protein (MP) from the supply of

microbial protein in the rumen; the rest is absorbed into

microbial true protein (MTP) alone. The exception is

the bloodstream, converted to urea in the liver and

the high-yielding dairy cow, where supplementary

excreted in the urine.

digestible undegraded feed protein is required to meet

There are many situations where the animal's

the animal's total requirement for metabolizable

requirements can be met entirely by microbial protein.

protein (see below). In this situation, there may also be

However, in some situations, particularly with high-

a requirement for supplementary essential amino acids

yielding dairy cows, the animal requires more protein

such as methionine and lysine.

than that supplied by the microbial cells and it is nec-

Much of the dietary protein eaten by the animal is

essary to supply additional dietary protein to the abo-

degraded in the rumen to ammonia by the microbial

masum that has not been broken down *en route* through

population and it is possible to include non-protein

the rumen.

nitrogen, such as urea, in diets which are deficient in

degradable protein. Such deficiencies might arise in

situations where the dietary ingredients, such as straw

Microbial protein synthesis

or maize silage, are low in total protein, or where the

Microbial protein is of high value to the ruminant in degradability of the feed protein is relatively low, as a that its balance of essential amino acids is very close to result of heat-treatment during processing.

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Table 9.12

Amino acid composition of bacterial protein and FME is therefore the total metabolizable energy animal proteins (g amino acid/100 g protein). From Van Soest minus the gross energy content of the lipids and (in the (1994).

case of fermented feeds like silages) the content of fermentation acids. The yield of microbial crude protein

Amino acid

Microbial

Milk

Beef

(MCP) per MJ of FME, known as Y, depends on the

protein

APL because at lower APL the outflow rate from the rumen is reduced and at the lower outflow rate bacte-

Isoleucine

5.8

5.6

5.1

ria and protozoa die before passing out of the rumen

Leucine

8.0

10.2

8.0

Lysine

9.2

8.2

9.1

and are digested by other rumen microbes. Their

Methionine

2.5

2.9

2.7

protein is recycled to produce new microbial protein.

Cysteine

1.4

1.0

1.3

However, this process requires energy so additional

Phenylalanine

5.3

5.4

4.5

FME is utilized with no net increase in yield of

Tyrosine

4.9

4.5

3.8

microbial protein.

Threonine

5.7

5.0

4.6

The limit to the total yield of MCP is either the

Tryptophan

1.5

1.4

1.3

supply of ERDP (g per day) from the diet itself, in

Valine

5.8

7.4

5.3

which case MCP production equals the supply of

Arginine

5.3

4.0

6.7

ERDP. Or, when the limit to the total yield of MCP is

Histidine

2.1

3.0

3.7

FME, the total amount of MCP produced is equal to

Alanine

6.8

3.8

6.4

the yield of MCP per MJ of FME (Y, which ranges from

Aspartic acid

11.9

8.5

9.6

Glutamic acid

12.4

23.0

17.3

8 g MCP/MJ FME at an APL of 1.0 to 11.5 at an APL

Glycine

5.4

2.2

5.6

of 4.0) multiplied by the total supply of FME in MJ

Proline

3.6

9.4

5.1

per day. The *lesser* of the two values is taken as the Serine

4.7

5.9

4.5

production of MCP in the rumen.

It is unusual to find a situation where the two limiting factors to the production of MCP – ERDP and FME – are equal and where the calculation of MCP produc-

The production of microbial protein in the rumen tion by the two methods gives the same result. For depends on the supply of degradable protein and the example, most grass silages contain an excess of ERDP supply of fermentable metabolizable energy (FME).

and a deficit of FME, whilst maize silage has a deficit of

The supply of degradable protein depends on the feed ERDP relative to FME. The skill in formulating diets source and also on the time the feed spends in the

is to achieve the correct overall balance between the rumen. Rumen outflow rate depends on the APL. The total supply of ERDP, the total supply of FME (see Figs APL is therefore higher for high-producing animals

9.16 and 9.17 for examples of the ERDP and FME con- than for low-producing animals. Degraded protein is not

tents of different feeds) and their respective rates of used with complete efficiency for microbial protein synthesis in the rumen.

thesis because some of the quickly degraded fraction is Having calculated the production of MCP, this value lost as ammonia through the rumen wall and is converted to urea in the liver. The proportion of quickly 0.75 of MCP is true protein and that it is absorbed into degraded protein (QDP) which is lost from the rumen is the blood with an efficiency of about 0.85 (Fig. 9.15). presumed to be 0.2, so that only 0.8 of the QDP is available. Thus microbial true protein supply is only about 0.64 of the MCP produced.

degraded protein (SDP) is potentially available to the micro-organisms and, together with 0.8 of the QDP, is the *effective* rumen degradable protein (ERDP) and is

Metabolizable protein

the amount of protein (or nitrogen) available for microbial growth and metabolism. However, the amount of

The digestion and metabolism of dietary protein are ERDP which the microbial population can utilize shown in simplified form in Fig. 9.15. Dietary crude depends on the amount of energy available, known as protein (CP) (Total N \times 6.25) contains true protein, the fermentable metabolizable energy or FME. Some polypeptides, peptides and non-protein nitrogenous sources of energy in foods are considered to be of low value to the microbes, particularly those which yield low and ammonia. Urea, recycled to the rumen in saliva, is levels of ATP during their digestion in the rumen. Thus produced in the liver from ammonia which is in excess the energy in lipids and in silage acids is discounted, and to that used by the microbial population of the rumen the energy in undegraded protein should also be discounted to synthesize microbial protein (see above). The amino acid content of microbial true protein is very constant,

RDP

UDP

QDP

SDP

ADIN

0.80

1.00

0.90

ERDP

FME

Y

DUP

MCP

0.64

Faeces

MP

Fig. 9.15

The metabolizable protein

1.00

0.59

1.00

0.85

0.68

0.26

system. Reproduced from Chamberlain &
Wilkinson (1996) with permission of Chal-

NP

NP

NP

NP

NP

NP

Maintenance Weight gain

Weight Loss

Pregnancy

Lactation

Wool

combe Publications.

and similar to the amino acid composition of tissue

Table 9.13

Example of differences in the proportion of
protein (beef) and also of milk (Table 9.12).

degraded and undegraded soyabean meal protein according to The degradable fraction, or ERDP, which yields class of livestock. Reproduced from Orskov, 1998 with permission of Chalcombe Publications.

protein to the animal (Fig. 9.15). The protein which is

Dairy cows

Growing cattle

Suckler cows

not degraded in the rumen is termed undegraded dietary protein, of which a proportion, insoluble in

ERDP

DUP

ERDP

DUP

ERDP

DUP

acid detergent (ADIP or ADIN ¥ 6.25), is indigestible.

The remainder is known as the digestible undegraded

0.50

0.50

0.65

0.35

0.85

0.15

protein or DUP.

The extent of degradation depends not only on the

ERDP = effective rumen degradable protein.

inherent characteristics of the protein source, but also

DUP = digestible undegraded protein.

on the time the material is exposed to degradation in

the rumen, i.e. on outflow rate from the rumen. Thus

the same protein may have quite different values when

The higher the APL, the faster the rate of passage of

given to different types of cattle (Table 9.13).

feed through the rumen. Thus MP supply depends not

The supply of MP to the animal has two components

only on the feed itself but also on the type of animal to

(Fig. 9.15): MTP derived from the growth of bacteria

which the feed is being given. The requirement of the

and protozoa in the rumen (see the section on micro-

animal for MP depends on its level of productivity. The

bial protein above) and digested undegraded feed major requirement for MP in adult female ruminants is protein (DUP), which has passed intact through the rumen and has been digested in the abomasum together with the microbial protein. The rate of production of growth, including the growth of the fetus during pregnancy, and for the production of wool. MP is released from the breakdown of muscle tissue during periods of body weight loss. MP is presumed to be utilized with variable efficiencies (net protein, NP) depending on its the speed of flow of digesta through the rumen (Table 9.13). The speed with which feed particles pass through the rumen depends mainly on the APL of the animal.

tation (0.68), growth (0.6) and wool (0.26).

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Table 9.14

Metabolizable protein (MP) requirement for main-

Table 9.16

Metabolizable protein (MP) requirement for preg-

tenance. Reproduced from Chamberlain & Wilkinson, 1996 with nancy.

Reproduced from Chamberlain & Wilkinson, 1996 with

permission of Chalcombe Publications.

permission of Chalcombe Publications.

Live weight (kg)

MP requirement for

Week of gestation

MP requirement (g/day)

maintenance (g/day)

5

1

400

216

10

3

450

236

15

8

500

256

20

17

550

275

25

34

600

293

30

64

650

312

35

114

700

329

40

191

750

347

Table 9.17

Metabolizable protein (MP) allowances for weight
change in dairy cows. Reproduced from Chamberlain & WilkinTable 9.15

Metabolizable protein (MP) requirement for milk
son, 1996 with permission of Chalcombe Publications.

production. Reproduced from Chamberlain & Wilkinson, 1996
with permission of Chalcombe Publications.

Live weight change (kg/day)

MP requirement (g/day)

Milk yield

Milk protein (% per litre)

-1.0

-131

(litres/day)

-0.5

-66

3.00

3.20

3.40

3.60

0

0

0.5

122

MP requirement (g/day)

1.0

245

10

427

456

484

513

20

855

912

969

1026

30

1282

1368

1453

1539

The protein requirement of the bovine animal is

40

1710

1823

1937

2051

generally considered in terms of the main activities of

50

2137

2279

2422

2564

the body – the maintenance of essential functions, lactation, pregnancy and weight change. The requirements of cattle of different liveweights for metabolizable protein for maintenance are shown in Table 9.14.

The requirement for MP for lactation is quantita-

Requirements for metabolizable protein

tively the most important and varies with milk yield and

The requirement of the animal for MP depends on the

also, though to a much lesser degree, with the protein

amount of protein produced in tissue growth or in milk.

concentration in the milk. Values for the requirement

The young calf has a high rate of lean tissue growth

for MP for milk production are shown in Table 9.15.

relative to its feed intake. Hence the concentration of

The requirement for MP to support fetal growth in

protein in its diet needs to be relatively high. The

pregnancy is very low in the early stages and only

cow yields more milk and eats less feed in early lactation

becomes of significance in the final two months of gesta-

tion (Table 9.16). Allowances for MP for weight loss and

the milk is higher than that consumed in the feed

the requirement for weight gain are shown in Table 9.17.

because the cow is using stored reserves, mostly body

The total requirement for MP is calculated as the sum

fat. However, fat from body reserves yields no metabolizable protein and as a result she requires more protein of the requirements for the appropriate bodily functions. Thus, for a dairy cow weighing 600 kg liveweight, per unit of feed in early lactation than later on.

yielding 20 litres of milk of 3.2% protein, in her

If sufficient energy is provided to maintain the

fifteenth week of pregnancy and gaining 0.5 kg

animal, then microbial protein produced from ERDP is

liveweight/day the total requirement is 293 g for main-

likely to be sufficient to meet the maintenance require-

ment for MP. On the other hand, if the animal is

9.15) + 8 g for pregnancy (Table 9.16) + 122 g for weight

restricted to a submaintenance level of feeding, then it

gain (Table 9.17) = 1335 g MP/day.

will lose not only body fat but also protein from muscle

In some situations the total protein requirement is

tissue.

expressed as a recommended concentration of crude

Table 9.18

Recommended concentrations of crude protein in diets for cattle.

Dairy cows

Beef suckler cows

Growing beef cattle

Milk yield

Dietary

Milk yield

Dietary

Liveweight

Dietary crude

(litres/day)

crude protein

(litres/day)

crude protein

(kg)

protein (g/kg

(g/kg dry

(g/kg dry

dry matter)

matter)

matter)

ME of total diet

(MJ/kg DM)

11.0

12.0

0

135–145

0

120

100

180

210

10

145–155

5

150

200

140

150

20

155–165

10

150

>200

130

140

30

165–175

40

175–180

50

180–190

protein in the total diet dry matter. This may appear to

Table 9.19

Classification of feeds. From MAFF (1986b) and

be an oversimplification, but where the degradability

Lonsdale (1989).

characteristics of the protein in some dietary ingredi-

Mainly cellulosic

Mainly non-cellulosic

ents are not known, or where there is uncertainty about

(NDF > 500 g/kg DM)

(NDF < 500 g/kg DM)

the actual animal production level, then diets should be formulated according to the recommended concentra-

Straw

Kale

tions of crude protein in Table 9.18.

Hay

Very young grass

Tests of the MP system with dairy cows have revealed

Grass (except very young

Maize silage

that the system works reasonably well at low to medium grass)

Fodder beet, root crops

levels of milk yield (20 to 30 litres/day), but that at higher Silage (except maize)

Cereal grains

levels of production the MP required is underestimated.

Brewers' grains

Molasses

This implies that the efficiency of utilization of MP for

Malt distillers' draff

Molassed sugar beet pulp

milk production is not constant at 0.68 (see section on

Pectin-extracted fruit

Maize gluten feed

metabolizable protein), but that it decreases at increased

Coffee grounds

Soyabean meal

Unmolassed sugar beet pulp

Fat

levels of output. The values for MP requirement for lac-

Bran

Cottonseed cake

tation in Table 9.15 have been adjusted upwards to take

Wheat feed

Distillers' dark grains

account of the decrease in efficiency of utilization of MP

Citrus pulp

for milk production above a milk yield of 25 litres/day.

Legume seeds

Other systems of assessing protein supply and require-

ments, for example the French PDI system, also take

account of the relatively greater requirement for MP of different types of bacteria and at different rates (see the high-yielding cow.

section on fermentation in the rumen, p. 96). Cellulosic feeds are fermented at a slower rate, occupy more

Composition of feeds

space in the rumen and are usually eaten in smaller quantities (i.e. at a slower rate) than non-cellulosic feeds.

Cellulosic and non-cellulosic feeds

When formulating diets it is useful to recognize the Feeds for cattle are best described in terms of the different fermentation patterns of the two types of feed major components that undergo fermentation in the and their different rates of intake by the animal. With rumen. In other words, the conventional division into productive cattle it is important to avoid too much of concentrates and roughages is only a crude way of one type, or intake may be depressed – by acidosis if distinguishing between feeds that contain mainly too much non-cellulosic material is eaten, or by a slow

non-cellulosic (starch, sugar and protein) or cellulosic fermentation in the rumen and slow outflow rate if too (plant cell wall) material.

much cellulosic material is eaten.

The division into cellulosic and non-cellulosic feeds

Feeds are classified into those which are mainly cel-

is relevant because the two fractions are fermented by

lulosic and those which are mainly non-cellulosic, as

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Table 9.20

Classification of raw materials according to their energy and protein contents. The degradability of the protein is also indicated*. Reproduced from Lonsdale (1989) with permission of Chalcombe Publications.

Protein

Metabolizable energy content (MJ/kg DM)

content

(g/kg DM)

High > 12.0

Medium 9.0–12.0

Low < 9.0

High

Maize gluten meal (prairie meal) (B)

Rapeseed meal (B)

Cotton cake (undec.) (B)

> **200**

Groundnut cake (A)

Sunflower seed meal (B)

Sunflower seed meal (undec.) (B)

Soyabean meal (B)

Cottonseed cake (B)

Safflower meal expeller (A)

Sesame meal (B)

Safflower meal (A)

Soya beans (whole processed) (C)

Malt culms (B)

Condensed corn steep liquor

Brussels sprout packhouse waste (A)

Lupins (sweet) (B)

Malt residual pellets (B)

Pot ale syrup (A)

Brewers' grains (B)

Linseed meal (B)

Palm kernel meal (extr.) (B)

Spent wash syrups (A)

US corn distillers' dark grains (C)

Beans (field) (C)

Wheat distillers' dark grains (C)

Malt distillers' dark grains (C)

Peas (B)

Delactosed whey syrup (A)

Copra expeller (B)

Maize gluten feed (B)

Medium

Maize germ meal (B)

Wheat bran (B)

Rice bran (C)

120–200

Whey (A)

Dried forages (grass, lucerne) (B)

Shea nut meal (D)

Triticale (A)

Wheatfeed (A)

Rape meal (D)

Wheat (A)

Low

Barley (A)

Pectin extracted fruit (B)

Oatfeed (C)

< **120**

Oats (A)

Apple pomace (B)

Sugar beet pulp (molassed, dried,
pressed, ensiled) (B)

Potatoes (A)

Maize grain (B)

Carrots (A)

Citrus pulp (B)

Molasses (A)

Manioc (B)

* Degradability: category A = 0.71–0.90, B = 0.51–0.70, C = 0.31–0.50, D = <0.31.

shown in Table 9.19. Some forage feeds, which would at

Energy and protein in feeds

first sight be considered cellulosic, are not. High-quality grass, for example, typically contains less than half of

The two most important nutrients are energy and protein. The DM as cell wall or neutral detergent fibre (NDF) is low. Other nutrients, particularly minerals, can limit efficiency of feed use, but in practice if a wide range of feeds is included in the diet the risk of mineral imbalance is low. Most farmers add proprietary mineral supplements to the diets of their cattle. However, particular deficiency situations do arise due to inadequate man-
Fodder beet contains 650 g sugar per kg DM, but it is contained within cell walls and is released in the rumen for fermentation at a slower rate than, say, the sugar from molasses.

46).

Nutrition • 113

150

Good grass

Good silage

100

Average silage

Poor silage

Average grass

Maize silage

Fodder beet

ERDP (g/kg DM)

50

Hay

Barley straw

Wheat straw

Fig. 9.16

Typical concentrations of ERDP

and FME in selected forage crops. Reproduced from Chamberlain & Wilkinson

0

4

5

6

7

8

9

10

11

12

(1996) with permission of Chalcombe

FME (MJ/kg DM)

Publications.

300

Rapeseed

Fishmeal

250

Soyabean

200

150

Maize gluten

ERDP (g/kg DM)

Brewers grains

Wheat

100

Barley

Palm kernel

Molasses

50

Fig. 9.17

Typical concentrations of ERDP

and FME in selected concentrates. Repro-

duced from Chamberlain & Wilkinson

0

6

8

10

12

14

(1996) with permission of Chalcombe

FME (MJ/kg DM)

Publications.

A simple classification of feeds is one which takes into ERDP relative to FME, whilst fishmeal, rapeseed meal, account the concentrations of ME, FME, ERDP and soyabean meal and maize gluten feed typically contain DUP (see sections above for definitions of terms), as in an excess of ERDP relative to their concentrations of

Table 9.20. A fuller description of the composition of FME.

feeds is given in the UK Ministry of Agriculture, Fisheries and Food's book *Feed Composition* (MAFF, 1992).

Physical form

An alternative way of considering feed sources is in terms of the balance between ERDP and FME, shown in Figs 9.16 and 9.17 for selected forages and concentrates, respectively. Maize silage, fodder beet, hay, straw, molasses and cereal grains are typically deficient in

There are three easily recognizable categories for the physical form of feeds: (i) liquids, (ii) moist solids and (iii) dry solids. Examples of each category are shown in Table 9.21.

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Table 9.21

Examples of the physical forms of raw material feeds. Reproduced from Stark & Lonsdale (1989) with permission of Chalcombe Publications.

Liquid

Moist solid

Dry solid

Condensed corn steep liquor

Apple pomace

Bran (wheat and rice)

Delactosed whey syrup

Brewers' grains

Cereal grains

Fresh whey

Brussels sprout

Citrus pulp

Molasses

packhouse waste

Distillers' dark grains

Pot ale syrup

Carrot rejects

Dried grass and lucerne

Maize gluten feed*

Legume seeds

Pectin-extracted fruit

Maize germ meal

Potatoes

Maize gluten feed*

Sugar beet pulp*

Maize gluten meal

Malt culms

Malt residual pellets

Oatfeed

Oilseed residues

Sugar beet pulp*

Wheatfeed

Whole oilseeds

* Available as either a moist or dry solid.

Liquids range from very low DM materials such as whey, which has handling characteristics similar to and water. Hence preservation may be achieved by water, to viscous liquids such as molasses, which has a typical DM content of 750 g/kg fresh weight. With the exception of whey, most liquid feeds have been condensed prior to shipment, in an attempt to reduce haulage costs.

Drying

Moist solids comprise those which contain fer-

Drying is the most effective form of feed preservation.

mentable sugars (e.g. fresh grass, molassed sugar beet

It is also the most costly and therefore tends to be used

pulp) and are usually stored as silage (see section on

with the more valuable feeds (e.g. cereals) and with

feed preservation, see below), and those which are low

feeds that are prone to deterioration (e.g. citrus pulp).

in fermentable components (e.g. apple pomace), and

The DM content of cereals is normally increased by

which benefit from the addition of a preservative.

drying to 850 to 870 g/kg prior to storage. Hay is dried

Dry solids are a common form of feed and include

in the field to about 800 g DM/kg fresh weight, unless it

cereal grains and by-products from the flour milling

is to be dried artificially in the barn, when it may be har-

industry, hay, straw and residues from oil seed extrac-

vested in a moist state at between 650 and 750 g DM/kg.

tion. The DM content of dry solids is usually in the

Drying in the barn proceeds until the safe DM content,

range 830 to 930 g/kg fresh weight. Higher moisture more than 800 g/kg, has been reached and the crop contents increase the risk of spoilage during storage shows little sign of heating and moulding.

and for this reason most manufacturers of dry feeds aim to approach 900 g DM/kg fresh weight if possible. Hay is usually stored at an initial DM content of around

Ensiling

800 g/kg, but final DM content is typically 850 g/kg fresh weight.

The process of ensilage involves the fermentation of plant water-soluble carbohydrate (WSC) monomers (simple sugars, mainly fructose and glucose) to organic

Feed preservation

acids, principally lactic acid. The acidity thus produced effectively 'pickles' the crop or feed in a stable state in

The principle of preservation is to prevent the development of spoilage organisms such as the putrefying

Fermentation is an anaerobic process. The crop must bacteria and moulds. These organisms prefer warm

be completely sealed from the air to facilitate the growth

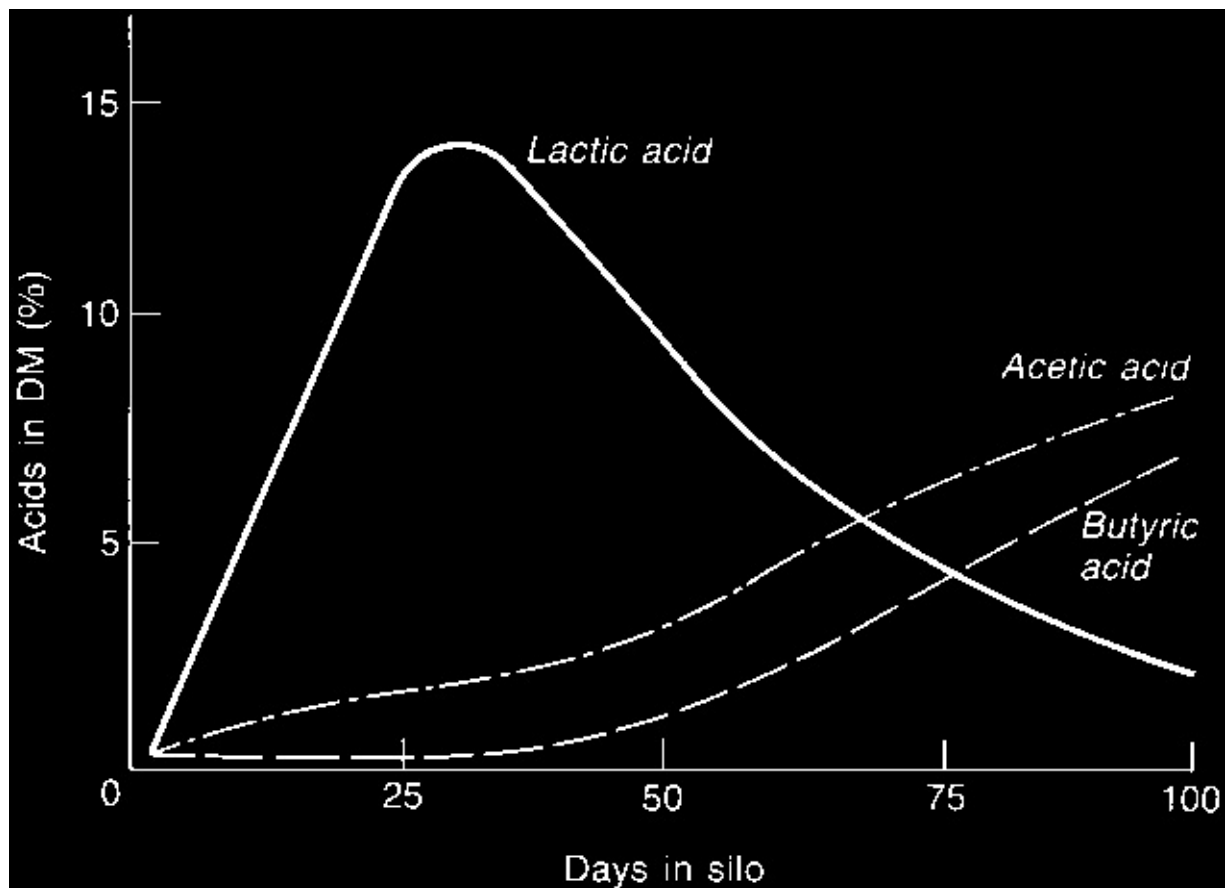
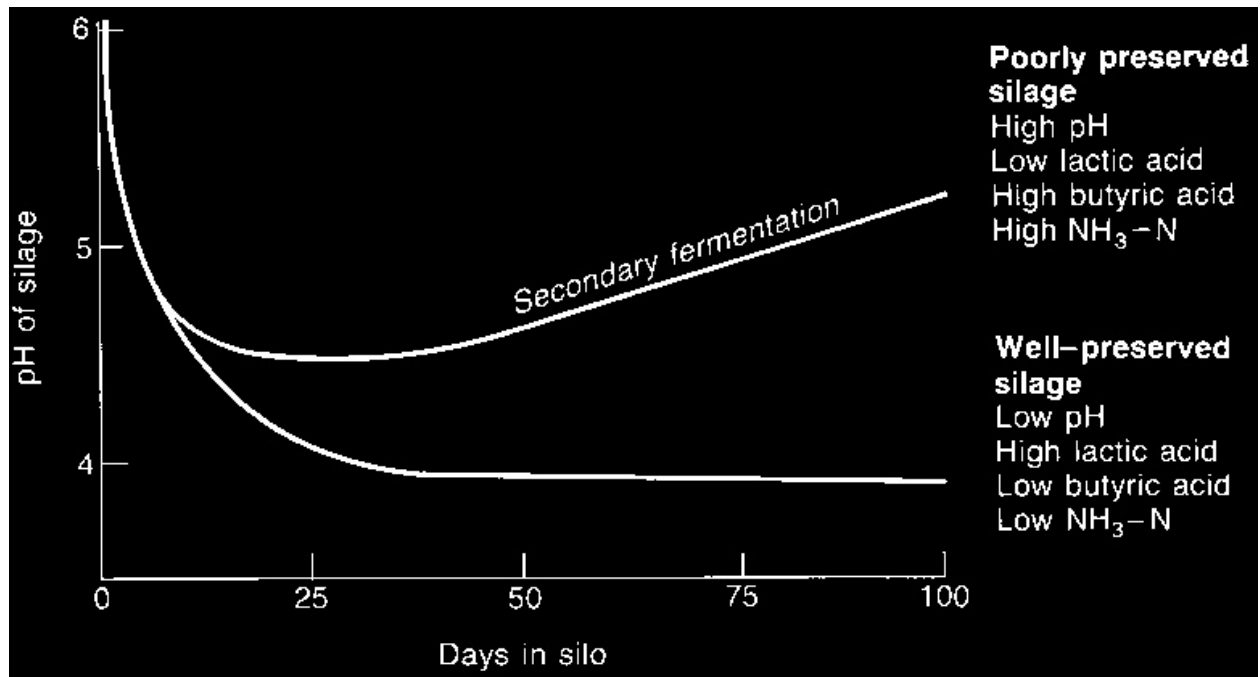


Fig. 9.18

Patterns of fermentation and changes in the pH value of silage as a result of secondary fermentation.

of the desirable anaerobic bacteria that are present on the crop in the field in relatively small numbers. As much air as possible should be removed from the crop at the time of ensiling, so that residual oxygen is exhausted as rapidly as possible. This is achieved by first chopping the crop as it is harvested from the field, by consolidating it once the crop is in the silo and finally by sealing it completely using a plastic sheet.

Primary and secondary fermentations

It is important to distinguish between primary and secondary fermentations, and to recognize that both are quite distinct from the process of aerobic spoilage that

Fig. 9.19

Typical changes in the content of fermentation acids occurs on exposure of silage to the air at the time of in silage as a result of secondary fermentation.

feed-out.

Primary fermentation essentially comprises the conversion of sugars or WSC, mainly fructose and glucose, to lactic and other acids as the result of the metabolism of bacteria. This process can be rapid and completed in a short period of time. Acidity increases as the conversion of a few days.

sugars to acids continues.

Secondary fermentation, which sometimes follows Primary fermentation proceeds until either the supply of fermentable substrate is exhausted or the lactic and other acids, with the formation of volatile amount of free water (i.e. that not associated with smelling acids like butyric acid. The process can also be products of fermentation) is reduced to a sufficiently low level to restrict bacterial activity. A stable low pH is reached. The silage contains lactic acid as the main product of fermentation. The main organisms involved in secondary fermentation are the obligate

fermentation acid and the amount of protein completely degraded to ammonia is small (see Fig. 9.18). anaerobic clostridial bacteria.

At the point of entry to the silo the pH of the fresh

However, if the supply of sugar in the crop is low,

crop is usually about 6.0. The crop is still alive, but it is

or its resistance to acidification or buffering capacity is

consuming the products of photosynthesis (sugars) by

relatively high, secondary fermentation may occur

respiration and producing carbon dioxide, water and

during the storage period. In this situation the silage

heat. In addition, aerobic bacteria such as the Enter-

is unstable. Wet crops of low sugar content are par-

obacteriaceae (coliforms) consume sugars to produce

ticularly prone to secondary fermentation. Typical

acetic acid and degrade protein to ammonia.

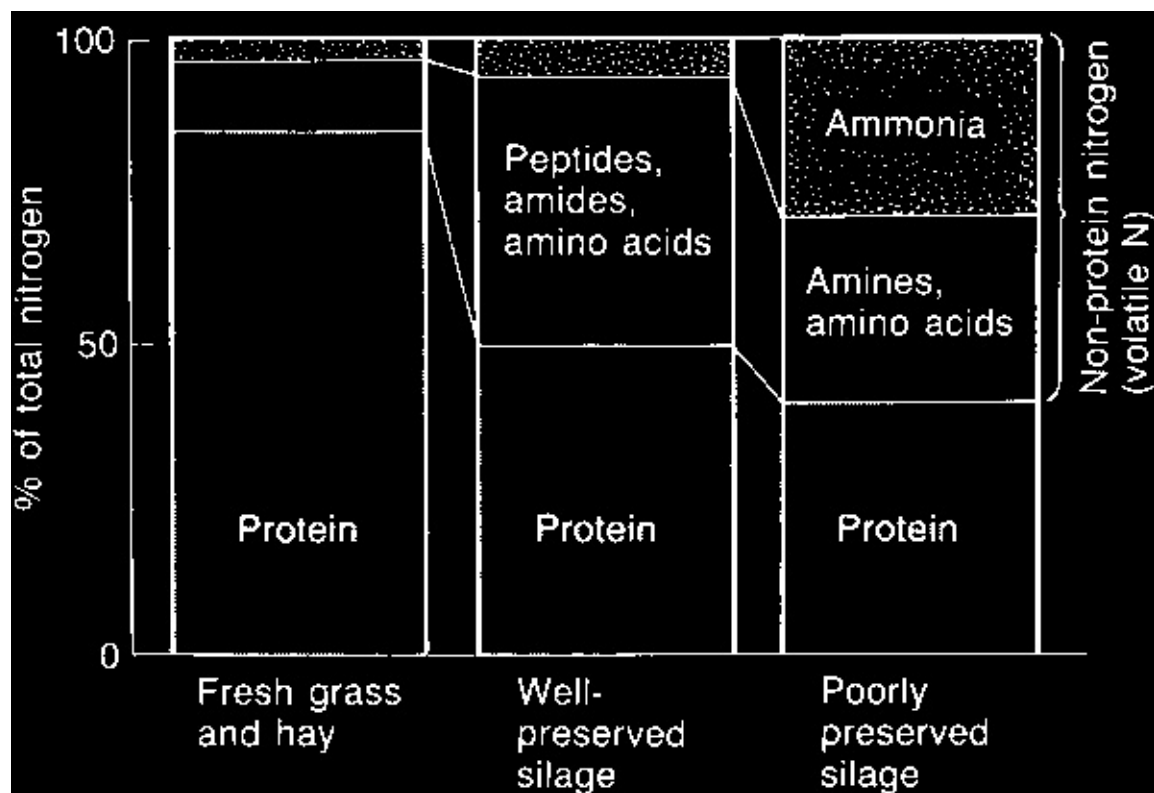
changes in the content of fermentation acids in silage

As the supply of oxygen is exhausted, the primary

as a result of secondary fermentation are shown in

fermentation dominates, with lactic acid the predomi-

Fig. 9.19.



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Table 9.22

Crops ranked in order of their 'ensilability'. Repro-

Typical analytical composition of silage

duced from Wilkinson (1990) with permission of Chalcombe Publications.

Typical values for the composition of grass and maize silages made in bunker silos under European conditions

Good

Maize

are shown in Table 9.23. The most notable differences

Whole crop cereals

between grass and maize silage are in crude protein and

Italian and hybrid ryegrass

NDF, which are higher for grass than maize, and in

Perennial ryegrass

starch which is very low in grass silage. Poorly-

Brome grass

preserved grass silages normally contain elevated levels

Timothy/meadow fescue

of ash, fibre, acetic acid, butyric acid and ammonia-N,

Permanent pasture

whilst energy values are depressed compared to well-

Cocksfoot

preserved material. Amino acid-N is reduced as a pro-

Arable silage containing legumes

portion of the total non-protein nitrogen, indicating

Grass/clover

Red clover

greater degradation to amines, amides and ammonia.

Poor

Lucerne

Wilted grass silages generally undergo less extensive fermentations and consequently have higher pH values than wetter silages, residual (i.e. unfermented) WSC, lower concentrations of fermentation acids and less ammonia-N (Table 9.23).

Additives for silage

The main objective in applying an additive at the time of harvest is to prevent the multiplication of clostridia. The lactic acid bacteria are more tolerant of acid conditions than are the clostridia, so traditionally their growth has been inhibited by direct acidification of the crop with an organic acid such as formic, or an inorganic acid such as sulphuric. The former is relatively more expensive, but it does have a specific antimicrobial action against clostridia, whilst sulphuric acid acts solely

Fig. 9.20

The nitrogenous components of fresh grass, well-preserved silage and poorly preserved silage. Source: Wilkinson (1985). through its effect in reducing pH. Some products comprise mixtures of acids and synergistic properties are

claimed for them.

If the pH of the crop can be reduced from 6.0 to

Some crops, because they have a relatively low about 4.5 by acidification at the time of harvest then the content of WSC or have relatively high buffering risk of clostridial growth is greatly reduced.

capacities, are more prone than others to secondary

An alternative approach is to accelerate the production of lactic acid by the direct inoculation of the crop fermentations – their ‘ensilability’ is relatively low.

Crops are ranked in order of their ensilability in Table

at harvest. Provided sufficient live bacteria are added

9.22. Maize combines adequate WSC with low buffering capacity, whilst the converse is true for the legumes, provided the content of fermentable sugar in the crop lucerne in particular.

is sufficient for their growth then good preservation

Wilting in good weather prior to harvest has the

quality should be assured. Problems can arise with effect of concentrating the sugars in the crop, and this

inoculants if there is insufficient sugar, either because is beneficial in terms of reducing the risk of secondary the crop itself is deficient or because the crop is too wet fermentation.

at the time of ensiling. The most common species of Silages that have undergone secondary fermentation bacteria in inoculants is *Lactobacillus plantarum*.

are poorly preserved, but paradoxically they tend to be Enzyme additives, containing hemicellulase and cel- relatively stable on exposure to air. A major conse- lulase, are another type of additive; the object in this quence of clostridial activity is an increase in the pro- case is to generate extra sugar from cell wall compo- portion of nitrogen present as ammonia in the silage nents to ensure sufficient acidification during primary (Fig. 9.20). Digestibility and energy value are also fermentation so that clostridia are inhibited.

reduced due to secondary fermentation; hence losses of Products are now available which contain both lactic nutrients are elevated in poorly preserved silages.

acid bacteria and enzymes. It is essential, however, to

Table 9.23

Typical composition of grass and maize silage stored in bunker silos.

Grass silage

Maize

silage

Direct-cut

Wilted

Poorly Well

preserved

preserved

pH

5.5

3.8

4.5

4.0

DM (g/kg fresh

160

180

360

300

weight)

Ash (g/kg DM)

110

80

90

55

Crude protein

160

160

150

90

(g/kg DM)

NDF (g/kg DM)

650

550

550

450

WSC (g/kg DM)

0

0

50

85

Starch (g/kg DM)

0

0

0

250

Lactic acid (g/kg DM)

50

150

65

100

Acetic acid (g/kg DM)

25

20

35

45

Butyric acid (g/kg DM)

35

0

0

0

DOMD (g/kg DM)

675

700

700

720

ME (MJ/kg DM)

10.8

11.2

11.2

11.5

NH₃-N (g/kg total N)

300

75

40

30

Amino acid-N (g/kg

405

700

750

650

total soluble N)

establish that the active ingredients in these biological
With fibrous feeds such as straw, intake is often
additives really are active, and have not been destroyed
limited by the speed with which long particles are
by processing, packaging or storage.

reduced in size by chewing so that they can pass out
of the rumen. Grinding removes this restriction to
intake, but a consequence is that particles pass out of

Additives for hay and moist grain

the rumen before they are fully digested. The net result
The risk of development of moulds and mycotoxins in
is that although dry matter intake is increased,
moist hay and moist grain may be reduced by adding
digestibility is reduced and nutrient intake is little
an effective preservative at the time of storage. Propi-
changed.

onic acid and salts of propionic acid such as ammonium
Chopping long forages also reduces the opportunity
bis-propanoate, added at 15 kg active ingredient per
for the animal to select the best quality material on
tonne of fresh crop at the time of harvest, are effective

offer. However, if straw is to be used in a complete diet, in reducing mould development in both hay and grain coarse chopping is a useful way of incorporating the and also in big bale silage, where moulding due to feed uniformly into the total mixture.

incomplete sealing is a common problem.

Long fibre with an average particle length of at least 150 mm is essential for milk fat synthesis and for the maintenance of rumen function on high-concentrate

Feed processing

diets. It is therefore important to bear in mind that physical processing of roughages can diminish their

Physical processing of roughages

value with respect to milk quality and the health status of the rumen.

Traditionally, feeds like hays and straws were chopped or ground prior to feeding. There is now increasing evidence to indicate that although these procedures

Chemical processing of roughages

may be convenient for the processor, they are less than ideal from the point of view of the nutrition of the

The use of alkalis, such as sodium hydroxide, for animal.

upgrading straw is not new, but recently additional

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benefits from the use of the technique have been

ing should be as small as possible: it is sufficient to crack recognized.

the seed coat. Crimping is better than rolling, but treat-

Essentially, the technique involves adding sodium

ment with sodium hydroxide, which has the effect of

hydroxide to straw at about 50 kg per tonne fresh

breaking the seed coat by swelling it, is probably the

weight. The alkali degrades and swells the plant cell

best method from the animal's point of view. In addi-

wall material, with a consequent increase in digestibil-

ity and intake. The benefits are valuable where straw is

in the grain, the residual alkali buffers acidity in the

plentiful and where it comprises a high proportion of

rumen.

the total diet, for example in the case of beef suckler

cows.

The residual alkalinity in straw treated with sodium

Feeding management

hydroxide can help to reduce the risk of ruminal acidosis in high-yielding dairy cows given diets containing

Successful nutrition of the bovine not only requires an large proportions of concentrates and/or highly acid understanding of the principles of nutrient requirement, nutrient supply and animal response; it also maintain milk fat content but the overall energy involves appropriate management of feed resources.

content is required to be high at the same time, the

This includes presenting the correct amount of feed to provision of a source of long fibre of enhanced the animal for the appropriate period of time, formu- digestibility is also valuable.

lating diets from available resources that meet require- Ammonia (usually added in aqueous form at 30 kg ments, taking into account the condition of the animal NH_3 per tonne fresh weight) is a useful alternative

and the desired direction and rate of change in weight
alkali to sodium hydroxide. It has the advantage of also
and condition and budgeting ahead so that rapid
contributing nitrogen, a valuable feature if the diet is
changes in diet are avoided and performance targets
deficient in rumen degradable nitrogen, but a disad-
are achieved.

vantage if silage of high protein content is the major
forage in the diet.

The importance of selective eating

Urea may also be used to upgrade straw, but both the
moisture content of the straw and ambient tempera-
The need to maintain stability in the rumen has led to
ture need to be relatively high for the technique to be
the presentation of feeds to the animal over a large pro-
successful.

portion of the day. This is particularly so with forages
The effect of an increase in digestibility of straw on
and straws, where rate of intake is relatively slow. It is
intake can be considerable. Thus a 10 per cent improve-
also important that the animal has access to feed for

ment in digestibility can lead to a 50 per cent increase most if not all the time when selectivity is desirable. in dry matter intake. Since the feed consumed is more At pasture, maximum intake is only achieved when digestible, the increase in ME intake is even greater. herbage is offered in substantial excess (Figs 9.7 to 9.9). In general, the amount of herbage on offer should be three to four times the amount eaten in order to achieve

Processing of cereals

maximum potential voluntary intake (see section on Grinding of cereals was traditionally considered essential to achieve complete digestion by cattle. A trade-off was accepted between rapid digestion in the rumen and grazed pasture, p. 101).

tial to achieve complete digestion by cattle. A trade-off was accepted between rapid digestion in the rumen and

Targets for sward surface height

possible acidosis on the one hand and poor digestibil-

at pasture

ity with undigested grains appearing in the faeces on the other. It is now accepted that the passage of a few undigested grains has no measurable effect on digestibility, A simple guide to the amount of herbage on offer is the

sward surface height of the grazed pasture. Regular but that forage digestibility can be reduced when grain measurement of sward height or sward mass is a useful is overprocessed.

tactical aid to pasture management. Targets for sward

The need for cereal processing depends on the size height for growing, lactating and dry cows are shown in of the reticulo-omasal orifice. In the case of sheep and Table 9.24.

calves less than 150 kg liveweight, it is difficult for whole

It is important to measure sward height in grazed barley and whole oat grains to pass through the orifice, areas, since herbage that is rejected due to contamination, whilst it is easy in larger cattle. Thus whole grains may be given to young calves, but some processing is necessary until the height of herbage in grazed areas is well below sward surface height. If possible, the extent of processing that at which intake is restricted below maximum.

Target sward surface heights (cm) for rotational and continuous grazing.

Spring

Early summer

Late summer

Autumn

Lactating cows and
growing cattle

Rotational grazing

Pregrazing

10–15

12–16

14–18

10–15

Postgrazing

6–7

7–8

8–9

6–7

Continuous grazing

6–7

7–8

8–9

6–7

Dry cows

4–5

4–5

4–5

4–5

a Postgrazing, rotational and continuous grazing.

Buffer feeding

Traditionally, dairy cows were given concentrates, usually in the form of milled compounded feeds, in the

The concept of buffer feeding was developed as a

parlour at milking and forages *ad libitum* for the rest of way of reducing losses in output from grazed pasture,

the day. With ever-increasing milk yields per cow, the

especially when herbage allowance was apparently

pressure on the available time in the parlour meant that

adequate but intake less than maximal due to poor

the cow had difficulty consuming a large feed of com-

weather, inadequate grass growth, short day length or

pounds during the milking period. Further, the rapid

reduced time of access to pasture.

fermentation of the two relatively large meals (some-

The buffer feed should be less acceptable than the

times as much as 5 kg/meal) meant that rumen pH

herbage on offer at pasture, otherwise the buffer acts as

decreased, fibre digestion slowed down and forage

a substitute rather than a supplement for grazed grass.

intake was reduced as a result (see Fig. 9.21). Accord-

Typically, silage or hay are used as buffer feeds, offered

ingly, a mid-day feed of compounds or of by-products

ad libitum at the farm buildings to dairy cows after each such as dried sugar beet pulp was introduced to reduce

milking or placed in the grazing field for other classes

the quantity of compound to be fed through the

of stock.

parlour.

Sodium bicarbonate may be a suitable ingredient of

Frequency of feeding and ruminal acidosis

the diet when the cow is suffering from acidosis.

However, the problem with bicarbonate is that it is

It is significant that when cereal concentrates are

relatively unpalatable and large quantities are required

offered *ad libitum* the animal eats a little at a time. Thus to have a significant effect. Thus the addition of 100 to

the general practice in feedlots, and in areas of the

150 g/cow per day is insignificant by comparison with

world where milk is produced from high-concentrate

the 3 kilogrammes of bicarbonate produced in saliva

diets, is to allow the animals 24-hour access to all feeds

daily. It is better therefore to include feeds, like hay or

(both concentrate and roughage) to avoid ruminal aci-

straw, to stimulate rumination and salivation.

dosis, otherwise known as 'feedlot bloat'. This condition

is caused by the rapid fermentation of starches and

sugars, and can occur as the result of infrequent inges-

Out-of-parlour feeders

tion of large feeds of concentrates. It can also occur as

the result of inadequate long fibre to maintain rumina-

The advent of computerized cow identification has

tion and saliva production. Essentially, the condition is

enabled cows to be rationed accurately in the parlour

due to the dominance in the rumen fermentation of

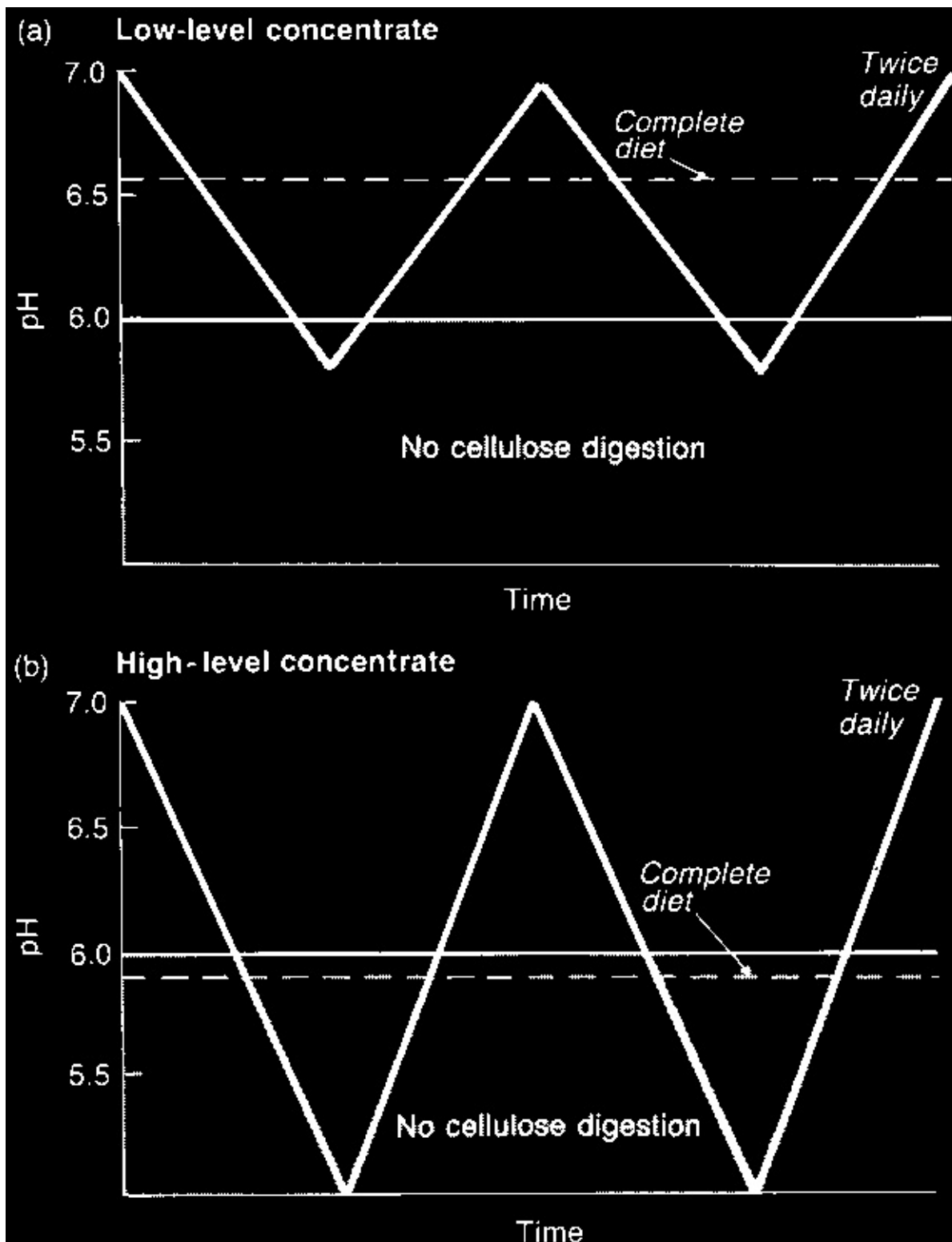
according to yield, and it was a relatively logical step to lactic acid producing bacteria (p. 829). Ruminant stasis introduce out-of-parlour feeders to allow the higher- can occur at low rumen pH (less than pH 5.5) and the yielding individuals to eat compounds and concentrates inability of the animal to eructate leads to the accumulation during the rest of the day. The nutritional advantages of gas and the resultant bloat (p. 832).

were that higher levels of compounds could be given

Cattle given concentrates *ad libitum* should therefore to those cows that 'deserved' to receive them, and in a

also be allowed access to long forage or a source of relatively large number of meals per day. Feeders could roughage such as straw, at about 15 per cent of the total be programmed to deliver equal proportions of the diet dry matter.

total day's allocation in up to 12 feeds per day; cows not



In practice, higher-yielding cows in the herd eat more

silage and it is important that the silage is on offer

ad libitum otherwise these animals are underfed. Peak lactation yields tend to be lower than in herds given

concentrates to yield, but the rate of decline in yield

post-peak tends also to be slower than in herds fed to

yield, giving a flatter lactation curve for flat-rate

feeding.

Total mixed rations or complete diets

The main principle of complete diet feeding is that the animal receives an intimate mix of slowly and rapidly fermented feeds, thus balancing the inflow to the rumen of feeds with widely differing rates of fermentation and (it is hoped) thereby maintaining stable conditions in the rumen.

However, with high proportions of concentrates in the mix, the level of acidity in the rumen may be so high that fibre digestion is permanently depressed (Fig.

9.21). In this situation, the proportion of propionate in the rumen volatile fatty acids is elevated and as a result cows tend to gain in weight and produce milk of relatively low fat content. Overfat cows with fatty livers

were a feature of early mismanagement of herds given

Fig. 9.21

It is important to prevent the pH of the rumen from
complete diets.

falling below 6.0 for long periods of time, otherwise cellulose Thus, provided
cows in late lactation and dry cows are

digestion will be greatly reduced. The problem is less at low

not given the same high-energy mix as higher-yielding

levels of concentrate feeding (a) than at high levels (b). Repro-animals, complete
diets offer a logical approach to

duced from Orskov (1998) with permission of Chalcombe

feeding management. By-products can be incorporated

Publications.

into complete diets with greater ease than in other

feeding systems. Higher voluntary feed intakes are

usually achieved than with separate feeding, and milk

taking their feeds were readily identifiable and could be

compositional quality is often improved as a result of

checked for disease immediately.

the higher energy status of the animal and improved

fibre digestion in the rumen. On the other hand, the

investment in mixer-wagons can be considerable and

Flat-rate feeding

there is little evidence of improved overall efficiency of
The introduction in Europe of milk quotas placed a
feed utilization by animals given total mixed rations
limit on output, and the emphasis switched to simplify-
compared to the same diet in separate feeds of forages
ing the day-to-day feeding management of the dairy
and concentrates.

herd, especially in those herds where the input of con-
centrate was moderate and where the supply of grass-

Diet formulation

land for grazing and silage was adequate and reliable
throughout the growing season.

Diets are formulated to meet calculated requirements

The principle of flat-rate feeding is that all cows
for major nutrients (see sections on energy and protein
receive the same amount of concentrate, irrespective of
requirements, pages 103, 106), taking account also of the
their actual or potential milk production, together with
needs of the animal for macro- and microelements.

silage *ad libitum*. The system works best when there is Recent research in the

USA indicates that formulations a compact calving pattern, with most cows at the same for lactation should also take account of dry matter stage of lactation, and when the input of concentrates content and the content of NDF (cell wall) in the total is relatively low. Silage quality should be relatively high diet. The dry matter content of the whole diet should and if necessary the level of concentrate should be be between 500 and 650 g/kg fresh weight for maximum reduced when the herd is in late lactation to prevent intake, whilst the content of NDF should be between 350 and 450 g/kg DM for maximal output of milk cows becoming overfat.

tween 350 and 450 g/kg DM for maximal output of milk

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Table 9.25

Target condition scores for beef suckler cows.

Table 9.27

Feed budget for suckler cows (t fresh weight/cow

Reproduced from Fuller (1988) with permission of Chalcombe and calf per annum). Silage and other feeds 25 per cent DM.

Publications.

Grazing

Stage of

Target condition score

Hill pastures and semi-arid lowlands: 0.5–1.0 cows/ha

reproductive

Upland pastures: 1.2–1.8 cows/ha

cycle

Autumn-calving Spring-calving

Lowland temperate pastures: 1.5–2.1 cows/ha

cows

cows

Winter feeding

Autumn calving

Spring calving

At calving

3

2.5

At mating

2.5

2

Lowland herds

Mid-pregnancy

2

3

Concentrates

0.4

0.2

Silage

3.5

2.5

Straw

1.0

0.8

Other feeds

0.3

0.4

Table 9.26

Feed budget for a dairy herd of 100 cows, average

(wet byproducts)

milk yield 6500 l/cow, calving September to November.

Upland herds

Concentrates

0.4

0.2

Feed

Budget

Silage

4.7

4.5

Straw

0.6

0.5

Grazing

Other feeds

0.2

0.4

Spring

5 cows per hectare

Hill herds: as for upland herds except silage 5.5 t /head to

Early summer

3.5 cows per hectare

allow for longer winter period

Late summer and autumn

2.5 cows per hectare

Silage

10 tonnes fresh weight per

cow at 10.5 MJ ME/kg DM

Table 9.28

Feed budgets for growing beef cattle.

Concentrates

When cows are housed

1.4 tonnes per cow

Grazing

When cows are grazing

0.1 tonnes per cow

Target liveweight (t /ha) at 250 kg N/ha

May

2.2

June

2.0

July

1.7

fat. Constraints in diet formulation programmes now

August

1.6

include dry matter and NDF in addition to conven-

September

1.4

tional prediction equations for voluntary intake and

October

1.4

nutrient requirements.

Average

1.7

Increase by 4 kg for each extra kg of N

Condition score

Silage and concentrates (t /head)

Silage

Concentrates

Condition scoring cows is relatively easy; there is no

(25% DM, 10.5 ME)

fleece to hide the animal's fatness (or thinness). The

Overwintered

essential areas for physical examination are: (i) the

store cattle

transverse processes of the lumbar vertebrae at the top
and finishing

of the loin, halfway between the last rib, (ii) the hip
suckled calves

3

0.3

bone and (iii) the tail head (see Chapter 2, Fig. 2.1).

18-month beef

5

0.9

Scoring should ideally be carried out by feeling the

Silage beef

6

0.8

(15–16 months)

extent of subcutaneous fat cover at the tail head and at

Cereal beef

*

1.6

the lumbar area. With practice, visual scoring is possi-

(11–12 months)

ble taking into account the visibility of the ribs and the amount of fat at the tail head. Cows in condition score * 0.2 t straw/head to maintain rumen function.

1 have no fat at the tail head and their ribs are prominent. The ribs are visible in condition score 2 but not in condition score 3 and above. As condition score rises Regular condition scoring helps to identify overthin above 2, the amount of fat visible at the tail head cows that require preferential treatment (extra feed) increases.

and overfat cows, especially those in late pregnancy, Target condition scores for cows at different stages of that are at risk of dystokia at calving. Scoring in early the production cycle are shown in Table 9.25.

lactation to monitor the effectiveness of nutrient inputs

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to achieve weight gain at mating is an essential part of Gibb, M.J. & Treacher, T.T. (1978) The effect of herbage successful rebreeding. Economies may also be made in allowance on herbage intake and performance of ewes high-energy feeds at times when the whole herd is

and their twin lambs grazing perennial ryegrass. *Journal of* scored as being slightly overfat.

Agricultural Science, Cambridge, **90**, 139–47.

Hodgson, J. (1975) The influence of grazing pressure and stocking rate on herbage intake and performance. In

Feed budgeting

Pasture Utilisation by the Grazing Animal (ed. by J.

Hodgson & D.K. Jackson), Occasional Symposium No. 8,

Feed budgeting is the extension of daily diet formula-

British Grassland Society, pp. 93–103.

tion to annual feed planning. Future levels of output are

Lonsdale, C.R. (1989) *Straights: Raw Materials for Animal* set and a feeding policy is developed which takes into

Feed Compounders and Farmers. Chalcombe Publications,

account the home-produced feeds available, or likely to

Lincoln, pp. 1–87.

become available during the year, and the type and

Ministry of Agriculture, Fisheries and Food (1975) *Energy*

quantity of purchased feeds required to meet the target

Allowances and Feeding Systems for Ruminants. Technical level of output. A buying strategy is developed to meet

Bulletin 33. HMSO, London, pp. 1–79.

the projected requirements.

Ministry of Agriculture, Fisheries and Food (1984) *Energy*

Simplified examples of feed budgets for grazing and

Allowances and Feeding Systems for Ruminants. Technical Bulletin 433. HMSO, London, pp. 1–85.

winter feeding are shown in Tables 9.26 to 9.28 for dairy

Ministry of Agriculture, Fisheries and Food (1986a) *Nutrient* cows, suckler cows and growing beef cattle, respectively.

Allowances for Cattle and Sheep. P2087. HMSO, London, pp. 1–6.

Ministry of Agriculture, Fisheries and Food (1986b) *Feed*

References and further reading

Composition. Chalcombe Publications, Marlow Bottom, Bucks, pp. 1–69.

Agricultural Research Council (1980) *The Nutrient Require-*

Ministry of Agriculture, Fisheries and Food (1992) *Feed Comments of Ruminant Livestock*. Commonwealth Agricul-

position, UK Tables of Feed Composition and Nutritive Value tural Bureaux, Farnham Royal, Bucks, CABI Publishing,

for Ruminants, 2nd edn. Chalcombe Publications, Canter-Wallingford, pp. 1–347.

bury, Kent, pp. 1–99.

Agricultural Research Council (1984) *The Nutrient Require-*

Orskov, E.R. (1998) *The Feeding of Ruminants: Principles and ments of Ruminant Livestock, Supplement No. 1*. Common-Practice, 2nd edn. Chalcombe Publications, Lincoln, pp.

wealth Agricultural Bureaux, Farnham Royal, Bucks, pp.

1–90.

1–45.

Osbourn, D.F. (1967) The intake of conserved forages.

Agricultural and Food Research Council (1992) Technical

In *Fodder Conservation* (ed. by R.J. Wilkins), Occa-

Committee on Responses to Nutrients, Report No. 9, Nutri-

sional Symposium No. 3, British Grassland Society, pp.

tive Requirements of Ruminant Animals: Protein. *Nutrition*

20–28.

Abstracts and Reviews, Series B: Livestock Feeds and

Stark, B.A. and Lonsdale, C.R. (1989) Straights for com-

Feeding, **62**, 787 .

pounders and farmers In: *Ruminant Feed Evaluation and*

Allen D.M. (2001) *Rationing Beef Cattle*, 2nd end. Chalcombe *Utilisation* (ed. by B.A. Stark, J.M. Wilkinson & D.I.

Publications, Lincoln, pp. 1–89.

Givens), Chalcombe Publications, Lincoln.

Chamberlain, A.T. & Wilkinson, J.M. (1996) *Feeding the Dairy* Van Soest, P.J.

(1994) *Nutritional Ecology of the Ruminant*, 2nd Cow. Chalcombe Publications,

Lincoln, pp. 1–241.

edn. Cornell University Press, New York, pp. 1–476.

Forbes, J.M. (1983) *The Voluntary Food Intake of Farm*

Wilkinson, J.M. (1985) *Beef Production from Silage and Other Animals*. CABI Publishing, Wallingford, pp. 1–206.

Conserved Forages. Longman, London, pp. 1–140.

Fuller, R. (1998) *Suckled Calf Production*, 2nd edn. Chal-Wilkinson, J.M. (1990) *Silage UK*, 6th edn. Chalcombe

combe Publications, Lincoln, pp. 1–105.

Publications, Lincoln, pp. 1–185.

Chapter 10

Alternative Forages

M. Tame and the late P.S. Jarvis

Introduction

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minerals for maintenance and production for lactating

Forage maize (corn)

123

and fattening animals. Knowledge is incomplete, and

Fodder beet

124

the art of feeding stock relates to adjusting feeds, intro-

Swedes

125

ducing different ingredients and measuring and observ-

Stubble turnips (leaves and roots)

125

ing the results.

Kale

125

The figures shown in Table 10.1 act as a starting point

Forage rape

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in the compilation of dairy cow rations, particularly as

Lucerne

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Red clover

127

feeds can vary in analysis. Alternative forages will need

White clover

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to fit the criteria laid down for each animal's produc-

Peas

129

tion. However some forages, such as maize and lucerne,

Sainfoin

129

are DMI enhancers and so they can be perfectly satis-

Lupins

129

factory at lower energy densities in winter rations.

Rye

129

Dairy heifers, calving at two years old, need to grow

Triticale

130

at 0.7–0.8 kg/head daily to attain a satisfactory calving

Whole crop silage: whole wheat

130

weight, and beef animals in the final stages of fattening

Whole crop cereal with peas

130

require growth rates in excess of 1 kg/head daily.

Wet byproducts used in dairy cow rations

Total farm profitability determines whether or not alternative forages feature in a farming system. Factors to be considered include soil type, labour availability,

Introduction

working and fixed capital requirements, value for money, yield variability, rotational considerations and The main forage crops in the UK and many other feeding facilities. The majority feature in winter rations, countries for cattle are conserved grass in the form of but kale and stubble turnips are often grazed in the hay or silage, otherwise straw. Economic and nutritional summer and early autumn, while lucerne and red clover considerations have led to a quest for alternative are versatile protein sources in the summer. They can forages.

be grazed (with caution because of bloat) or conserved Feeding dairy cows, dairy heifers, suckler cows and as silage. Red clover is usually in a grass ley mixture, beef animals is a mixture of science and art. Any ration but lucerne is generally grown as a pure stand.

needs to be balanced for energy, protein, minerals,
A brief description will be given of some of the crops
vitamins and water, to meet the required production
available together with their use and integration into
objectives. Energy sources will include starches, sugars
cattle rations.

and digestible fibre, whilst protein will be of rumen
degradable and digestible undegradable protein and

Forage maize (corn)

will contain a wide range of amino acids. The ration
must be palatable, digestible, appetizing and free from
The typical analysis of forage maize silage per kg dry
dirt, stones, harmful bacteria and moulds. Some feeds
matter (DM) is:

are enhancers of dry matter intake (DMI), whilst others
substitute for existing ingredients in the ration. Thus
DM = 28 per cent, metabolizable energy (ME) = 10.8 MJ,
brewers' grains, sodium hydroxide-treated straw, maize,
digestible crude protein (DCP) = 70 g, neutral deter-
etc., are enhancers of DMI whereas, for example, barley
gent fibre (NDF) = 360 g, modified acid detergent fibre

and wheat substitute for each other.

(MADF) = 215 g

The UK Ministry of Agriculture, Fisheries and Food

Minerals: Ca = 3.9 g, P = 1.8 g, Na = 0.2 g, Mg = 2.4 g

and the Agricultural and Food Research Council have

Yield on National Institute of Agricultural Botany

published standards for energy, protein and the major

(NIAB) plots = 12.0 t DM/ha

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Table 10.1

Winter ration criteria for Friesian/Holstein dairy

maize gluten, 2.5 kg rapeseed meal, 1.5 kg soya bean

cows.

meal and 0.3 kg mineral/vitamin supplement. As the

DM of maize silage increases, so does the DMI.

Milk yield (kg/day)

Maize silage can be introduced to young cattle at

under three months old, but additional protein, vita-

35

25

18

Dry

mins and minerals are necessary. Typically, a 300 kg
heifer growing at 0.65 kg/day requires 5.1 kg DM of

Minimum DM (%)

38

33

26

22

maize silage, 0.2 kg 50 per cent vegetable protein and

Energy density

12.0

11.4

10.8

9.5

Minimum MADF fibre (%)

15

18

22

25

one per cent non-protein nitrogen (urea) added to

Approximate DMI

20.9

17.1

14.7

9.3

maize silage; 1 kg of a 34 per cent protein/mineral/

vitamin supplement would also be suitable.

Note: protein guidelines are 400 g DCP for maintenance and 60–65 g Bull beef animals have higher intakes of maize than

DCP/kg milk, with sufficient undegradable as well as degradable grass silage. A beast weighing 300 kg needs 6 kg DM of

protein in the ration.

maize silage plus 1.5 kg of a 34 per cent protein/mineral/

For Jersey cows with milk yields of 25, 18, 13 and 9 kg respectively, vitamin concentrate supplement to gain 1 kg/day. This

the protein requirements are 350 g DCP for maintenance and 70–75 g DCP/kg of milk.

quantity of supplement is kept constant over a range of

weights so that the crude protein of the complete ration

declines as the animal matures.

Maize silage is best stored in long, narrow clamps to

Maize fits well into a grass silage system and can be

limit the amount of heating and hence spoilage when

self-fed or forage-box fed. It can be fed as a green crop the clamp is opened. Most maize is grown in the south prior to harvest, and used as a buffer feed in spring for ern counties of Britain but as more early-maturing cows at pasture.

varieties are being bred, the frontiers are being pushed further north.

The true value of maize (corn) is often underesti-

Fodder beet

mated by conventional equations. Maize silage costs less to produce than grass silage, and is complementary

The typical analysis of fodder beet per kg DM is:

to it. In a dull, wet summer, the ME can be under 10

DM = 18.3 per cent, ME = 11.9 MJ, DCP = 34 g,

but is still as good as most grass silages made in similar

NDF = 127 g, MADF = 83 g

weather. Generally, it is a high-energy, low-protein con-

Minerals: Ca = 3.9 g, P = 1.8 g, Na = 2.4 g, Mg = 1.4 g

served forage that has limited fibre and is low in min-

Yield on NIAB plots = 14.2 t DM/ha (excluding the

erals, particularly calcium, phosphorus, sodium, copper,

value of fodder beet tops (leaves), which can add up zinc, iodine and manganese. The greater the proportion to 30 per cent to the DM) of maize in the ration, the more deficiencies need to be corrected.

Ensiled tops are higher in protein minerals (especially phosphorus) and fibre and can complement the diet, but are difficult to harvest and utilize efficiently. A typical ration for a dairy cow yielding 25 kg milk might include 32 kg grass silage, 14 kg maize silage, 1 kg soya bean meal, 5.5 kg maize gluten and 0.15 kg high calcium problems but the problem is reduced with Phipps (pers. comm.), has shown that by including at least 25 per cent of the silage as maize increases freshly calved cows.

forage DMI, lowers the cost and increases milk production and hence the weight of fat and protein. The Fodder beet is used primarily as a low-cost concen-

trate replacer for dairy cows, at a maximum of 4–4.5 kg response is greater with 63 per cent digestibility ('D' DM (22–25 kg fresh weight). As a high-energy source, it value) silage compared with 68 D grass silage. With a will need to be balanced by additional degradable and ration based on 40 : 60 grass : maize silage and 6 kg con- undegradable protein. In limited cases, it can be used as concentrates, DMI and milk yield were increased by 2.8 and part of the maintenance ration but additional protein, 2.9 kg respectively compared with grass silage and 6 kg fibre (as hay or straw) and minerals will be required. concentrates. Maize silage combines well with lucerne For a dairy cow yielding 25 kg milk, with 40 kg grass silage, but needs an addition of 1–2 kg straw to provide silage producing more than maintenance, 15 kg of fibre.

fodder beet, 4 kg maize gluten and 2.25 kg of a propri- Maize silage can be used as the sole source of forage etary 20 per cent protein, high-energy concentrate for dairy cows. An example ration for a cow producing complete the ration. A mix of other dry concentrates

25 kg milk would be 27 kg maize silage, 3.5 kg caustic
such as barley and soya bean meal can replace maize
treated straw, 2 kg molasses, 2 kg sugar beet pulp, 3 kg
gluten.

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Harvested fodder beet can be fed from a hopper or

Swedes

behind an electric fence provided that sufficient feed
space is available to avoid uneven intakes between

The typical analysis of swedes per kg DM is:

cows in the herd. It is more commonly fed as part of
a complete diet in a forage wagon, but the beet must

DM = 10.5 per cent, ME = 13.1 MJ, DCP = 64 g,

be free from dirt and preferably finely chopped or

NDF = 140 g, MADF = 114 g

shredded to integrate in the ration.

Minerals: Ca = 3.5 g, P = 2.6 g, Na = 1.5 g, Mg = 1.1 g

Strip-grazing beet in the field produces a multitude

Yield on NIAB plots = 7 t DM/ha

of problems: poisoning from tops (even when topped

The crop is generally grown in the cooler west and

this can be via secondary growth in the spring), waste north of Britain. Fungal problems such as powdery of feed and foot troubles (see Chapter 31).

mildew and club-root can limit crop yields.

When used as a concentrate replacer, fodder beet Swedes have a low protein and high energy content, enhances the butterfat and protein percentages in the but a relatively low yield of DM. They are used milk, but observations suggest that it depletes the composition primarily for beef and sheep as well as for human positional quality when used as part of the maintenance consumption.

ration.

Swedes could be utilized by dairy cows on a similar

A combination of fodder beet and soya bean meal

DM basis to fodder beet, but are more costly to

can add to DMI and hence milk yield and quality, par-

produce. Despite their low DM (10.5 per cent) they are

ticularly replacing a 10.8 ME grass silage. The grass

likely to be DMI enhancers. Similarly, they can be fed

silage uptake has been shown to decrease from 9.6 to

to dairy heifers by incorporation in straw-based rations.

7.0 kg DM/cow per day. When fodder beet was added

The low DM of swedes could assist consumption, but

to a poorer silage and with no soyabean meal, compo-

chopping or slicing is advantageous. Swedes may be

sitional quality was not improved. As well as lowering

grazed *in situ*, especially by sheep.

the ration cost, fodder beet partially acts as a concen-

trate replacer.

Fodder beet should be finely chopped or shredded in

Stubble turnips (leaves and roots)

the rations for dairy heifers over one year old when

they are changing their teeth. For these animals average

The typical analysis of stubble turnips per kg DM is:

grass silage up to 2 kg DM (11 kg fresh weight) of fodder

DM = 8.0 per cent, ME = 11.6 MJ, DCP = 130 g

beet can be fed in place of sugar beet pulp to ensure

Figures for fibre levels and minerals are not available

good growth rates and conception. Using a straw-based

Yield on NIAB plots = 3.8 DM/ha (relates to the tops

ration, a 300 kg heifer growing at 0.8 kg/day could

and that portion of the root that is above ground)
receive 3 kg straw, 14 kg fodder beet, 1.2 kg barley and
1 kg soya bean meal. If ammonia or urea-treated straw
These quick-growing turnips are a very useful catch
is used, 11 kg fodder beet and 1 kg barley could produce
crop being sown in the spring as a prelude to a grass
the desired results. Alternatively, 2 kg DM from both
reseed in the autumn or after winter barley in July as a
fodder beet and brewers' grains with *ad libitum* straw break crop.
should produce a palatable and productive ration.

Stubble turnips are grazed by dairy cows *in situ*

Amounts will, of course, be increased as the animals
behind an electric fence in midsummer and autumn,
grow.

particularly where grass production is limited. In the
Gleadthorpe Experimental Husbandry Farm (1984,
summer, stubble turnips and *ad libitum* ammonia-
1985) developed a fodder beet system that utilised its
treated straw complement grass, while in the early
low cost when compared with concentrates for Friesian
autumn the same combination can substitute for a

¥ Hereford heifers and steers. Crushed beet was introduced when the animals weighed 200 kg. Both fishmeal and soyabean meal have been used as the protein sources with minerals and straw also added if necessary. because they are unlikely to be constrained by an electric fence. In many cases, fodder beet requires much additional capital for sowing, harvesting, storing, cleaning and chopped the beet, but it can be grown in most areas of and alternaria leaf spot can limit crop yields.

the UK and other temperate areas. In a wet autumn on heavy soils, harvesting can be difficult, but it is a relatively cheap source of energy. Mangels, which have a

Kale

DM of 10–15 per cent, are in the main similar to fodder beet for feeding but crop yield is less.

The typical analysis for kale per kg DM is:

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DM = 13.6 per cent, ME = 12.6 MJ, DCP = 119 g,

DM = 29 per cent, ME = 9.5 MJ, DCP = 134 g,

NDF = 208 g, MADF = 161 g

NDF = 464 g, MADF = 357 g

Minerals: Ca = 12.1 g, P = 3.5 g, Na = 1.2 g, Mg = 1.4 g

Minerals: it is claimed to be high in minerals, except

(varieties differ in analysis)

sodium, and to have good buffering capacity in the

Yield on NIAB plots = 8.6 t DM/ha

rumen. Ca = 17.6 g, P = 3.0 g, Mg = 1.8 g

Yield on NIAB plots = 14.7 t DM /ha

Kale can be grown in most areas of the UK but yields

can be adversely affected by dry weather.

Lucerne (alfalfa) is now the most important forage

Protein and minerals are contained in the leaves and

crop on a worldwide basis. While some 1 million ha are

carbohydrates and fibre occur in the stem. Thus, the

suitable for the crop in the UK, only around 22 000 ha

proportion of leaf to stem influences the feed value of

are cultivated. The crop grows best in areas where the

kale.

night temperature remains above 10°C and where

Kale is a low DM, high-protein feed that is high in daytime temperature is in the range 15–25°C. It is an calcium, but low in phosphorus, copper, manganese and erect, deep-rooting and hence very drought tolerant iodine. Goitrogens can interfere with iodine use in the crop which is well suited to cutting. The exceptionally dairy cow (see pp. 257, 586). Excessive amounts of kale deep rooting habit requires a well-fissured alkaline can lead to haemolytic anaemia (see p. 941).

subsoil. The soils also need to be free draining as the

In the past, kale was strip-grazed in the autumn and plant is not tolerant of a high water table. Top soil pH up to midwinter and, together with hay, provided the should be maintained at 6.2 or higher, preferably above maintenance portion of the dairy cows' ration. The 6.5 as root nodulation does not occur at pH levels practice has been largely curtailed as herds moved below 6.2. Lucerne is not a competitive crop (Sheldrick towards autumn calving, and a continuity of feed during

et al. , 1995).

the winter season became necessary. In addition, foot

The advantages of lucerne are its reduced fertilizer

troubles were very common and udders needed addi-

requirement (it fixes very high levels of nitrogen); its

tional washing. Currently, kale is grazed *in situ* in the persistence (it will stand regular cutting for up to 5

summer and early autumn in a similar fashion to

years); its drought resistance; its high protein level (up

stubble turnips with ammonia-treated straw. It tends to

to 24% protein at tight bud stage); its high digestibility;

be fed to mid- and late-lactation cows and should not

the slow fall in D value; its low level of lignification and

exceed 30 per cent of the ration. In practice, 3–4 kg

its high levels of calcium and vitamins A and E

DM/head per day are consumed together with 1–2 kg of

(Koivisto, 1999, pers. comm.).

ammonia-treated straw. Kale can be cut and carted to

Its disadvantages are that in the small seedling stage

all classes of stock, and may be strip-grazed by sheep in

it is frost susceptible. It is not tolerant of wet conditions

winter.

requiring a free draining soil. It is susceptible to stem eelworm and sitona weevil and some varieties are sus-

Forage rape

ceptible to verticillium wilt. In some conditions downy mildew and various leaf spots may occur.

The typical analysis of forage rape per kg DM is:

All seed should be checked to ensure that it has been fumigated against stem eelworm and all seed should be

DM = 14 per cent, ME = 9.5 MJ, DCP = 144 g

inoculated with *Rhizobium meliloti* in areas where it

(no fibre figures are available)

has not been grown in the last few years. A seed rate of

Minerals: Ca = 9.3 g, P = 4.2 g, Na = 2.3 g, Mg = 2.1 g

20–25 kg/ha is recommended, except in particularly

Yield on NIAB plots = 5.4 t DM/ha

favourable areas where the rate may be reduced to

Rape can be grown in most areas of the UK. Forage

15–20 kg/ha. The target seedling density should be 425

rape can be used as a catch crop like stubble turnips and

plants per square metre, although a mature stand may

kale but is lower yielding than the latter and higher only have 120–150 plants per square metre. The crop yielding than the former. The principles of feeding and should be sown between late April and the first week the reservations on use of forage rape are similar to in August. Lucerne should not be sown with grass as the kale. Forage rape can cause taints in milk and should latter is too competitive.

be introduced into the ration slowly. It can be grazed *in*

Another disadvantage is its low yield in the establish-
situ from July to December, or be zero-grazed.

ment year, although this can be overcome by undersow-
ing it into a spring cereal crop or a semi-leafless pea crop.

After establishing the nurse crop and controlling weeds

Lucerne

the lucerne should be broadcast and either rolled or
raked in. Alternatively the two crops can be drilled at the

The typical analysis of lucerne silage per kg DM is:

same time, although this makes weed control more diffi-

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cult. If there is adequate late summer/early autumn rain-

has spearheaded a revival of interest in the last five
fall the crop can be established in late summer, no later
years.

than mid-August, after a winter cereal crop.

Red clover yields well on a range of soils with a pH

Decaying lucerne roots exude chemicals known as
of 6 or higher and adequate soil moisture. Summer rain-
medicarpins which inhibit germination of lucerne so it
fall is also a prerequisite for good yields. Water-logged
is not possible to 'stitch in' seed to fill areas where the
soils, on the other hand, should be avoided and heavy
crop has died out.

soils subject to structural damage from smearing and
Fertilizer requirements are relatively low as there is
the use of heavy machinery will shorten the life of the
no requirement for nitrogen after establishment. The
crop. Unlike white clover, red clover does not produce
main requirements are for phosphate and particularly
stolons (runners) and has a single crown to the plant
potash.

that is susceptible to damage.

Lucerne is ideally suited to a cutting regime. The crop
As a general rule red clover is sown with Italian
should be allowed to flower before the first cut and
rye grass and with the more modern varieties a fairly
should then be cut at intervals of 35–40 days, with the
aggressive rye grass is required as the clover tends to
last cut being made in September to allow at least 6
take over in the second year. Seed rates are usually
weeks for recovery before the first frost. After the first
around 3 kg/ha with a target plant density of 200/m².
frosts the crop will normally stop growing, at which time
It is important that the red clover seed is fumigated
it is safe either to cut or lightly graze the crop. The ideal
against stem eelworm. Modern varieties will usually
cutting time is mid-bud stage.
give two heavy cuts, with a third lighter cut over the first
Dry matter yields from a well-established crop of
two years. There is a significant decline in productivity
lucerne under ideal conditions could be as high as 14
in year three, although the new variety Milvus is
tonnes dry matter/ha, although average yields will be

claimed to give good production over a three-year period. Growing red clover with Italian rye grass expected to contribute about 35 per cent of the total, will not only give a heavier crop, by up to 30 per cent annually, but also raise overall digestibility. 20 and 10 per cent, respectively.

Annual yields of dry matter are usually in the range 9–12 tonnes/ha for pure stands and 11–13 tonnes/ha when grown with companion grasses. Yields in organic systems will be almost, if not as high as in conventional systems because of the high levels of nitrogen fixed. Red clover can fix up to 245 kg nitrogen/ha (Lampkin, 1990).

Lucerne is not usually grazed because of the risk of bloat, although there are instances where it has been grazed successfully. This requires a slow introduction of lucerne to the grazing system and all the usual precautions to avoid tympany. Potash level can be improved by the appropriate use of fertilizers. Protein level can be improved by the appropriate use of slurries to which it responds well. If high protein levels are to be achieved in the first cut the use of some artificial nitrogen may be necessary as nitrogen fixation by lucerne is temperature dependent and in cold and wet seasons little nitrogen may be fixed before the first cut is made. The protein level in the first cut material is often very low in organic systems where the use of artificial nitrogen is not permitted. However, it has usually increased to around 18–20 per cent by the time the

Red clover

Red clover is sensitive to soil potash levels as large amounts are removed from the field under cutting. Potash level can be improved by the appropriate use of fertilizers and all the usual precautions to avoid tympany. Red clover responds well to slurries. If high protein levels are to be achieved in the first cut the use of some artificial nitrogen may be necessary as nitrogen fixation by red clover is temperature dependent and in cold and wet seasons little nitrogen may be fixed before the first cut is made. The protein level in the first cut material is often very low in organic systems where the use of artificial nitrogen is not permitted. However, it has usually increased to around 18–20 per cent by the time the

The typical analysis for red clover silage per kg DM is:
second cut is made.

The low levels of sugar in clover can lead to poor fer-

DM = 22 per cent, ME = 9.8 MJ, DCP = 135 g

mentation in ensiled crops of pure red clover. Ensiling

Figures are not available for the fibre levels and mineral

grass/red clover is often more successful because of the

content, but like lucerne, red clover has high intake

sugar contributed from the grass. The quality of the fer-

characteristics and the ME is likely to be close to

mentation is also likely to be improved by wilting the

10 MJ/kg DM

crop to approximately 30 per cent dry matter whenever

Yield on NIAB plots = 13.5 t DM for first harvest year,

possible, but care should be taken not to exceed this as

and 11.1 t/ha for the second harvest year

leaf loss due to shattering occurs above this level. If the

The use of red clover declined dramatically to the late

material has to be ensiled at a significantly lower dry

1990s. However, the recent interest in organic farming

matter the use of an inoculant is recommended.

Weed control can be a problem as red clover is

Production levels from grass/white clover swards are sensitive to many of the herbicides currently available.

lower than from grass/red clover swards and are likely

Control of weeds in organic systems is often achieved

to be of the order of 6 tonnes of dry matter/ha with no

by undersowing into a spring cereal nurse crop, a tech-

artificial nitrogen input to 7.6 tonnes/ha with 200 kg

nique which could also be used in conventional systems.

artificial nitrogen/ha. However, at this level the clover

A gap of at least four and preferably five years should

content of the second cut will be suppressed (Sheldrick

be allowed between red clover crops to reduce the risk

et al. , 1995). These same authors state that under ideal of pests and diseases, particularly stem eelworm and

conditions herbage yields equivalent to those from

Verticillium.

grass swards receiving in excess of 300 kg nitrogen/ha

The risk of bloat in cattle is reputedly higher with red

have been recorded. It is recommended that no more

clover swards than white clover swards and as a result than 50 kg of nitrogen/ha is used. In organic systems red clover is grazed less frequently (p. 833). However, where the use of artificial nitrogen is not permitted it with careful management it can be avoided. The red should be noted that the protein content of the first cut clover should be introduced slowly and once introduced is often very low, particularly in cold and wet seasons it should be a consistent component of the diet. The risk when it is typically between 10 and 12 per cent (Weller is highest in cold wet weather and when the animals are & Cooper, 2001; Tame, pers. obs.).

particularly hungry. Lambs in particular grow very well The proportion of grass to clover can be managed to on grass/red clover swards and increased growth rates some extent by alternating cutting with grazing. The can also be achieved with young stock and beef cattle growth of the white clover is light sensitive; allowing the (Thomas *et al.*, 1982).

sward to grow will mean that eventually the clover will Because of its ability to fix large amounts of nitrogen,

be shaded out as the grass grows taller. This is particularly so in autumn and early spring when the temperatures are low enough to suppress clover growth but not grass growth. The clover content can be increased by an intensive grazing and/or cutting regime, although the

proportion of clover increases naturally through the season (Weller & Cooper, 2001). Ensiling grass/white clover is less difficult than grass/red clover because of increased significantly and it is increasingly being used the generally lower clover content and higher sugar

White clover

proportion of clover increases naturally through the season (Weller & Cooper, 2001). Ensiling grass/white clover is less difficult than grass/red clover because of increased significantly and it is increasingly being used the generally lower clover content and higher sugar in grazing swards. Again, interest has been fuelled by level contributed by the grass, but even so wilting to the increasing interest in organic husbandry.

around 30 per cent dry matter is again recommended.

Varieties tend to be classified according to leaf size,

At dry matter levels much higher than this leaf shatter with the smaller leafed varieties being more suited to

will be a problem.

sheep grazing and the larger leafed varieties being

Weed control can be a problem in newly established

more suited to cattle grazing and/or conservation. More

swards, particularly with annual weeds such as chick-

recently, varieties have been introduced which are more

weed. This can be controlled by the careful use of an

cold tolerant, resulting in earlier growth and nitrogen

appropriate herbicide, although most will have some

fixation. Soil pH should again be 6 or higher for good

adverse effect on the clover. In organic systems suc-

germination and growth, and on some soils this may

cessful control is achieved by the use of mechanical

mean the regular application of lime to maintain this pH.

control or by undersowing into a nurse crop of spring

White clover is much longer-lived than red clover

cereals.

and will typically be expected to survive for six years

Grass/white clover swards are used principally for

or more under good management. Typical seed mix-

grazing and it is worth noting that milk production will

tures will contain between 1 and 2 kg/ha in a total
be 1–1.5 litres/cow higher from a grass/clover sward
mix of 12 kg/ha (the total seed rate may need to be
than from an all-grass sward.

higher in adverse conditions, e.g. low rainfall and
Care needs to be taken in the autumn to ensure that
light soils). White clover seed is very small and germi-
late grazing by sheep does not damage the stolons of
nates more successfully if broadcast on to the surface
the clover as such damage may result in a loss of clover
rather than being drilled into the seedbed. Grass vari-
in the following spring.

eties need to be chosen with care to avoid either the
White clover also plays an important part in soil
grass or the clover becoming the dominant species.

fertility building in organic systems because of its
Work in this area has been done by the Kingshay
ability to fix nitrogen even though it fixes less than red
Trust, from whom recommendations are available to its
clover, 150 kg/ha under good conditions (Lampkin,
members.

1990).

Alternative Forages • 129

Peas

effect of reducing the rate of protein breakdown in the rumen.

The typical analysis for pea silage per kg DM is:

Again sanfoin is well suited to organic systems

although, like lucerne, its inclusion in a rotation will

DM = 25.9 per cent, ME = 8.8 MJ, DCP = 146 g,

extend the length of the rotation considerably.

NDF = 517 g, MADF = 384 g

Minerals: Ca = 12.7 g, P = 4.4 g, Na = 0.4 g, Mg = 2.0 g

Yield (estimated) = 7 t DM/ha

Lupins

Pea silage can be a byproduct from the production of vining peas and may be more like straw. Peas can also

Interest in this crop has been growing in recent years

be grown on their own, but need some physical support

particularly as large areas are grown in continental

and are usually sown in conjunction with spring barley

Europe, 60 000 ha in northern Germany alone, and

on the basis of 40 : 60 peas : barley by seed weight. particularly in Australia, 1 million ha. While the crop Newman & Luffman (1984) have produced the arable is normally grown as a grain crop it can also be harvested as a forage crop. When cut at ten to twelve weeks a DM of 43.2 per cent, ME of 10.2 MJ and DCP of 147 g. They observed that the silage yield at 10 t DM/ha forage. Crude protein levels may be as high as 20 per cent, with a good energy level and up to 20–25 per cent when fed to 200 dairy cows in Somerset, it produced starch.

risers in DMI between 10 and 20 per cent over grass Recommended types are the white, apically dominant varieties or the yellow branched varieties. The range of 7–10 kg depending on the quality and quantity of the silage.) The arable silage mixture can also be

that the pH is within the range 5–7. The crop should be fed to dairy heifers and beef animals. Peas are not an easy crop to establish and whole crop cereal silage may depend on type and the seed should be inoculated. be preferred.

Weeds need to be controlled in the early stages of the crop. Yields as high as 10 tonnes of dry matter/ha have been reported, although it is too early to know whether

Sainfoin

this is typical under UK conditions.

Lupins seem to be particularly well suited to organic systems because of the high level of nitrogen fixation

A typical analysis for sainfoin silage per kg DM is:

and the fact that the crop is in the ground for such a

DM = 24 per cent, ME = 8.4 MJ, DCP = 124 g

short time means it fits well into rotations.

Figures for fibre and minerals are not available

Yield (estimated) = 7.5 t DM/ha

Sanfoin is a little grown crop although there has been

Rye

some revival of interest in the last few years. A research programme is currently under way at the Royal

The typical analysis for grazed rye per kg DM is:

Agricultural College at Cirencester (Lane & Koivisto,

DM = 23 per cent, ME = 9.5 MJ, DCP = 88 g

1999). In most aspects the requirements, establishment

Figures are not yet available for fibre and mineral

and management of sanfoin are very similar to those for
content

lucerne.

Yield (estimated) = 7.7 t DM/ha

The main differences are that the most commonly
available varieties of sanfoin are significantly less pro-

Rye is grown on its own or in a mixture with Italian

ductive than lucerne with fewer cuts. However, some

ryegrass to produce an early bite in the spring on light

recent varieties that will soon be available from

land with a low pH. It is usually strip-grazed by dairy

Eastern Europe, notably Hungary, may well be almost

cows once daily and its feed value often decreases

as productive as lucerne. Another difference which

rapidly. Zero-grazing is possible. Any surplus can be conserved as big bale silage and used for any class of stock. In some areas it is followed by forage maize and means that there is no risk of bloat from grazing it. Indeed, very high growth rates have been achieved in maize in the autumn. Many dairy farmers prefer buffer lambs grazing sanfoin (Koivisto, pers. comm.). It is also likely that the tannin content of sanfoin may have the feed as an alternative to growing small areas of rye.

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Triticale

35–40 per cent (Kristensen, 1995). Winter barley should be harvested slightly earlier at four to five weeks post ear emergence and at a dry matter of 30–35 per cent. which is a hybrid of wheat and rye. Triticale can perform However, in the UK the suggestion is that winter wheat

a similar function to rye, and is said to have lower DM can be harvested at up to 45 per cent dry matter (MDC, yields than rye in the earlier part of the season, but its 1995). Yields for winter wheat are 10–12 tonnes dry digestibility stays at a higher level than rye as the crop matter/ha with conventional systems and 6–10 tonnes matures. Feeding value is likely to be similar to whole in organic systems depending on where it is placed crop silage. If cut in mid June, it can have a DM of in the rotation and whether it is undersown with 25 per cent and an ME of 9.6 MJ. Both sugar and fibre grass/clover. If the dry matter is much higher than levels are higher than grass.

50 per cent it is likely that grain will be excreted, undigested, the amount increasing with higher dry matter.

Whole crop cereal is well suited to use as a buffer

Whole crop silage: whole wheat

feed to supplement grazing in the summer, particularly in organic systems where its starch and low protein

A typical analysis for whole wheat silage (Newman,

content can be used to balance the very high levels of
pers. comm.) per kg DM is:

degradable protein in the grass/clover swards from mid-
summer onwards. Indeed, whole crop cereal silages fit
DM = 50 per cent, ME = 10.7 MJ,

very well into organic systems as a whole as the early
crude protein = 9 per cent, NDF = 400 g, pH = 8

harvest allows a grass/clover ley to be established in
Yield (estimated range) = 7–10 t DM/ha

August. It also allows a degree of flexibility in the

There has been a considerable increase in interest in
system as it can be taken either for grain or for forage
whole crop cereal silage over the last decade both in
depending on the season.

fermented and in urea-treated form.

The addition of whole crop cereal silage to the diets
of both cattle and dairy cows results in a significant

Whole crop cereal with peas

increase in dry matter intake, particularly with the urea-
treated form. However, there are some very significant

The major disadvantage with whole crop cereal silages

differences between the two forms.

is that they are low in protein. This can be overcome to
The fermented form of whole crop has the advantage
a greater or lesser extent by growing whole crop cereals
that it has a much higher digestibility and results in a
as a bi-crop with peas. The target should be for at least
larger increase in milk output and a small increase in
40 per cent peas in the crop. The peas should be a short-
milk quality, mainly in butterfat. The disadvantage is
strawed, coloured, flowered variety. Sowing rate will
that it is lower in protein and is aerobically unstable,
depend on the proportion of peas required, but with a
although this latter disadvantage can be overcome to
50 : 50 mix the seed rates would be around 125 kg/ha
some extent by adequate chopping, good consolidation
wheat and 270 kg/ha peas depending on variety and
and good clamp management.
seed size.

Urea-treated whole crop has the advantages that

The crop should be harvested at 14–15 weeks post
there is a large increase in dry matter intake, it is aero-

sowing when the cereal grain is at the doughy stage and bically stable and the added urea results in a higher the pea seed is at the yellow wrinkled stage. The protein nitrogen content. However, the disadvantage is that the and starch are optimized at this stage (Adesogan, pers. digestibility, particularly of the starch, appears to be comm.). When harvested at this stage the silage should much lower and the large increase in dry matter intake have a crude protein content of 16–18 per cent, with is reflected in only a small increase in milk output and 40 per cent peas in the mix. If higher crude protein quality (Sutton *et al.* , 1997). As a result urea treatment levels are required the proportion of peas should be of whole crop cereal silage is not recommended. increased. In the same study digestibility is reported to The objective should be to grow the crop as for a be higher than grass silage when fed to sheep. A further good grain yield with winter wheat being sown at a rate trial showed that the total intake, milk yield and milk of 150–200 kg/ha and spring wheat at up to 240 kg/ha. If composition of cows fed whole crop/pea silage was

a spring crop is to be undersown the seed rate should be higher than or comparable with cows fed first-cut/ be roughly half the figure just given. There will of ryegrass silage and 4 kg more concentrate. As with fer- course be a yield penalty with the lower sowing rate. mented whole crop, the silage needs to be very well con- Winter wheat should be harvested at between five and solidated in the clamp and clamp management at six weeks after ear emergence and at a dry matter of feed-out must be very good.

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Table 10.2

Analysis of byproducts fed to dairy cattle.

Food

DM

ME

DCP

NDF

MADF

Ca

P

Na

Mg

(%)

(MJ)

(g)

(g)

(g)

(g)

(g)

(g)

(g)

Brewers' grains

22.0

11.7

189

572

219

3.3

4.1

0.1

1.5

Cabbage

10.6

13.7

181

586

140

7.2

4.5

1.2

1.5

Carrots

13.0

12.8

62

—a

—

5.9

3.4

0.8

1.8

Parsnips

15.0

13.3

67

—

—

—

—

—

—

Potatoes

20.4

13.3

80

73

39

0.4

2.0

0.2

1.0

Pressed sugar beet pulp

25.4

12.3

60

557

259

9.0

1.1

0.6

2.0

Apples

13.6

12.2

—

111

101

0.5

0.9

0.1

0.3

Apple pomace

23.0

8.7

—

503

375

1.7

1.4

0.2

0.6

a —, no reliable information available.

Yields under a conventional system should be

Kristensen, V.F. (1995) The production and feeding of whole

around 11–12 tonnes dry matter/ha, while under

crop cereals in Denmark. In *Whole-Crop Cereals* (ed. by organic management it should be 7–10 tonnes of dry

B.A. Stark & J.M. Wilkinson). Chalcombe Publications,

matter/ha.

Lincoln, pp. 21–38.

Lampkin, N. (1990) Crop nutrition. In *Organic Farming*.

Farming Press, p. 84.

Wet byproducts used in dairy

Milk Development Council (MDC) (1995) *Making the Most of Whole Crop Cereals*. MDC.

cow rations

Newman, G. & Luffman, B.J. (1984) Lucerne, red clover and

forage peas: management, utilization and incorporation into

The products shown in Table 10.2 can be incorporated

feed systems. In *Occasional Symposium of the British*

in winter rations for dairy cows and other stock. They

Grassland Society (ed. by D.S. Thompson), pp. 147–51.

are likely to be fed with straw and other ingredients for

Sheldrick, R.D., Newman, G. & Roberts, D.J. (1995) White

other cattle, and to supplement grass silage and act as

clover. In *Legumes for Milk and Meat*. Chalcombe Publications, Lincoln, p. 27.

a concentrate replacer for dairy cows.

Sutton, D.J., Abdalla, A.L., Phipps, R.H., Cammell, S.B. & Vegetable waste, e.g. Brussels sprouts, cabbages and

Humphries, D.J. (1997) The effect of replacement of grass

cauliflowers from vegetable processing plants, could

silage by increasing proportions of urea treated whole crop

have a similar feed value to kale. Potato waste, pressed

wheat on food intake and apparent digestibility of milk

sugar beet, peas and beans are occasionally available.

products by dairy lows. *Animal Science*, **65**, 343.

Good storage facilities and the avoidance of waste are

Thomas, C., Aston, K., Daley, S.R. & Hughes, P.M. (1982) A necessary for the above products. They will all need to

comparison of red clover with grass silage for milk produc-

fit in with the ration criteria for the appropriate class of

tion. *British Society of Animal Production Winter Meeting*, stock.

Harrogate.

Thomas, C., Aston, K. & Daley, S.R. (1985) Milk production from silage, 3. A comparison of red clover and grass silage.

Animal Production **41**, 23–31.

References

Weller, R.F. & Cooper, A. (2001) Seasonal changes in the

crude protein concentration of mixed swards of white

Gleadthorpe Experimental Husbandry Farm (1984) In

clover/perennial ryegrass grown without fertilizer N in an

Gleadthorpe Experimental Husbandry Farm Annual

organic farming system in the United Kingdom. *Grass and*

Review, pp. 15–17.

Forage Science, **56**, 92.

Gleadthorpe Experimental Husbandry Farm (1985) In

Wilkinson, J.M. & Stark, B.A. (1990) *Whole Crop Cereals* –

Gleadthorpe Experimental Husbandry Farm Annual

Making and Feeding Cereal Silage. Chalcombe Publications, *Review*, pp. 38–40.
pp. 1–86.

Part 2

DISEASE

Chapter 11

Diagnosis and Differential Diagnosis

in the Cow

R.G. Eddy and P.J.N. Pinsent

The clinical attitude

135

Thus normality is relative to environment. The vet-

The clinician's approach to the clinical case

136

erinarian, therefore, must consider the environment as

The clinical examination

136

critically as he does the patient. Is it suitable? Does it

The main presenting syndromes

138

predispose to, or even cause, the abnormality present

Drooling at the mouth (kiddling, slobbering)

138

in the patient? Does it cause stress? Remember that

Vomiting

139

the environment can only be appreciated if one con-

Acute abdominal pain (colic)

139

siders the animal's viewpoint: for example, if it is

Acute abdominal distension

140

Anterior abdominal/posterior thoracic pain

142

thought that the ventilation in a calf house is suspect, it

Chronic bloat (chronic and subacute ruminal tympany)

146

may well be advisable to get your head down among

The liver

147

the calves!

Constipation

148

If it is accepted that disease is often related to envi-

Diarrhoea

149

ronment, nutrition or management, then it follows that

Tenesmus (straining)

150

in an adverse environment other animals in the group

The unthrifty or emaciated cow

150

may also be affected. In such circumstances, it is always

The 'downer' cow

151

well worthwhile looking carefully at the rest of the

The cow ill during or after calving

151

group, if necessary, animal by animal. It may well be

Pyrexia of unknown origin (PUO)

151

that others are showing the same signs albeit to a lesser

Sudden death

152

extent. Such an observation may be very helpful not

Jugular stasis (cording)

153

‘Redwater’ (blood or blood pigments in urine)

154

only in diagnosis, but also from the viewpoints of treat-

Cows breathing badly (hyperpnoea and dyspnoea)

155

ment and prevention.

Acute nervous and convulsive syndromes

155

The veterinarians of today must concern themselves with the health of the herd as well as the individual and it is almost negligent to omit from the examination of the individual cow, a look, however brief, at the other

The clinical attitude

stock in the group.

The other side of the coin is that, in unusual envi-

Veterinary clinicians must develop certain special

ronmental conditions, when you are not certain

attributes in relation to their environment. They must

whether the cow's response is normal or not, it is often be inquisitive, questioning, curious and observant. They very helpful to look at the rest of the group. If they are must notice everything around them, and, if possible, all showing the same response, then either they are all explain it. They must always be critical in the true sense abnormal, which is unlikely, or they represent normal- of the word. They spend their lives diagnosing disease ity, in which case, the patient is also normal.

and must understand quite clearly that, as health is nor- It should be remembered that the owner and his staff mality, then disease is simply deviation from normality. are an important part of the patient's environment, and From this, it follows that veterinary surgeons will never can play a large part in the disease process and progress. be able to detect and recognize disease, i.e. deviation They, equally with, or perhaps even more than, other from the normal, until they know the normal. They environmental factors, merit critical assessment. Are must learn the normal in all domestic species in many they competent? Are they humane? Are they inter-

different circumstances. For normality is relative, not
ested? Are they truthful? Have they contributed to the
absolute. It is relative to breed, age, nutrition, the stage
disease process? They merit critical study, for the staff
of lactation or pregnancy, management and many other
are inextricably involved with their animals and the one
factors.

cannot be considered without the other.

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The important diagnostic principles that should

(1)

The time of year. Many diseases are seasonal, e.g.

always be followed are shown below.

the coughing calf indoors in November probably

has a viral or bacterial based pneumonia, but the

(1)

Be systematic.

coughing calf at grass in August probably has

(2)

Adopt a routine that is found suitable and stick to

husk.

it.

(2)

The part of the country. Each type of country has

(3)

Take nothing on trust.

its own disease problems, e.g. in the hills one meets

(4)

Apply continual critical assessment to oneself and

malignant catarrhal fever, bracken poisoning and

monitor one's own attitudes and techniques.

piroplasmosis, while in intensive lowland dairying

(5)

Consider all the time:

areas one meets coliform mastitis, milk fever and

(a)

Is there a problem?

hypomagnesaemia.

(b)

If so, then define it.

(3)

The individual farm. The clinician will often know

(c)

Having defined it, what is best done about
that a specific farm has a particular disease
it?

problem, e.g. a parasitic problem among the
young stock, because they are overcrowded and

The clinician's approach to

underfed.

(4)

The type of management. For example, heavily

the clinical case

stocked cubicle yards with straw bedding often
lead to environmental mastitis.

There is nothing magical about clinical work. It depends
upon care, patience, thoroughness, method and logical

Such patterns of disease become second nature to
routine; a routine of examination that covers all the
veterinary surgeons, and always hold good for the part
likely eventualities. Once having evolved such a proce-
of the country in which they work, although revision

dure, there should be no short cuts without good
might be necessary in a practice 400 miles away.
reason, and, however cursory some sections of the
The clinician will, therefore, arrive at the farm with a
examination, for various reasons, sometimes have to be,
diagnosis already half formed. It will be adaptable, not
nothing should be omitted without due consideration.
dogmatic, a *working hypothesis*, to be confirmed or
refuted; however, it is quite remarkable how often this
hypothesis stands up to the test of full clinical exami-

The clinical examination

nation, and, more importantly, refutes the presence of

The owner's message

any other disease process.

Thus, if the clinician knows that the cow to be visited
The owner's message (usually termed the owner's com-
is showing convulsive signs at pasture, that it is early
plaint) should include name and full address, and may
May, that it is an intensive dairy area in Britain the
reach the clinician by direct word of mouth, by tele-
chances that the disease condition is anything other

phone or by mobile or radio telephone direct to his car.

than hypomagnesaemia are too remote to be signifi-

The owner's complaint will also include the type of

cant. Nevertheless, a clinical examination is always

animal and the main presenting syndrome, e.g. a scour-

necessary.

ing cow, a downer cow, a convulsing cow or a bloated

cow. It is important to distinguish between the owner's

complaint and his diagnosis. The owner's message will

History

often be in the form of a diagnosis and the clinician

must be aware that this will frequently be wrong. For

History is all important and the foundation of

example, a farmer may assume that a recumbent cow

diagnosis.

and recently calved will be suffering from milk fever.

(1)

Immediate history is the story of the present

However, a full clinical examination may reveal acute

illness.

mastitis or calving paralysis.

(2)

Past history is anything in the animal's earlier life

There are a limited number of main presenting syndromes that may be relevant, e.g. calving and service dates, and the clinician should learn differential lists something similar at the same point in a previous for each of them, to include only the major differential lactation, and so on.

conditions, not the rarities. It should be remembered

(3)

Herd or group history is important when adverse when considering the differential lists that the common nutritional or management factors may be relevant to the present illness. things occur commonly; that is why they are common!

The clinician should know the main presenting syndrome and its differential list, and during the journey History taking is an art – it does not come naturally, to the farm should consider the following points.

but must be learnt and practised. After a few pleasant

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introductory words, questioning can begin while

(1)

One must know the normal.

walking to the animal, which may be approached, but

(2)

If the animal is frightened, pushed about or even

should not, if at all possible, be disturbed. First, look.

handled, abnormalities may appear due to annoy-

Check the owner's statements mentally by reference to

ance and fear. The minutiae that might have given

the animal and the surroundings. Do not hurry the

a diagnostic lead will be masked. Condition,

owner at first: let him or her talk, then ask your own

vigour, demeanour, respiratory pattern, appetite,

questions, checking each statement and following a

rumination, faecal passage and type, and response

routine that suits you. You are not wasting time for you

to environment should all be noted.

are, meanwhile, carrying out the next two parts of the

clinical routine.

Preliminary examination

Do not ask leading questions in the hope of saving time, or in the hope of confirming one's half-formed diagnosis, for they often produce inaccurate or misleading answers. Human nature being what it is, the examination includes the rate and character of the pulse, the temperature and a check on skin, eye, membranes, udder and ruminal movement. Although suggestive.

pulse character and rate are very important, it is frequently quite impossible to examine the pulse properly. Study the owner or stockworkers. Are they reliable? Are they telling the truth? Are they covering up errors in management? Are they guessing? Every clinician others, driven into a crush and clamped by the neck. It must be interested in the human animal; after all, it is usually not possible to do more than check the heart

the owner whom you serve, advise, encourage or
and heart rate with a stethoscope.

console. In a way, the owner is as much your patient as
The clinician may well have arrived at a diagnosis at
the cow. Admittedly, owners seldom deliberately
this stage, but if not is probably aware of the system of
falsify their story, but it is wise to beware the guilty
the body involved. In this case, he may only need to
one, the bombastic one, the one who knows it all or the
confirm his views, by examination of that system and by
one involved in the sale or purchase of the affected
checking out the other systems quickly, even mentally.
animal.

Nevertheless, the position may still be far from clear,
and the clinician will then have to embark upon a sys-
tematic examination. The procedure so far described

Description

will have taken no more than a few minutes, but if a full
systematic examination becomes necessary, the case
A formal description is more relevant to the examina-
becomes much more time consuming.

tion of a horse, but it is often very useful for the clinician to classify the animal under observation mentally while the history is unfolding, e.g. a young Friesian cow,

Systematic examination

recently calved and heavily in milk. It is always dan-

This includes all the manual and instrumental techniques necessary as part of such an examination, even under examination as it may lead to loss of face in disincluding surgical intervention in some cases. The clinician, according to training and inclination, may interpret the word systematic as meaning (i) thorough, starting at the nose and finishing at the tail or (ii) system by system, in which case a mental list of the systems or sections that should be examined can be ticked off mentally. Like the description, this inspection, *note* inspection not tally when satisfied. examination, takes practically no time, and is carried There is merit in adopting the second routine. It

Preliminary inspection/observation

out during history taking. The clinician observes the enables one to examine first the system or disease behaviour of the animal or group of animals and section that seems most likely to be affected from the makes a mental note of the conditions in the animal's preliminary evidence. Should the provisional diagnosis environment.

be confirmed, then a great deal of time will be saved, At this stage, do not touch, do not disturb. Stand well for it will only be necessary to check the remaining back and observe from a distance, looking over the pen systems briefly, even, in some cases, mentally, to make wall or gate. Put the picture in its frame. One is looking sure that there is no other disease or lesion present. If for the deviations from normality that constitute there is no provisional diagnosis in mind, then it is re-disease, so:

sonable to begin the systematic examination with the

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digestive system. In cows, this is the most likely cause a logical course of treatment and, where relevant, pre-

of illness in obscure cases. Subsequently, the less
vention. The clinician must be prepared to think, form
important systems and sections can be examined as
a working programme even on insufficient evidence,
necessary.

have enough confidence to convince the owner and
A logical list of systems and disease groups for exam-
enough adaptability to modify the programme tactfully
ination is as follows:

if, and when, new evidence appears.

Owners, of course, are only partly interested in diag-

- Digestive system

nosis. Prognosis is much more important to them. Will

- Respiratory system

the cow get better? Will she be fully productive again? Is

- Circulatory system

she worth treating? And today, unfortunately, another

- Urinary system

factor enters the equation. Will it be inconvenient to

- Genital system

treat the cow; will she need nursing or extra care? If so,

- Locomotory system

she is likely to be slaughtered, provided that she has

- Nervous system

antibiotic clearance and is likely to pass meat inspection.

- Skin

- Sense organs

- Lymphatic system

The main presenting syndromes

- Liver

- Udder

It is not intended to discuss here the detailed techniques

- Parasite problems

of the clinical examination of the various body systems.

- Metabolic disease

Every clinician has learnt these techniques as part

- Allergies.

of basic training, and there is excellent coverage in

The last five headings on this list are, of course, not many textbooks, e.g. Boddie's *Diagnostic Methods in* systems: nevertheless, they are very important disease

Veterinary Medicine (1970). Instead, this chapter sets sites (liver and udder) or

disease groups that are logi-out to consider the differential diagnosis of some, cally considered as entities on the clinician's list.

although by no means all, of the main presenting

A rectal examination is an important and useful aid

syndromes that occur from day to day in the dairy cow.

to diagnosis and must always be included if the pre-

senting signs are not conclusive. The bladder, uterus

Drooling at the mouth

and ovaries, kidney and rumen are easily palpable, as

(kiddling, slobbering)

are the caecum and small intestines if dilated.

This is due to factors causing excess salivation, or

factors preventing normal swallowing or combinations

Laboratory and special examinations

of both.

These are very important today, although increasing

costs and the decline of dairying have unfortunately

Bacterial or viral lesions in mouth

decreed that most laboratory work, apart from statu-

tory Ministry investigations, or that done in the prac-

In countries where foot-and-mouth disease (FMD) has

tice, is limited to the diagnosis of herd problems, rather than individual cow disease.

infection must always be considered (see Chapter 43c).

It is important that the clinician should never become

Thus the UK outbreak of 2001 shows how important it

a slave to the laboratory. The laboratory investigation

is that the disease is always remembered in differential

is intended to confirm or refute the provisional diagnosis.

Its occurrence was 33 years after the last

sis (working hypothesis) already in your mind.

major outbreak. The consequences of failure to recognize

Clinical examination comes first. Laboratory results

nize this disease are quite terrifying. Any salivating

can be inaccurate, irrelevant and confusing as well as

bovine animal, whether lame or not, whether pyrexia or

expensive, but if used logically and critically they can be

not, must be treated as a possible case of foot-and-

of great help.

mouth disease until the clinician is satisfied that it is not.

Laboratory investigations include: haematology, bio-

Other pyrexial diseases with oral lesions may give cause
chemistry, bacteriology, virology, parasitology, serology,
for concern, particularly bovine virus diarrhoea
biopsy (histology) of tissues or fluids and, finally,
(BVD)/mucosal disease (see p. 853), infectious bovine
necropsy.

rhinotracheitis (IBR) (see p. 289) and occasional cases
Even after full examination, one may not have a com-
of malignant catarrhal fever (see p. 935), but, fortunately,
plete diagnosis. However, there should be sufficient evi-
the mouth lesions and signs caused by these three dis-
eases to make a provisional diagnosis, which will allow
eases are usually relatively superficial and mild.

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Actinobacillosis causes mechanical difficulty in

Excess salivation of CNS origin

tongue movement. The lesion is obvious, but painless

This may be seen in lead poisoning, for the toxin affects
even when ulcerated (see p. 824).

the salivary nucleus. Blindness and aimless wandering

Calf diphtheria is limited to the young animal, causes in the cow, abdominal pain

and convulsions with bel-pain as well as difficulty in mastication or suckling and lowing in the calf, should help the diagnosis.

produces inflammatory swelling with necrosis of the

In the 'licking mania' ketotic complication of milk mucous membrane of the cheek (see pp. 250, 822, 825).

fever or in 'nervous acetonaemia' (see p. 794) excess salivation is due to the compulsive chewing so charac-

Foreign bodies

teristic of the disease.

These are rare in the mouths of cattle, but the lids of cans occasionally become wedged between the lower

Rhododendron poisoning

molars and the cheek. Rarely, a stick becomes impacted between the rows of lower molars, or a molar itself

This may cause profuse salivation, and even vomiting, loosens (see p. 825).

as well as diarrhoea (see p. 943).

Interference with swallowing

Organophosphorus compounds

This may be due to paralysis of the fifth or seventh

These may also cause salivation and diarrhoea (see

cranial nerves which, in turn, may be due to listeriosis, p. 940).

meningeal abscesses or, particularly in the case of the seventh nerve, to trauma to the side of the head or face.

Acute photosensitization syndromes

Fifth nerve paralysis makes the cow unable to close her mouth. These may well cause profuse salivation, as well as

mouth completely. The lips are in apposition but the lacrimation, jaundice and, later, the onset of necrosis of teeth are not, a fact which is easily checked by flicking the non-pigmented skin areas (see p. 884).

the lower jaw upwards, when the clicking together of upper and lower molars may be heard. Fifth nerve

Acute dyspnoea

paralysis may often lead to accumulation of partly masticated food or cud in the pharynx, producing marked

This condition, as in very severe pneumonia, may cause discomfort and gagging.

obvious salivation as a result of mouth breathing.

Seventh nerve paralysis causes unilateral or bilateral paralysis of the lips and cheek, and if of central origin,

Acute anaphylactic shock

as in listeriosis (see p. 904), also causes flaccidity of the

This often causes profuse drooling of saliva as well as eyelids, with resultant keratitis and drooping of the ear tachypnoea, subnormal temperature, cold extremities, on the affected side. This condition also leads to drop-oedema, and a very rapid heart (see p. 927).

ping of the cud bolus out of the mouth on the affected side and thus, ultimately, to loss of condition.

Vomiting (see p. 825)

Oesophageal choke, particularly in the high oeso-

Vomiting is very rare in the cow and, to all intents and phageal position, causes anxiety, even distress, with

purposes, only occurs in *laurel* and *rhododendron* poi-arching of the neck and profuse salivation. Choke may

soning (see p. 943). Very occasionally a peptic ulcer of also cause varying degrees of ruminal tympany (see

the abomasum will cause problems (see p. 844). Regur-p. 826).

gitation of rumen fluid or content (false vomiting) may

Dilatation and diverticulum of the oesophagus, particu-occur in cases of acute bloat (see p. 852), acidosis (see

larly in the low cervical position, may produce similar,

p. 829) or where there are lesions in the oesophageal though milder, signs, as accumulation of food in the groove (see p. 147), e.g. actinobacillosis or a foreign diverticulum may eventually lead to blocking of the body ('wire') (see p. 837).

oesophageal lumen, with resultant accumulation of Occasionally, cases of oesophageal dilatation and food and saliva above the lesion.

diverticulation may accumulate material above the obstruction until it overflows into and out of the mouth.

Actinobacillosis of the oesophageal groove may lead not only to low-grade ruminal tympany but also to reflux of

Acute abdominal pain (colic)

ruminal and reticular fluid into the mouth, so that in shippen-housed (i.e. tied) cows, a pool of such fluid may

It is interesting that acute colic pain of the type seen in be found in the manager in the morning (see p. 823).

the horse is relatively rare in cattle, and several of the

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syndromes that cause the most acute pain are not, in

Acute fermentative colic

fact, obstructive gut lesions, as one would expect, and This condition, involving the caecum and colon and pro- may not even involve the alimentary tract at all.

ducing right flank tympany, may occur on rich pasture, particularly in wet weather, and may be difficult to dif-

Acute gut obstruction

ferentiate from acute obstructive conditions. Fortu-

This may be due to the following causes:

nately, these conditions generally respond to analgesics and antispasmodics before serious diagnostic errors

- *Abomasal torsion (see p. 842);*

occur (p. 847).

- *Torsion of caecum, colon, or ileum or common mesentery (see p. 847);*

- *Strangulated mesenteric hernia (see p. 1113);*

The passage of a calculus down the ureter

- *Strangulated scrotal hernia (see p. 1113);*

- *Gut tie: the entrapment of gut in peritoneal tears at*

This may produce the most spectacular colicky pain,

the edge of the internal inguinal ring due to castra-

which fortunately is usually relatively transient. Diag-

tion by traction in the slightly older animal (see p. 1113);
nosis is obviously extremely difficult (see p. 263).

- Intussusception (see p. 847);

Photosensitization involving teats

- Obstruction of ileum by the stalk of a lipoma (see p. 849).

Acute photosensitization (see p. 884) in cattle, e.g. Ayrshires with sparingly pigmented teats and udders, Abomasal or large gut torsion produces a degree, can produce an inflammatory exudative lesion of teats sometimes very considerable, of right-sided abdominal and skin of the udder that is so painful that the cow distension, not normally seen in small gut obstructions. behaves exactly as if suffering from an acute abdominal catastrophe. In gut tie, there may be considerable distension in the inguinal region, whilst scrotal hernia obviously produces distension of the scrotum on the affected side. whatever may seem to be affecting a cow, the udder and

It is, however, very easy for the clinician to overlook teats must always be examined.

scrotal or inguinal swelling unless aware of the possibility. These acute obstructive conditions cause the most acute colicky abdominal pain in the very early

Acute abdominal distension

stages, but such pain is often very transient and by the

This is one of the important and relatively common pre-time the case is presented to the veterinary surgeon,

senting syndromes in the cow. All causes of abdominal temperature is falling, pulse rate is rising, body surfaces

distension in the cow are covered by the memory rhyme

are becoming cold and even clammy, and a dull tox-

(the seven Fs), which runs as follows: fat, fetus, fluid,

aemic and shocked appearance develops as circulatory

flatus, faeces, food or foreign body. Severe abdominal

obstruction, necrosis and gangrene of the obstructed

distension may originate at several sites.

gut supervenes. Intussusception is sometimes an excep-

tion in that many cases of bovine intussusception, par-

ticularly if large gut is involved, show little more than

Ruminal

depression and dullness even in the early stages. Rectal

Dietary ruminal impaction: This occurs in housed fat-examination is a useful diagnostic procedure.

tening cattle on store diet, hay or straw, with limited water (see p. 236). The massive ruminal impaction,

Acute enteritis (Chapters 14, 15, p. 850)

absence of ruminal movement, raised temperature and

Acute enteritis, particularly salmonellosis (see Chapter

pulse rate, painful frequent abdominal grunt, and hard

15), is similar to acute cereal overeating, e.g. barley

scanty faeces produce a syndrome requiring differenti-

poisoning, in that some cases show spectacularly acute

ation from acute traumatic reticuloperitonitis ('wire') abdominal pain, often with some degree of tympany,

(see p. 837) in its very early stages, although the

which remains noticeable for a considerable time, often

impactive mass in the 'wired' rumen is never so great.

several hours, before the characteristic profuse diar-

A similar condition, although much less severe, may

rhoea begins. Laparotomies have been performed on a

occur in dairy cows tied in shippens during the winter number of occasions at this stage of the disease in the should the water bowl cease to function, a mishap that belief that the condition was one of acute gut obstruction. Once diarrhoea appears, temperature falls often to worth checking. Modern systems of building and man-normal or below, while signs of dehydration, electrolyte agement have markedly decreased the incidence of imbalance and shock appear.

these two conditions.

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Impaction of the rumen with grain (see p. 829) occurs fails for various reasons, allowing a virulent diffuse in cows that have broken into a food store and eaten exudative process to spread across the peritoneal greedily, but even when the attendant is unaware of this, cavity. Usually, the condition stems from the break-or will not admit it, the nature of the case generally down of the defensive mechanisms isolating 'wire' in becomes apparent in two to three days when profuse

reticular diaphragmatic adhesions, but the leakage or diarrhoea, staggering, recumbency, subnormal temperature of a superficial liver abscess may produce the same result.

effects occur in severe cases. In cases seen soon after The peritoneal cavity fills with thin evil smelling pus ingestion of large quantities of grain, palpation of the of Arcanobacterium (Actinomyces; Corynebacterium) left sublumbar fossa may produce the same sensation pyogenes: there are widespread adhesions, temperature as handling a sack of grain. If, as is generally the case falls, pulse rate rises, scanty diarrhoea develops, body in Britain, the grain is barley, the interim period of condition falls away and the cow is toxic and in pain. impaction, with arched back, grunt, depression and The fluid wave is very obvious and confirmation by moderate tympany is usually transient, and within a few paracentesis is easy. The condition most likely to cause hours the profuse diarrhoea so characteristic of barley confusion is the massive milky ascites that may follow

poisoning appears.

extensive liver damage and obstruction to the portal circulation. Inspection and examination of peritoneal

Vagus indigestion (see p. 855): This condition is usually fluid provides straightforward differentiation.

a complication of 'wire' that has produced adhesions, involving the medial wall of the reticulum and the

Peritoneal tympany: A gas-filled abdominal cavity may cranial sac of the rumen, interfering with the function occur following perforation of an abomasal ulcer at a of the vagus nerve receptors in their walls. The atonic point free of attached omentum, causing acute pain rumen, gradually filling with water and saliva, eventually produces a massive left-sided fluid distension in a right side. Grunting, tooth grinding, falling temperature cow steadily losing body condition.

and rising pulse rate are followed by progressive peritoneal tympany due to leakage of gas from the abomasal lumen into the abdominal cavity.

Acute ruminal tympany (see p. 832): Acute ruminal

tympany presents relatively little diagnostic difficulty.

The condition includes clover and kale bloat and

Abomasal

oesophageal obstruction (choke). The history, environmental circumstances, season of the year and the

Dilatation and/or torsion of abomasum (see p. 842):

gagging, retching and salivating associated with most

Dilatation and/or torsion of the abomasum on the

cases of choke are generally reasonably conclusive,

right produces right-sided distension, very marked in

although in very acute cases, of course, it pays to relieve

cases of torsion, which also causes acute and even

the tympany via the left sublumbar fossa before worry-colicky pain.

ing too much about the aetiology.

It is worth remembering that a thirsty cow, housed

Acute left abomasal displacement (see p. 839): This condition in old-fashioned accommodation and

dition produces significant abdominal distension of the

released to drink its fill of very cold water, may easily

left flank, but such cases are exceptional. All abomasal

become, transiently, quite alarmingly tympanitic, due cases are amenable to diagnosis by normal auscultation presumably to the chilling effect of the frosty water on methods.

the rumen musculature.

The acute ruminal tympany of a cow that, for one reason or another, e.g. milk fever, has collapsed into

Intestinal

lateral recumbency should not require mention. It is,

Acute tympanitic fermentative intestinal colic: This

however, surprising that it is still quite commonplace to

condition, which usually resolves without too much dif-

find that such a cow has not been supported in sternal

ficulty, can produce massive right-sided distension.

recumbency, as the condition demands.

Torsion of caecum and colon on the common mesentery

(see p. 847): This serious and usually fatal complication

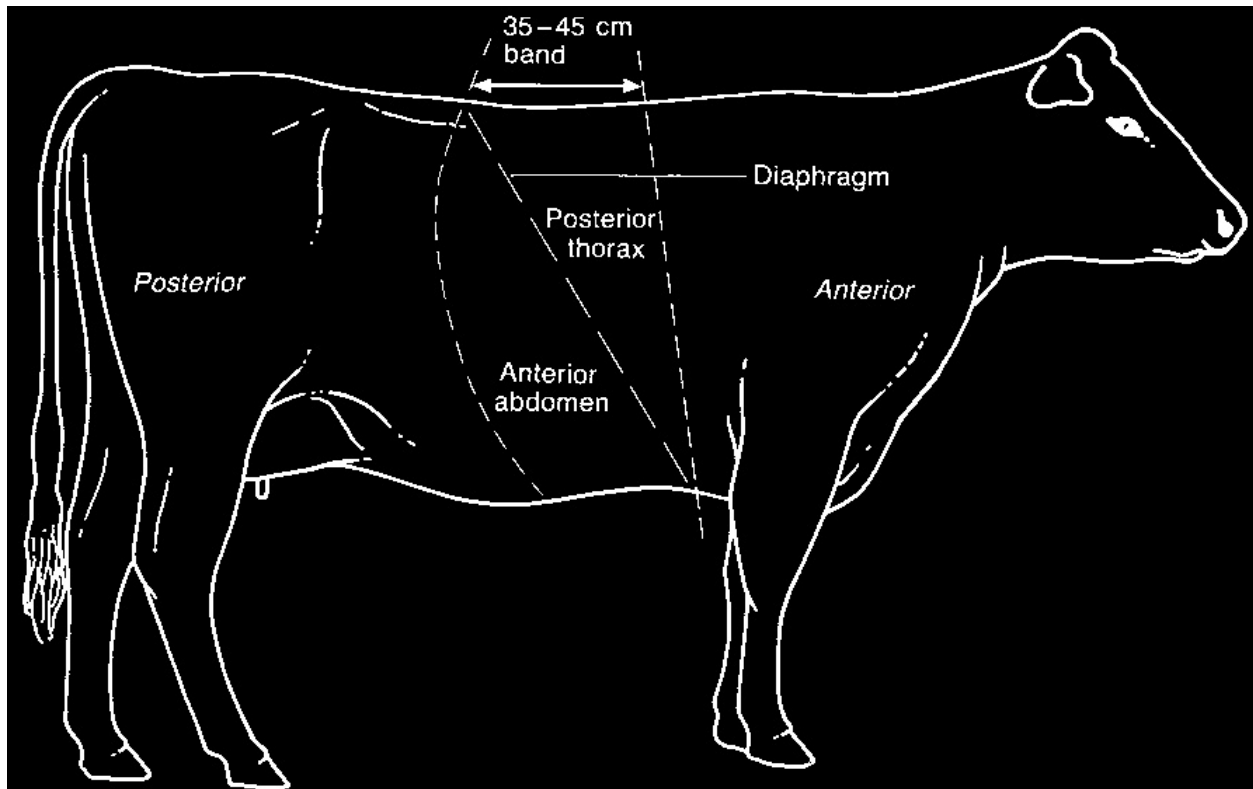
Peritoneal

of fermentation and atony also produces massive

Acute diffuse peritonitis (see p. 849): This condition right-sided distension.

Differentiation depends upon

occurs when the special ability of the bovine peritoneum to withstand and localize peritoneal infection progress of the case following initial medication.



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Fig. 11.1

*Lateral view of cow showing
anterior abdominal/posterior thoracic
area.*

Uterine

Traumatic reticulo-peritonitis

Hydrops amnion and allantois (see p. 1119): These con-
The classical and traditional condition affecting this
ditions produce very marked abdominal distension.
area is traumatic reticulo-peritonitis ('wire') (see p.
Diagnosis is helped by rectal examination. Uterine
837) and it is very convenient to use this condition, less
rupture producing extra-uterine pregnancy in the last
common nowadays, but well known and recognized, as
third of pregnancy is marked by the formation of much
a yard-stick with which to compare the numerous other
fibrinous exudate in an abdomen filled with free uterine
conditions of the posterior thorax and anterior
fluid and containing fetus and membranes. Such an
abdomen.

abdomen is distended and shows a fluid wave which, on
The possibility of overlooking a penetrating foreign
rectal examination, appears to be unassociated with the
body, a serious mistake for both patient and clinician,
uterus. There is abdominal pain, rapid pulse and often
is always at the back of one's mind when examining
severe illness. Diagnosis depends upon an accurate

a bovine abdomen. It is always a considerable relief if history and the ballottement of a fetus in the lower one can eliminate the possibility of a foreign body and abdomen, even though rectal examination reveals a its complications, and such a process is, of course, very partially involuted uterus.

important to the surgeon when considering the site of a possible exploratory laparotomy. Nevertheless, neither the confirmation nor the refutation of a provi-

Anterior abdominal/posterior thoracic pain

sional diagnosis of traumatic reticulitis is easy without The organs and structures enclosed within a 35–45 cm exploratory laparotomy. Thus, the clinical syndrome band encircling the cow immediately behind the withers presents such wide variations in both the extent and provide a group of diseases of great importance to the intensity of signs that it is probably true to say that the bovine clinician. Viewed from a lateral position, the only constant clinical sign is the presence of some diaphragm divides this area into the posterior thorax degree of pain in the anterior abdomen. The range of

anteriorly and dorsally, and the anterior abdomen ventrally and posteriorly (Fig. 11.1).

several groups of clinical conditions, each capable of The band described encircles lungs, mediastinum, confusion by virtue of exhibiting one or more of the heart, pleural cavity and great vessels in front of the signs associated with reticulitis.

diaphragm, and rumen, reticulum, omasum, abomasum, The acute clinical syndrome not infrequently encountered at the onset of the condition includes complete peritoneal cavity posteriorly with much of the greater inappetence, and ruminal and reticular atony, resulting and lesser omental sheets. Differential diagnosis is in ruminal impaction and slight tympany, with absence often difficult and is not helped by the fact that both of ruminal movement and cudging. Constipation may thoracic and abdominal lesions occur within this imaginary belt.

(104–105°F) with a pulse rate of 80–90/minute. There is

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a marked drop in milk yield. The painful focus in the

traumatic reticulitis if seen in the early stages

anterior abdomen results in general rigidity with arched

before diffuse peritonitis, collapse and death

back and protruding neck, disinclination to lie down

occur. However, there is likely to be acute pain

and spontaneous grunting accentuated by movement,

over a much wider area of the anterior abdomen,

defaecation and micturition, and very marked on

particularly on the right side, than is the case in

pinching the withers or applying upward pressure to

reticulitis. Any temperature rise is likely to be

the xiphisternal region.

transient only, but the pulse rate will be consider-

This acute syndrome is by no means constant, even

ably in excess of that expected in reticulitis and

at the onset of reticulitis, and when present usually

may exceed 100/minute. There will be grinding of

abates in 24–36 hours as the intensity of pain lessens

the teeth and loud groaning as well as grunting of and varying degrees of ruminal and reticular motility a similar type to that seen in acute reticulitis. The return. Temperature tends to fall into the 39–39.5°C cow will not eat, is greatly depressed and remains (102–103°F) range, pulse rate may be in the normal largely recumbent. In a proportion of cases, range or slightly raised, appetite, although poor, is not though by no means all, the abdomen becomes completely absent, ruminal impaction and tympany distended with gas, presumably by leakage largely disappear and faeces regain normal consistency. through the perforation, and a state of true peri- The painful rigid stance relaxes and although the back toneal tympany develops. This syndrome must remains somewhat stiff and arched, pain may require also be considered in the differential diagnosis of elicitation by the clinician rather than being obviously acute abdominal distension. spontaneous. Rumination, however, nearly always remains absent, or irregular and occasional.

(4)

*A peritonitis syndrome varying from a very acute
In some cases the only clinical signs are slightly
picture associated with shock to a subacute picture
depressed appetite and rumination, subnormal milk
sometimes associated with straining is occasion-
yield and indications of pain so slight that they amount
ally met with as the result of penetration of the
to no more than unwillingness to depress the back, so
rectum by a foreign body, usually a broom or
that even mild and painless diseases such as aceton-
pitchfork handle, introduced through the anus by
aemia have to be considered in differential diagnosis.
a person with sadistic tendencies. Such a lesion is
always detectable by rectal examination.*

Localized peritonitis

(5)

*The more acute lesions of tubercular peritonitis
(see p. 863), as seen in the breakdown forms of
The acute and subacute syndromes described above
the disease, occasionally produce an abdominal*

are, of course, essentially those of localized peritonitis, syndrome similar to that of traumatic reticulitis.

and pictures similar in broad outline will result from

Unless there are obvious coincidental signs of other causes of localized peritonitis, although the veterinarian may be able to differentiate the focus of pain. the abdomen with the intention of performing

(1)

Penetration of the involuted uterus or the vaginal rumenotomy when the blood-streaked, caseating fornix by a catheter or damage to the anterior and even exudative lesions of the acute disease vagina at service may produce a picture similar, may be only too obvious. This syndrome is now save for considerably less interference with alimentary motility and for pain response on should the disease again break out.

pressure over the posterior abdomen or on rectal examination. Such injuries frequently

*Having considered the differentiation of traumatic
evoked straining, indicating the need for vaginal
reticulitis from other causes of peritonitis, one must
examination.*

*now be prepared to differentiate moderately severe
forms of the disease from conditions which, although
(2)*

*Postoperative peritonitis may well provide a syn-
not involving peritonitis, cause pain in the anterior
drome similar to that of moderately acute trau-
abdomen or posterior thorax. This question of pain in
matic reticulitis, but the fact of recent operation
the anterior abdomen or posterior thorax is, of course,
and the possibility of peritonitis resulting there-
very important in the diagnosis of reticulitis. One must
from will be obvious to the clinician, particularly
remember that the reticulum, diaphragm, liver, aboma-
after procedures such as trocharization for the
sum, omasum, heart, pleurae, oesophagus and the pos-
relief of bloat.
terior lung areas all lie approximately along the vertical*

(3)

Perforation of an abomasal ulcer (see p. 844) may line between the point at which one pinches the withers present a picture broadly similar to that of acute and the point at which one applies the bar.

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Bacterial endocarditis (see p. 726)

sand or gravel, may occur very occasionally. There is a slow diminution in appetite and milk yield and progressive ruminal impaction comprising solid food material with, occasionally, a little gas. Rumination ceases entiation from other cardiac diseases, but it is not general and constipation occurs. Temperature is never more erally realized that, in its earlier stages, before signs of than slightly raised, but pulse rate may eventually venous congestion and circulatory stasis supervene, exceed 100/minute. At first, there is slight anterior endocarditis can easily be confused with traumatic reticulitis abdominal pain only, but as the disease progresses,

culitis. Pain, often intermittent in nature, and causing pinching of the withers and pressure over the xiphisternum are resented markedly. Pain may, in contradistinction to the case in reticulitis, be evoked by pressure over the withers, xiphisternum and left ventral aspect of the chest, is probably due to infarction of the lungs or myocardium. This is not unlike that due to a foreign body penetrating the reticulum. Once venous congestion is clinically obvious, the fact that the heart is distended becomes apparent and from this point the exploratory laparotomy reveals the distended doughy abomasum.

larly traumatic pericarditis.

The author believes that true abomasal impaction is Previous to the development of venous congestion, very rare, and that most of the cases described as abo-accelerated respirations with dyspnoea and coughing masal impaction in the past have shown distension of on exercise, the peculiar 'shifting' lameness of endo-the fundic portion of the abomasum with material like carditis, the tendency towards a markedly high pulse dry rumen contents and an accumulation of fluid within rate even when temperature is responding to antibiotics the rumen, which suggest very strongly that they are, and the presence, in some cases, of recognizable abnor-in fact, cases of 'vagus indigestion' (p. 855). Lym-mality of heart sounds may all help in differentiating phosarcoma can cause the problem (p. 693). endocarditis from reticulitis. The author is cautious on the subject of heart sounds, for although he has frequently been assured by skilled cardiologists that a Painful conditions of the liver (see p. 147) murmur will always be audible in this disease, he has These obviously present a problem in differentiation

frequently failed to detect one in cases with a right-
within the group of diseases causing pain in the ante-
sided lesion. It is noticeable that endocarditis cases tend
rior abdomen or posterior thorax. The liver is a very
to retain a relatively bright demeanour and reasonable
difficult organ from the clinician's viewpoint. It is
appetite until the late stages of the disease. The white
anatomically inaccessible and, in spite of the consider-
cell picture is of limited value as many cases present
able volume of work carried out in recent years, there
total and differential counts similar to those produced
are still no entirely satisfactory tests of liver function in
by a penetrating foreign body, although there is a ten-
the bovine species.

dency for both total white cell count and neutrophil

The cow has large reserves of liver tissue, and very
percentage to be higher than in that disease.

considerable damage may occur without the production
of a clear-cut syndrome. Liver biopsy is of limited value

Certain cases of pneumonia and pleurisy

in that the portion of liver obtained may be quite unrep-

(see Chapter 49a,b)

representative of the whole. The author prefers to make an
These conditions, particularly the latter, exhibit signs of
incision behind the last rib, in the right sublumbar fossa,
posterior thoracic pain that may simulate reticulitis and
sufficiently large to allow a manual examination of the
careful auscultation of the chest is necessary in an
liver and even, using a small torch, limited visual exam-
attempt to confirm the presence of abnormal thoracic
ination. A biopsy specimen can, if required, be obtained
sounds. Pleurisy is very painful in the early dry stages,
through such an incision with the minimum of risk.
but becomes painless as effusion develops. Respirations
are rapid and shallow, but as effusion builds up, they
Pyelonephritis (see p. 725)

become deep and swinging.

In its more severe forms pyelonephritis produces pain
that, although not sited in the anterior abdomen, can

Impaction of the abomasum (see p. 844)

easily lead to confusion with traumatic reticulitis, par-

Impaction of the abomasum, involving primarily the

particularly as the white cell count is also usually raised. It
pyloric outlet with large quantities of fibrous foodstuffs,
may be said that, in spite of the raised temperature and
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pulse rate, the arched back and the grunt not infre-
Subacute and chronic ruminal tympany (see p. 832):
quently present, urine examination leaves the diagnosis
These do occur in traumatic reticulitis, particularly in
beyond doubt. Nevertheless, failure to observe the
the early stages, and often superimposed on a degree of
urine may easily lead to error. Rectal palpation will
ruminal impaction. A number of other conditions also
reveal an enlarged kidney or differentiate cystitis from
result in slight ruminal tympany, which forms a differ-
pyelonephritis (p. 725).

ential group in its own right (see chronic bloat, p. 832).

Impaction of the omasum

Cases of traumatic reticulitis (see p. 837) requiring Another confusing condition,
which is practically

differentiation from other causes of anterior

impossible to diagnose in life, is impaction of the

abdominal pain and conditions causing stiffness

omasum. The disease, which is fortunately very rare,

and rigidity of stance

produces slow weight loss, inappetence, low-grade

Tetanus (see p. 733): This is not infrequently misdiag- anterior abdominal pain and general dullness. Ulcer-

nosed as reticulitis in the first instance. Not only is there

ation and necrosis of the abomasal leaves are found at

subacute tympany, but the arched back, stiff unbending

post-mortem examination.

stance and marked constipation of tetanus can be quite

confusing.

Diaphragmatic hernia (see p. 848)

Bilateral solar ulcer (see p. 417), laminitis (see p. 420) In cattle diaphragmatic hernia produces signs much

and other hind foot lesions (see Chapter 31): Similarly, more suggestive of anterior abdominal pain, particularly

but with less justification, cases of bilateral solar ulcer,

reticulitis, than of any thoracic involvement. It is, in fact, laminitis (coriosis) and other painful bilateral hind foot

probable that many cases arise due to weakening of the

lesions may cause similar confusion.

diaphragmatic muscle by previous foreign body ('wire')

damage. The hernial ring is usually small, involving parts

Injury to lower cervical vertebrae: Occasionally, cows of the reticulum and sometimes the omasum. The result-suffer injury to the lower cervical vertebrae, resulting in

ing low-grade pain may actually be due to areas of peripain, stiffness and reluctance to bend the lower neck or tonitis caused by the original 'wire'. The interference back, and such cases have sometimes been diagnosed as with reticulum and omasum, due to the constriction of traumatic reticulitis.

the hernial ring and the development of adhesions, sometimes leads to 'vagus indigestion' (p. 855).

Complications of, and sequelae to, traumatic reticulitis (see p. 837) causing confusion in diagnosis Uterine torsion (see p. 1118)

of anterior abdominal/posterior thoracic pain

It is worth remembering that the occasional cow which
(1)

Cases are encountered where a piece of wire loose develops uterine torsion very early in parturition may in the reticulum, by reason of its shape, repeatedly show subacute or chronic abdominal pain with pro-

pricks the reticular wall and is then dislodged, progressive inappetence and constipation. Such a case may produce minor episodes of pain and localized peritonitis that rapidly resolve. By the time operation has been decided upon, the animal is substantially

Cases of acute traumatic reticulitis (see p. 837) with normal and surgery is withheld, only for the syn-ruminal impaction requiring differentiation from other conditions presenting this feature

time. It is possible that such 'pricks' should be included with transient phases of abomasal

These conditions are discussed in the section on displacement as the reason for many of the 'non-ruminal impaction and include dietary ruminal specific inappetence' cases so well known to every impaction in yarded cows on fibrous feeds, e.g. hay or bovine clinician.

straw, with limited access to water. A less spectacular ruminal impaction may occur in dairy cows tied in ship-

A difficult problem is the case where traumatic reticulitis during the winter should the water bowl cease to function or in groups when the water supply is reduced carried out, only to find that in spite of the presence or ceases, e.g. freezing, bursts in piping, etc. of definite reticular adhesions, no foreign body can Impaction of the rumen with grain shows varying be found. There are three possibilities. Firstly, the degrees of ruminal distension and discomfort for adhesions may be longstanding and bear no varying periods before diarrhoea supervenes (p. 829). relation to the present illness, i.e. the diagnosis is

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incorrect. This possibility can be checked by applying digital pressure to the adhesions – if pain is provoked, they are probably pertinent to the present type occasionally interfere sufficiently with the bile ill health. Secondly, the foreign body may have

ducts to cause jaundice.

become dislodged and passed down the gut, or

(5)

Occasionally, traumatic reticulitis leads to acute even regurgitated, in which case prognosis is good.

diffuse peritonitis, the abdomen filling with pus

Thirdly, the foreign body may have passed com-

and producing abdominal distension, toxaemia,

pletely through the reticular wall and be buried in

depression, weakness and diarrhoea, leading to

adhesions and reactionary tissue beyond. Here the

death (see abdominal distension, p. 140).

prognosis is obviously grave.

(6)

Vagus indigestion is also a complication of trau-

One can only advise a wait-and-see policy, but such

matic reticulitis (see abdominal distension, p. 855).

cases do present difficulties in the management of

clients who expect the production of a foreign body

and, in its absence, are frequently inclined to doubt the

Chronic bloat (chronic and subacute

diagnosis and regard the operation as an error on the
ruminal tympany) (see p. 852)

part of the clinician.

All conditions producing low-grade ruminal tympany
(3)

An occasional, but nevertheless difficult, case is
must fall into one or other of two main groups: (i) those
the cow from whose reticulum a foreign body has
affecting normal rumino-reticular tone and motility
previously been removed and which now, weeks
and (ii) those causing partial obstruction to the escape
or months later, is showing a clinical syndrome
of gas from the rumen, motility and tone remaining
suggestive of 'wire'. Has there, in fact, been a pen-
normal.

etration by a further foreign body or are the signs
due to further infection or abscess formation in

Conditions affecting normal rumino-reticular

the old adhesions? In cases that do not respond
tone and motility

promptly to antibiotic therapy it is always wise to

re-operate, for even if no further foreign body is

Chronic inflammatory lesions of the mucous membrane

involved, an abscess may be found that can be

and wall of the reticulum and the oesophageal groove:

drained into the reticulum.

Actinobacillosis of these sites is the most important

condition of this group (see p. 823). The smooth pain-

(4)

A further group of conditions occurs where a

less fibrous plaque of this disease interferes with both

foreign body penetrating the reticulum has since

eructation and rumination, producing a mild tympany

penetrated another organ. Signs, in these cases, are

most obvious after feeding. If the oesophageal groove

usually related largely to this secondary occur-

is badly affected there is often a prolonged retching gur-

rence and the signs, all-important prognostically,

gling noise as the cow makes laboured attempts to bring

of the primary foreign body aetiology tend to be

up the first bolus of a new period of rumination. There

masked. The classical example of this type of

may be drooling from the mouth.

condition is traumatic pericarditis, producing a

Therapeutic response is normally rapid enough to be
syndrome very well recognized, but presenting
considered diagnostic.

considerable difficulty at times in differentiation

Occasionally, inflammatory thickening and *A. pyogenes*
from endocarditis.

abscessation of the area resulting from foreign body lac-

Penetration of the thoracic cavity to produce suppu-
erations and partial penetrations may occur, also affecting
rative pneumonia or pleurisy also occurs, tending to
eructation and rumination. Chronic ruminal atony can
produce a subacute thoracic syndrome with progressive
occur in calves with oesophageal groove problems.

loss of condition. It is of considerable importance prog-
nostically to know whether such a condition is due to

Inflammatory changes (peritonitis):

Inflammatory

a penetrating foreign body or not and it is often

changes involving the serous lining of rumen, reticulum

extremely difficult, in the presence of pain in the posterior or even abomasum are more important, leading to poor anterior thorax, to decide whether pain exists in the motility and even atony of the rumen-reticulum. The anterior abdomen as well. The white cell picture will classic syndrome in this group, as already mentioned, is not help, and exploratory laparotomy may be necessary *traumatic reticulo-peritonitis* ('wire', p. 837). Even in as a diagnostic aid. longstanding cases, where some degree of rumen Similarly, foreign body penetration of the liver may movement has returned, there may well be chronic occur causing a large area of suppuration, which may ruminal tympany often superimposed on low-grade produce a clinical picture similar to the acute liver fluke ruminal impaction.

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A peptic abomasal ulcer (see p. 844) sufficiently cattle (p. 698) and enlarged posterior mediastinal lymph advanced to involve the peritoneal lining may well form nodes in the adult. Such enlargement is an important omental adhesions, and may even perforate among

cause of chronic tympany and is usually due to one of these adhesions, so that there is no leakage into the three infective organisms: *A. pyogenes*, *Actinobacillus* peritoneal cavity itself. Such a cow, instead of dying

ligneresi and, occasionally, *Mycobacterium bovis*.

within 24–36 hours of acute diffuse peritonitis, toxaemia and shock with massive peritoneal tympany, as

Diaphragmatic hernia (see p. 848): This condition also described under abdominal distension, will pass into a

not infrequently leads to chronic tympany.

state of intermittent low-grade pain, abnormal or negligible rumino-reticular movement with slight and inter-

Diagnosis of chronic and subacute ruminal tympany

mittent ruminal tympany. There will be lethargy, weight loss and intermittent diarrhoea.

Diagnosis depends firstly upon a careful clinical examination to decide whether the tympany is related to a

Acidosis (see p. 829): This shows its classical features specific alimentary condition or whether it is due to loss

in barley poisoning, but many recently calved high-of gastric tone as a result of a more general disease. If yielding cows on a high-energy diet develop mild

the condition is primarily alimentary, and one believes acidosis after each feed. The slight ruminal tympany there is no oesophageal obstruction, then auscultation plus near-diarrhoeic faeces in a lethargic cow with sub-should be carried out to check that the left flank optimal appetite and milk yield is familiar in intensive tympany is ruminal and not abomasal (i.e. left dis-dairy herds.

placement). Auscultation should leave one in no doubt, although it is not always easy to distinguish between a *Vagus indigestion* (see p. 835): Occasional cases of gas-filled abomasum, early vagus indigestion (p. 855) vagus indigestion (see complications of traumatic reti-and reticular actinobacillosis (p. 823).

culitis, p. 837, and abdominal distension, p. 140) show an accumulation of ruminal gas forming a marked, but chronic, tympany that may even mask the fluid present.

The liver

The liver is practically impossible to examine clinically

Tetanus (see p. 733): Tetanus frequently produces a

by normal methods. It is unfortunately true that unless

moderate tympany (see rigidity of stance and gait, one adheres to a strict routine, it is only too easy to p. 145).

examine a cow without even thinking about the liver.

The liver has many functions and failure, therefore, may

Cold water: Under ruminal distension, the effect of produce a variety of signs.

cold water on the ruminal musculature is mentioned.

Signs include lethargy, slow weight loss, anaemia, low-

In some herds, in which all the cows drink water from troughs in the yard, the whole herd may show a degree of post-drinking tympany. In some herds, in which all the cows drink water from grade or acute abdominal pain, massive abdominal haemorrhage, ascites, abdominal distension, chronic of post-drinking tympany.

venous congestion, ataxia, ventral oedema, photosensi-

Botulism (see p. 721): With its generalized muscle tization, endocarditis, encephalopathy, dyspnoea with weakness, botulism shows subacute tympany as a minor part of a fatal progressive disease. pulmonary thrombosis, massive nasal haemorrhage

and, occasionally, jaundice. All these conditions may

originate in hepatic disease. Also, one must consider the diverse problems of the high-yielding cow calving down

Conditions causing partial obstruction to the

with a very fat liver predisposing to metritis, coliform
escape of gas from the reticulo-rumen

mastitis, ketosis, low solids, milk fever and even

Motility and tone being normal, these conditions will
infertility.

cause low-grade tympany.

The liver, it seems, not only is the site of specific
disease conditions in its own right, but also may be a
Oesophageal wall lesions (see p. 825): Usually traumatic
background factor predisposing to disease in other
traumatic in origin, these include oesophageal stricture,
organs and systems. In the face of this diversity of signs,
oesophageal wall abscesses, oesophageal dilatation and
all one can suggest is that the clinician should always
oesophageal papillomata, which are usually sited at the
consider the possibility of liver disease when examining
cardia.

a cow. He/she is then unlikely to miss liver pathology

when it occurs.

Lesions causing external pressure on the oesophagus (see Laboratory tests, e.g. haematology, serum proteins,

p. 832): These lesions will also produce chronic

serum enzymes (aspartate aminotransferase (AST),

tympy. They include thymic lymphosarcoma in young

serum alkaline phosphatase (SAP) and particularly

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gamma-glutamyl transferase (gGT)), may be helpful,

fluke infestation (p. 276). This condition must never be

though by no means conclusive. Paracentesis may occa-

forgotten. It occurs in adults as well as young stock, in

sionally help in the presence of ascites, while liver

herds as well as individuals, and although wasting, sub-

biopsy is at least theoretically useful. Unfortunately,

maxillary oedema and anaemia may well be present, the

these techniques are expensive and the farmer of today

disease may merely cause chronic unthriftiness and sub-

is very unwilling to authorize them in any but the most

optimal yield during the winter months, thus needing

valuable of individual cows.

differentiation from a nutritional energy deficit. In fact, So consideration of the liver must be as rigid a rule both problems may occur in the same herd, particularly as checking the udder, and a mental list of the syn- between parturition and peak yield.

dromes involved in hepatic disease can be a great help.

It is worth remembering that in severe fluke infestation, constipation is more likely than diarrhoea, and

Abscessation (see p. 830)

that during the migratory stage of the young flukes, a low-grade abdominal pain syndrome may occur that

This is usually due to *A. pyogenes* or *Fusobacterium* requires differentiation from 'wire'.

necrophorum en route from the rumen, and is probably

Liver fluke infestation (see p. 276) has far-reaching only clinically significant when extensive. Obviously, effects. It may lead to infertility, presumably due to cereal overeating and other conditions likely to damage weight loss, to low milk solids and to salmonellosis, or the rumen wall will predispose to liver abscesses. Bacteria to endocarditis due to bacteria passing to the liver and

teraemias also come from navel infections and pyaemias.

then into the circulation.

Complications of hepatic abscessation may be very

Cirrhosis also results from ragwort poisoning (p. 945),

serious:

causing weight loss, ataxia, encephalopathy, occasional

- Rupture into the abdominal cavity leading to an jaundice and terminal tenesmus in the affected animal. acute diffuse peritonitis.

The encephalopathy produces blindness, head pressing

- Rupture into a major vessel, leading to major haem- and dragging of the hind fetlocks reminiscent of lead poi- orrhage, shock and sudden death.

soning in the adult cow. Ragwort poisoning during the

- Vena cava thrombosis, usually where the vena cava grazing period may well trigger photosensitization.

passes through the diaphragm. This produce

hepatic portal obstruction leading to abdominal dis- tension and ascites; however, it may produce pul-

Other syndromes involved in liver disease

monary thromboembolism leading to pulmonary

Tuberculosis (see p. 862) and *neoplasia* of the liver are abscessation. There will be a painful cough, and dys-relatively rare. Neoplasia includes lymphosarcoma

pnoea, with rupture of abscesses into blood vessels

and adenocarcinoma and is, to all intents and purposes,

producing severe and often recurrent haemorrhage

impossible to diagnose in life, while fatty livers

via the nose and mouth (see p. 867).

may follow bacterial or chemical toxicity, but are much

more common as the result of overfeeding or inappro-

Hepatic necrosis

priate feeding.

Hepatic necrosis due to *Fusobacterium* invasion causes The fatty liver syndrome (see p. 801), resulting from

pyrexia, inappetence, lethargy, rapid pulse, weight loss,

excessive weight gain pre partum, has gained promi-

ataxia and occasionally signs of anterior abdominal

nence in recent years, but is probably less common

pain. The presence of jaundice is variable, but is diag-

today. The syndrome may be involved in many diseases

nostically helpful when it occurs.

in early lactation.

Cholecystitis

Constipation

This condition is rare and difficult to diagnose. Such
Quite apart from the acute gut obstruction syndromes,
cases show ataxia, anterior abdominal discomfort and
constipation may occur in diverse circumstances in
jaundice, with lowered appetite and milk yield. Tem-
cattle.

perature may be raised.

It is interesting that jaundice is much more likely

(1)

Unsuitable fibrous diet, e.g. straw, may produce
to occur in obstructive hepatic conditions than in
ruminal impaction.

parenchymatous change.

(2)

Insufficient water intake may produce ruminal
impaction.

Cirrhosis

(3)

External pressure on the gut, e.g. fat necrosis (see

Cirrhosis in cattle, for all practical purposes, means liver

p. 849), lymphosarcoma (see p. 693), adhesions.

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(4)

Pain:

Chronic diarrhoea

(a)

Postoperative pain.

(1)

Johne's disease (see p. 857): the classical form of

(b)

Injured back – faeces dry out in rectum.

chronic diarrhoea with loss of weight, particularly

(c)

Injured anus and rectum – painful bladder.

from the hindquarters, ventral and submaxillary

(5)

Weakness or paresis: milk fever, broken back.

oedema and anaemia.

Constipation is, of course, a very useful differen-

(2)

Tuberculosis of the intestine (see p. 862), very rare

tial sign of milk fever (p. 781).

today, is always secondary to pulmonary lesions,

(6)

Some poisons, e.g. lead (see p. 944) and ragwort and is similar, clinically, to Johne's disease.

(p. 945).

(3)

Ulceration of the abomasum (see p. 844) produces

(7)

Pyrexia.

scanty and intermittent diarrhoea in a cow losing

(8)

Anaemia. e.g. fluke (see p. 276), piroplasmosis

weight, becoming anaemic and showing signs of low-

(see p. 748). In both these diseases, constipation

grade anterior abdominal pain. Erosion of such an

becomes very marked in long-standing cases.

ulcer into a blood vessel in the abomasal wall pro-

(9)

Ketosis: mild constipation is a frequent clinical

duces more profuse, tarry faeces and may cause death.

sign (see p. 793).

(4)

Amyloidosis is very rare indeed (see p. 928).

(10)

Hypocalcaemia (see p. 781).

(5)

Lymphosarcoma of the large gut is also very rare

(11)

Peritonitis (see p. 849).

in cattle (see p. 693).

Constipation is, of course, rare in dairy cattle and if faeces are completely absent, acute gut obstruction (p.

Herd diarrhoea

1113) caused by intussusception (p. 847), caecal or intestinal torsion (pp. 846, 847) will be the most likely reason.

(1)

Winter scours, or winter dysentery, has long been recognized as an acute, pyrexia and occasionally

Diarrhoea

slightly dysenteric condition, which races through a housed dairy herd in winter conditions and then

Varying degrees of diarrhoea are normal today in intensifies out with minimal long-term damage. There

sive dairy herds feeding concentrates and silage.

has been discussion as to the aetiological organ-

Not all conditions causing diarrhoea originate pri-

ism, various viruses and *Campylobacter* spp.

marily in the digestive tract, but from the viewpoint of

having been incriminated (see p. 852).

differential diagnosis, it is best to consider all conditions

(2)

Various nutritional diarrhoeas:

causing diarrhoea together.

(a)

Spring grass.

(b)

Frosted roots or kale.

Acute diarrhoea

(c)

Fodder beet poisoning, the leaves of which

causes a hypocalcaemia-like syndrome and

(1)

Most toxaemic conditions produce diarrhoea, e.g. diarrhoea.

diffuse peritonitis (p. 849), acute mastitis (staphy-
(d)

Acidosis (see p. 829), whether due to exces-
lococcal, coliform or *A. pyogenes* (Chapters 23,
sive root feeding or more commonly due to
24)), acute septic metritis (p. 519), traumatic peri-
excessive cereal overload. The classical aci-
carditis. These conditions all involve damage by
dosis syndrome is that of barley poisoning
bacterial toxins, and the diarrhoeic faeces tend to
with its acute abdominal pain and low-grade
be relatively scanty, but dark and sticky with a
tympany, followed by profuse diarrhoea (see
fetid odour.

p. 830).

(2)

Several acute septicaemic diseases are associated

(3)

Mineral deficiencies, e.g. copper (see pp. 254, 298)

with acute enteritis and diarrhoea, e.g. anthrax and cobalt (see p. 295) deficiency, producing (see p. 717), transit fever (see p. 286) and other chronic diarrhoea with weight loss and anaemia. forms of acute pasteurellosis (see p. 728), and, of

(4)

Parasitic diarrhoea, e.g. parasitic gastroenteritis course, salmonellosis (see Chapter 15).

(see p. 267) and coccidiosis (see p. 282), largely

(3)

Some virus diseases, e.g. malignant catarrhal fever affecting young stock.

(see p. 935) or the BVD/mucosal disease complex

(5)

Toxicity, e.g. antibiotic contamination of the feed.

(see p. 853).

(4)

Several plant poisons, e.g. solanin from green

There is no easy way to diagnose the diarrhoeic

potatoes (see p. 943), water dropwort (see p. 943),

animal. It is necessary to consider all environmental

and rhododendron (see p. 943).

factors, including nutrition, stage of lactation, season of

(5)

Several chemical poisons, e.g. arsenic (see p. 941)

year, housing or pasture; whether the condition is an

and certain organophosphorus compounds (see p.

individual animal or herd problem and whether it is

940).

acute or chronic in nature.

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Other signs besides diarrhoea may indicate whether

months when autumn-calved cows are at peak yield and

the faecal changes are due to a primary gut problem or

possibly in energy deficit, accentuates the whole picture

are secondary to a systemic disease.

of energy deficit and ketosis.

Finally, one must utilize whatever laboratory tests

for bacteria, parasites, viruses or minerals are available

Chronic hypomagnesaemia (see p. 787)

under the circumstances. Even so, there will be one-off

In cold windy weather in late winter, heavily pregnant

conditions, rarely met with in one's practice area, which cattle develop a state of chronic hypomagnesaemia as will, from time to time, elude the diagnostic net!

a result of low food intake and cold, becoming thinner and poorer until some additional stress triggers off the

Tenesmus (straining) (see p. 838)

acute convulsive phase.

This condition occasionally occurs in cattle. *Coccidiosis* (see p. 282) in calves is an excellent example due to

These four conditions, interconnected to varying painful inflammatory changes in the hindgut. The

degrees, account for much of the unthriftiness found

BVD/mucosal disease (see p. 853) complex in young

in dairy herds. A further condition, which may also be

stock may have the same effect. Straining is also seen

involved, is copper deficiency (p. 298).

in the late stages of *ragwort poisoning* (see p. 945), in occasional cases of *terminal intussusception* (see p. 847) *Copper deficiency* (see p. 298)

and sometimes in *urolithiasis in the male* (see p. 263).

Copper deficiency may be either in its own right or

It may also follow *sadistic human behaviour*, when

resulting from molybdenum excess. In the latter case, sticks or broom handles have been forced into the rectum, frequently penetrating the wall of the hind gut grade copper deficiency, particularly at the beginning of some 30–45 cm (12–18 inches) proximal to the anus. the grazing season when the rapid grass growth dilutes Occasionally, there will be sufficient straining to pass the copper uptake, with weight loss and infertility the hard dry faeces in the cow with milk fever of some only signs.

hours duration, to produce transient doubt in the attendant's mind as to the possibility of there being a further

Other conditions leading to weight loss

calf *in utero*. Obviously, the most frequent causes of straining in the cow are obstetrical, e.g. parturition, dys- On an individual animal basis, Johne's disease (p. 857), tokia (p. 1115) or vaginal injury (p. 519).

which does not always cause diarrhoea in the early Rectal examination may make things much worse if stages, and, historically, tubercular emaciation (p. 862)

tenesmus is marked, but a look at the mucous membranes are important causes of weight loss, as are abomasal disorders such as left displacement (p. 839) and ulceration just within the anus with a pencil torch may be very helpful. In coccidiosis, cryptosporidiosis and the BVD complex, particularly, the acutely inflamed nature of the hindgut often spreads right to the anus itself. Chronic liver disorders, e.g. ragwort toxicity (p. 844), may also result in poor thriving to the point of emaciation before clear-cut aetiological pointers appear.

The unthrifty or emaciated cow

A number of other conditions lead to weight loss and suboptimal yield, but reasonable and sensible

Starvation

clinical examination should produce a definitive diagnosis. Even in developed countries today, starvation can still be seen in poorer areas as a primary condition, but the following.

much more frequently appears as a relative energy

- Lameness (laminitis, solar ulcer and white line deficit in high-yielding cows near peak yield. In the lesions) (Chapter 31), often ignored by farmers who period from parturition to peak yield, it is frequently completely fail to realize the serious nature of the associated with ketosis.

long-term effects.

- Chronic pyelonephritis (see p. 725), which should *Ketosis* (see p. 793)

be obvious provided the clinician remembers to

Ketosis is nothing more nor less than energy deficit and examine the urine.

may if untreated produce very severe weight loss.

- Actinobacillosis of the tongue or oesophageal groove area, already discussed on pp. 823–6.

Liver fluke infestation

- Paralysis of the fifth or seventh cranial nerve. (see p. 276)

- The long-term toxic and depressing effects of Liver fluke infestation, reaching its most dangerous in mastitis, metritis, or postoperative peritonitis with

terms of liver damage in the late autumn and winter adhesions and peritoneal abscessation.

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The ‘downer’ cow (see pp. 439, 797)

- Acute staphylococcal (see p. 332) or coliform (see p. 334) mastitis.

A ‘downer’ cow used to be defined as a cow that

- Acute septic metritis (see p. 519).

remained recumbent after treatment for milk fever due

- Hypomagnesaemia (see p. 787).

to a continuing hypophosphataemia (p. 791). But, by

- Cereal over-eating: acidosis (see p. 829).

general usage, the term has come to mean any cow

- Fodder beet poisoning (see p. 149).

recumbent at, or near, parturition, and thus there is a

- Botulism: a condition increasing in frequency and large differential list.

associated with the feeding of big bale silage. Diagnosis is aided by a normal temperature, slow pulse

Preparturient

rate and slowly progressive generalized muscular

weakness affecting both voluntary and involuntary

- Liver fluke (see p. 276) and/or starvation (see p. muscle (see p. 721).

796). These cows may have cirrhotic livers. They are

- Internal haemorrhage, with fast pounding heart, thin, weak and lethargic, but not ketotic.

white membranes, rapid respirations and often a

- The fat cow syndrome (see p. 796). Occasionally, subnormal temperature.

grossly fat preparturient cows with pathologically fatty livers may become recumbent.

Diagnosis of the 'downer' cow is never easy, unless there is an obvious major fracture.

It is important that every 'downer' cow should be

Parturient

given full doses of calcium and phosphorus, and possibly

(1)

Prolonged dystokia may lead to exhaustion.

also magnesium. It may be very logical to argue that a

(2)

Rupture of cervix or uterus leads to recumbency

particular cow cannot possibly be a case of hypocal-
through shock and is quickly fatal.

caemia, but it is easy to be wrong. It is much safer to treat, (3)

Traction injuries.

just in case. With reasonable care no harm can be done.

(a)

Sacroiliac disarticulation (see p. 448).

Once satisfied that metabolic possibilities have been

(b)

Sciatic nerve paralysis (see p. 439).

covered, a careful examination of the available parts of

(c)

Torn adductor muscles (see pp. 439, 459).

the skeleton should be carried out, but may not be very

(d)

Fractured pelvis (see p. 446).

fruitful. It is not easy to assess injuries to the legs of a

(e)

Fractured femur (see pp. 441, 444).

‘downer’ cow deep in sludge or slurry!

(f)

Peroneal paralysis, which is much more

It is vital that the udder and the uterus should be prop-

likely to occur after milk fever than after

erly checked and, as far as the udder is concerned, every parturition, because it is secondary to muscle

quarter must be examined however difficult access may

atony in recumbency (see p. 438).

be.

Parturient and postparturient

The cow ill during or after calving

- Complications of milk fever (see p. 781)

There is no excuse in cases of periparturient illness for

- Fractures, dislocation and muscle injuries (see

the taking of any form of short cut. The clinician must

Chapter 32).

be satisfied that the cow has not developed any form of

- Ruptured gastrocnemius (see p. 451).

milk fever, and even when satisfied, it may be wise to

- Peroneal paralysis (see p. 438).

give calcium/magnesium mixture subcutaneously as a

- Sciatic nerve paralysis (sensory and motor) (see

precautionary measure.

p. 439).

The uterus must be examined to make sure that it is

- Pressure ischaemia of hindleg or legs, which may not infected and not damaged and, above all else, that lead to ischaemic muscle degeneration, particularly there are no more calves within. Even after removing the the semimembranosus and semitendinosus muscles. third calf, the uterus should be checked for the fourth!

Serum AST and creatine phosphokinase levels rise

It is essential that the udder is properly examined. A markedly (see p. 439).

parturient dairy cow may be very ill and dangerously

- Ketosis and licking mania (see p. 793).

toxaemic before the udder shows more than a small

- The cows seems bright and well, but stays down.

crepitating area just above the teat, and that may be all

Never attempt to make a cow with milk fever get

that is noticeable in some cases.

up before it is entirely ready to do so (see p. 797).

Pyrexia of unknown origin (PUO)

Notice that the following conditions are all important in the differential diagnosis of the 'downer' cow (see pp. Obviously, many conditions may cause a marked rise 439, 797).

of temperature in a cow, but a lethargic cow with a

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temperature of 40.5–41°C (105–106°F) is more likely to *chauvoei* and bruising of muscle groups predisposes be a case of incipient mastitis (Chapter 23) than any- to the problem (see p. 723).

thing else, and should always be treated as such in the

- Wound gas gangrene infections: other clostridial absence of other signs.

organisms involved in wound infections, usually

The next possibility is an active pulmonary hyper- obvious at post mortem (see p. 724).

aemia as an early stage of pneumonia, while an acute

Occasional cases of acute coliform (see p. 334) infec- septicaemic condition, such as salmonellosis (Chapter tions, salmonellosis (see Chapter 15), pasteurellosis (see 15), may show pyrexia and little else for some 8–12

pp. 281, 728) and RSV pneumonia may produce a very hours before diarrhoea supervenes. A rectal examination may discover diarrhoea in the early stages that will not have been seen by the stockman. During this period, the total white cell count may be as low as

Acute pasture conditions

1500–2000 per mm³, with a neutrophil percentage of

- Bloat: classical signs and environment, with a less than 10 per cent.

reasonably clear post-mortem picture, provided

One should remember that *anthrax* (see p. 717) is that the carcass is examined within an hour or so of also an acute septicaemic condition, and there have death (see p. 832).

been many cases of early anthrax where lethargy and a

- Hypomagnesaemia: classical convulsive syndrome temperature of 41–42°C (106–108°F) are the only signs on spring grass. The carcass is often covered with present. In young stock, severe diarrhoea, often debris, sweat and mud, with signs of convulsive

bloodstained, with injected membranes, may supervene movement in the grass for a considerable distance within a few hours to be followed by ataxia, collapse around (see p. 787).

and death. In the adult cow, it is not infrequent for the

- Fog fever: occurs in late summer or early autumn.

hyperpyrexia early anthrax picture to be followed by

As it is a herd problem there are usually other

collapse, subnormal temperature, clammy skin, cyanotic

animals with signs. The lungs show a fairly charac-

mucous membranes, restlessness and anxiety, followed

teristic post-mortem picture (see p. 866).

quickly by coma and death. The clinician must beware

of a stage in this process when the cold recumbent cow

is easily mistaken for a severe milk fever case. Realiza-

Electrocution and lightning strike (see p. 930)

tion will come when it is seen that injection sites are

There may be no signs whatsoever on the carcass, partic-

trickling dark blood, while spreading haematomata

ularly in electrocution cases. The behaviour of neigh-

appear where a vaginal examination has been per-

bouring cows, if there is any witness, may help in formed, the teats have been handled or the stockworker electrocution, while it goes without saying that if a cow is has gripped the cow's nose.

to be struck by lightning, there must be a thunderstorm.

Sudden death

Accident or catastrophe

This is a dangerously misleading heading. Very many Road traffic accidents do occur but strangulation and cows that the owner regards as cases of sudden asphyxiation as a result of faulty yokes and feed trough death are in fact cows found dead, which is quite a fittings are probably more common.

different matter. A cow found dead may well have

Catastrophes also include such events as a wire in the taken 12 hours or more to die, depending on the diliricular wall being forced in one movement into the gence of the stockworker and when the cow was last heart. The perforated abomasal ulcer leading to shock, seen.

peritoneal tympany and toxæmia is also included in

If one can assume that sudden death means that a
this grouping.

cow has collapsed and died, if not immediately then
within the hour, then the differential diagnosis is fairly
clear-cut.

Acute haemorrhage

This may result from the following:

Acute infections

- Wire penetration into a great vessel.
- Anthrax: every case of sudden death is anthrax until
- Mammary vein rupture following injury.

proved otherwise (see p. 717).

- Teat vessel haemorrhage following injury.
- Blackleg (see p. 723): usually young stock but may
- The rupture of a coronary vessel.

occur in young adults. The lesion is usually obvious

- The erosion of an abomasal ulcer into an artery in
at post mortem. The causal organism is *Clostridium*
the abomasal wall.

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- Damage to uterine or vaginal vessels after forced

Nevertheless, the more common causes of jugular traction in dystokia cases.

stasis (distension) stem from the heart itself.

- The erosion of a hepatic abscess into a major vessel.
- A thromboembolic pulmonary lesion originating

Traumatic pericarditis (see p. 731)

from liver (see p. 867).

- A superficial haemangioma, often sited dorsally in

Previously the most common form of cardiac disease in the lumbar sacral area.

the cow, this condition has now become relatively rare

- Occasional acute cases of the pyrexia, pruritis and in Great Britain.

haemorrhage syndrome (see p. 884) bleed from the gut, and all other tissues. They also show raised

Chronic vegetative endocarditis (see p. 726)

temperature, severe general pruritis and aggressive behaviour before collapse and death.

This condition has become much more frequent. It is probable that, 30 years ago, many cases of endocarditis

It should be noted that all these haemorrhagic

were missed completely for the clinician was so used to syndromes, with the exception of rupture of a coronary 'wire' and pericarditis that all cases of congestive heart failure were diagnosed as pericarditis and sent to the knacker's yard after the most superficial examination.

anaemic animal with rapid pulse rate, rapid respirations, There are a number of important clinical differences.

loudly beating heart and subnormal temperature. Early

The pericarditis case is much more toxaemic and there-

anthrax, peracute salmonellosis, acute babesiosis and

fore more depressed. Transition from relative health to

acute bracken poisoning may produce the same col-

acute illness is much more sudden, although there may,

lapsed and anaemic syndrome.

of course, have been low-grade reticulitis signs at some

previous stage. Pain is more marked and very readily

Acute anaphylactic reactions (see p. 927)

elicited by wither pinching. Cardiac sounds start with

slight friction sounds synchronous with the heart beat,

There may be much saliva around the mouth with which is usually more than 100/minute. Within a day or oedema of the larynx, pharynx, eyelids, skin of face and so, the tinkling splashing sounds indicative of gas/fluid head, etc. On the other hand, the only lesions may be production within the pericardium can be heard, and in pulmonary.

a further day or so the sac is grossly distended with pus, and splashing and tinkling have ceased. The heart

Poisons (see Chapter 54)

sounds are now muffled and may, in fact, be louder on Theoretically, a number of poisons may cause sudden the right side because the pressure of the distended death in the cow but, practically, sudden death by pericardium against the left thoracic wall tends to poisoning will be due to one of the following:

extend the whole structure across to the right.

Gross jugular and mammary engorgement and

- Yew (see p. 947);

dependent oedema will now be present at jaw, neck,

- Water dropwort (see p. 943);

brisket and lower abdomen. Pulse rate may well reach

- Bracken: acutely haemorrhagic (see p. 946);

140/minute.

- Strychnine: rarely found today in Britain;

Endocarditis, on the other hand, runs a more gradual

- Arsenic: rarely found today (see p. 941);

course. For some time, the picture may be one of low-

- Lead: may cause sudden death in calves, but not grade anterior abdominal pain, plus a temperature rise usually in cows (see pp. 906, 944);

to 40–41°C (104–106°F). Pain is felt over a much wider

- Copper (see p. 948).

area of the chest. The pulse rate is not generally very greatly raised at first. The cow is often relatively bright

Jugular stasis (cording)

and may even eat a little, but exercise tolerance is very

Care must be taken in the interpretation of jugular

poor. Heart sounds, at first, are often no more than

stasis. There is often confusion between jugular stasis

loud, and even later in the disease it is not always pos-

and jugular pulsation, and it must be emphasized that

sible for ordinary mortals to hear the cardiac murmur
jugular pulsation may occur in perfectly normal cows,
that is stated to be invariably present in this disease. The
especially if the head is held low as while grazing.
reason for this seems to be that, in most cases, the lesion
Jugular stasis may occur in conditions involving
is right-sided.

space-occupying lesions in the anterior mediastinum,
At first, therefore, and for several days at least,
e.g. thymic lymphosarcomata in young stock, mediasti-
vegetative endocarditis falls within the anterior
nal lymphosarcomata in the adult (p. 693) or large
abdominal/posterior thoracic pain grouping and is
mediastinal abscesses.

easily mistaken for 'wire'. There may even be a wire, for

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although most cases derive the valvular infection from

Haematuria

the rumen via the liver, from the udder, uterus, pharynx
or feet, occasional cases are met in which the primary

- Chronic cystic haematuria (enzootic haematuria

(see p. 947) or chronic bracken poisoning (see p. 946));
pyogenic focus is a penetrating reticular foreign body.

It is interesting that, in most cases of vegetative endocarditis, the white cell count and the temperature

- Pyelonephritis (see p. 725);

fall during antibiotic treatment, but the pulse rate is

- Calculi (see p. 263);

unaffected.

- Neoplasia other than is involved in the first list point.

Eventually, jugular congestion, shifting lameness,

These four conditions show primary lesions within dependent oedema, pulmonary signs, and even haematuria may all appear.

turia may all appear.

Enzootic haematuria (see p. 946) has a regional inci-

In right-sided cases, pulmonary thromboembolism
dence dependent upon the prevalence of bracken. It
may cause marked thoracic pain, whilst ascites and
tends to occur in older home reared cows and the blood
engorged mammary and jugular veins are very notice-

in the urine, slight at first and slowly increasing, contains pus. In left-sided cases, pulmonary congestion causes very little pus or exudative material.

dyspnoea and coughing with less pain, but haematuria due to renal infarction is more likely.

Pyelonephritis (see p. 725) is much more likely to be encountered in dairy herds, but is, for some reason, a rare disease today in Britain compared with its inci-

Myocardial abscesses

dence in the 1950s and 1960s.

Myocardial abscesses of considerable size may some-

times occur, producing a clinical picture similar to that of endocarditis, but with lower temperature rises and absence of pus, debris and renal casts, as well as blood, in the urine. Rectal examination may help and the presence of pain is another differential point, for enzootic haematuria is painless and afebrile. Bacteriology on the slower course.

the urine. Rectal examination may help and the presence of pain is another differential point, for enzootic haematuria is painless and afebrile. Bacteriology on the

Other causes of jugular stasis

urine of pyelonephritis may, in fairly early cases, Fatty degeneration of the heart, and tubercular peri- produce a culture of *Corynebacterium renale*, which is carditis, are usually masked by more obvious systemic the primary causal organism. Remember that the pain, signs of the respective diseases.

the temperature and the arched back invite confusion with 'wire' unless the clinician keeps pyelonephritis in mind in such cases, particularly in the first third of

'Redwater' (blood or blood pigments

lactation, and insists on inspecting a urine sample.

in urine)

Urolithiasis (see p. 263) occurs largely in the young male, With very few exceptions, disease of the urinary tract in

and other signs such as straining, the absence of significant amounts of urine and, eventually, 'water belly' the differential diagnosis obviously includes the numerous haemolytic conditions producing urinary haemoglobin.

Neoplasia of the urinary tract, other than that due to Obviously, therefore, when presented with a 'redwa-chronic bracken poisoning, is very rare.

ter' case, the first stage in diagnosis is to decide whether the case is one of haematuria or haemoglobinuria.

A number of conditions occur in which haematuria is Microscopical examination or centrifuging the urine but one of a number of fairly obvious systemic signs, so will supply the answer, but usually the simple expedient of standing some urine in a container for a few minutes, while clinical examination is proceeding, will give a satisfactory answer. The clinician must remember that many cows with redwater are not presented as

• Vegetative endocarditis (see p. 726).

• Septicaemic conditions, e.g. anthrax (see p. 717) and acute pasteurellosis (see pp. 286, 728).

such, for stockworkers rarely notice or even see a cow urinate, except by chance. If asked whether the urine is

• Acute bracken poisoning: a disease with markedly raised temperatures and generalized haemorrhages,

normal, the invariable answer is that it must be or it which behaves like an acute septicaemia and probably would have been noticed. This is wishful thinking, upon ably is one (p. 946).

which no reliance can be placed.

Once differentiation between blood and blood

It is worth remembering that very high doses of sulphamides may theoretically produce crystalluria and haematuria, but sulphapyridine, the sulphonamide that comment on a cow's respiratory pattern, it is always most frequently produced these signs, is no longer used. worth comparing it with that of neighbouring cows.

Haemoglobinuria

Pathological

Piroplasmosis is an acute, pyrexia, and acutely

Respiratory rates:

haemolytic tick-borne disease of certain areas of Britain and other countries caused by *Babesia divergens*. The

(1)

Increase markedly in many diseases due to
clinical signs include profuse diarrhoea, followed by
pyrexia.

stubborn constipation in a non-premune cow, progres-
(2)

Increase and become jerky in acutely painful con-
sive anaemia, with very rapid pulse rate, loudly pound-
ditions, e.g. septic feet, acute laminitis (see p. 417).
ing heart, and deep port wine coloured urine (see p. 748).

(3)

Increase in acute toxaemic conditions, e.g. summer
mastitis (Chapter 24), coliform mastitis (see p.

Bacillary haemoglobinuria is a peracute and rapidly
334) and acute septic metritis (see p. 519).

fatal pyrexia due to *Clostridium haemolyticum*

(4)

Increase in metabolic disease, e.g. hypomagne-

(now renamed *Cl. oedematiens* type II). It affects young saemia (see p. 787) or
acidosis (see p. 829).

stock in certain rough hill areas, but is of relatively little (5)

Increase markedly with a much shallower thoracic

importance overall (see p. 719).

excursion in conditions that prevent full pul-

Postparturient haemoglobinuria is seen during the monary expansion, e.g. ruminal tympany, ruminal weeks following parturition in cows in certain parts of impaction and pleural effusion.

eastern Scotland and occasionally in England. It is non-
(6)

Increase in conditions in which the upper respiratory tract is blocked at least in part, e.g. malignant severe anaemia, and is possibly associated with root and catarrhal fever, IBR (see p. 289) and pharyngitis.

straw feeding and abnormalities in phosphorus metabolism.
(7)

Increase and become shallow in conditions that olism. It is too rare today to be of real significance (see interfere with the function of the respiratory
p. 792).

muscles, e.g. tetanus (see p. 733).

(8)

Increase very markedly in severe anaemic

Kale and rape poisoning are too well known to merit conditions, e.g. piroplasmosis (see p. 748) and detailed consideration. Large quantities are required haemorrhage.

and wilted kale is much less likely to cause problems. It

(9)

Increase and become laboured in conditions that, is worth remembering that rape in excess may also for various reasons, decrease the amount of active produce abdominal pain, nervous signs and/or dysp-lung tissue, for example:

noea (see p. 941).

(a)

active pulmonary congestion prior to pneu-

Leptospira icterohaemorrhagiae produces haemoglo-monia (see p. 860);

binuria in the calf, but this is most unlikely to occur in

(b)

chronic venous congestion, e.g. vegetative

the adult (see p. 735).

endocarditis (see p. 726);

(c)

acute anaphylactic conditions (see p. 927);

Very cold water, thirstily drunk, may cause haemoglo-

(d)

‘fog fever’ (acute pulmonary oedema and

binuria in the calf, but not in the adult.

emphysema) (see p. 866); and

Copper toxicity, increasing in the UK, may apparently

(e)

pneumonia (see p. 864).

produce haemoglobinuria (see p. 948).

Acute nervous and convulsive syndromes

Cows breathing badly (hyperpnoea and

Nervous signs occur relatively frequently in cattle of all

dyspnoea)

ages, and vary from mild signs of ataxia and head press-

The clinician must not assume that rapid respirations in

ing on the one hand to hyperaesthesia, circling, muscu-

cattle necessarily indicate the presence of pneumonia.

lar tremors, aggression, collapse and convulsions, on the

Cattle 'blow' for many reasons.

other.

Physiological

Acute hypomagnesaemia (see p. 787)

Cattle breathe more rapidly when full after feeding,

This condition occurs in dairy cattle on heavily fertilized high-protein pasture at turn-out in spring, and are nervous or frightened. A herd coming in for milking again in the autumn. It also occurs in winter and early on a hot summer afternoon after a mile walk from lush spring in beef cattle on exposed pasture. Hyperaesthesia, with muscular tremors of face, eyelids, ears and

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muscles of the head and neck, is followed by strabismus, may be complicated by dyspnoea, haemoglobinuria and generalized muscular tremors and collapse in general-constipation.

ized clonic convulsions.

Listeriosis (listerellosis) (see pp. 251, 904)

Transit tetany

Listeriosis occasionally occurs in silage-fed cattle, producing an initial pyrexia followed by hyperaesthesia, a tendency to aggression, circling, head pressing, facial paralysis with drooping of one or both upper eyelids in magnesium and calcium.

and a dry keratitis. One or both ears may also be involved, and occasionally the fifth cranial nerve as well as the seventh is affected so that chewing and swallow-

Bovine spongiform encephalopathy (BSE)

ing are impaired and food becomes impacted in the pharynx and mouth.

This shows more slowly developing hyperaesthesia with ataxia and behavioural changes prior to collapse. These

Ketosis (acetonaemia) (see p. 795)

changes include apprehension occasionally amounting to panic, obsessive licking of nose and lips, semaphor-

Ketosis is normally a sign of energy deficit in high-yielding dairy cattle between two and six weeks after calving. The condition may well be subclinical, doing nothing at other times and muscular tremors of neck and more than reducing yield. At its worst, it produces inappetence for concentrates, lethargy and constipation, with a normal or subnormal temperature and pulse rate.

Hepatic encephalopathy

Occasionally, a nervous form occurs producing ataxia and excitement, with marked head signs, the patient certain forms of liver disease, e.g. ragwort (see p. 945) or licking itself and anything within reach in an obsessive fashion, holding the bars of the shippen or parlour in its teeth and chewing to such an extent that tongue and

Signs are ataxia, dullness, slow circling, head pressing
lips may bleed forming a bloodstained froth. Ketone
and collapse. Occasional periods of excitement are seen.
bodies are present at high levels in blood, milk and
urine.

Occasionally, this licking frenzy may occur as a com-
Tubercular meningitis (see pp. 251, 862)

plication of milk fever. It is known as 'licking mania'
Not to be forgotten in these days when bovine tuber-
and is believed to be ketotic in origin.

culosis is on the increase, tubercular meningitis may be
seen in half-grown cattle as a sequel to congenital
Acute inflammatory, exudative and/or haemorrhagic
tuberculosis or the consumption of milk from cows with
lesions in the brain

mammary tuberculosis. Ataxia, stumbling, bellowing
and an inco-ordinate aggression with head pressing fol-
Acute inflammatory, exudative, and/or haemorrhagic
lowed by collapse were frequently seen in this condi-
lesions within the brain may cause marked excitement
tion. In the days of horned cattle, fracture of one or

bordering on mania before collapse occurs. This picture both horns often occurred as a result of head pressing. may occur in anthrax (see p. 717), in which case diagnosis will be helped by generalized haemorrhage and oedema, and is also sometimes seen in malignant catarrhal fever accompanied by marked nasal and Blindness in one or both eyes might also occur.

Lead poisoning (see p. 944)

ocular discharge, with heat and pain over the sinuses of the head. Horns, if present, feel hot and in cattle with aimless wandering, often complicated by trauma due to collisions with walls, trees, etc. Bellowing frequently fact, become loose. There is usually profuse diarrhoea. occurs.

Convulsive syndromes that are relatively frequent

Rape poisoning (see p. 941)

among calves

Rape poisoning may produce excitement, aggression,

Lead poisoning (see pp. 906, 944): This is still common, blindness and

bellowing, a nervous syndrome, which

causing abdominal pain, blindness, salivation and con-

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vulsions, with marked bellowing and leading rapidly to

Muscular dystrophy (see pp. 258, 302): Calves dying of death.

heart failure due to the cardiac form of muscular dys-

trophy will normally be housed calves fed only on dam's

milk. Vitamin E intake may therefore be low, and at two

Magnesium tetany (see p. 255): In beef calves fed dam's to three months of age any sudden excitement, such as

milk alone without supplement magnesium tetany

the arrival of the dam at feeding time, may trigger off a

appears at 12–16 weeks of age, producing star gazing,

cardiac failure episode with cyanosis and anoxia which,

stilted gait, muscular tremors and hyperaesthesia

nevertheless, may superficially resemble a convulsive

leading to convulsions and death. Abdominal pain,

syndrome.

blindness and bellowing are not features of magnesium

tetany.

Cerebrocortical necrosis (see p. 261): Cerebrocortical necrosis (CCN) occurring in housed or yarded calves

Gammexane (gamma BHC) poisoning (see p. 942):
may produce star gazing, hyperaesthesia and ataxia but,
This condition is due to feeding milk in buckets con-
occasionally, leads to a severe convulsive picture.
taminated with gammexane, after mixing insecticides,
and causes very severe clonic convulsions and death.

References

Linseed poisoning (see p. 941): The acute dyspnoeic
picture with gasping and muscular spasm that follows

Boddie, G.F. (1970) *Diagnostic Methods in Veterinary Medicine* the feeding of warm
wet linseed to calves may look

cine, 6th edn. Oliver and Boyd, Edinburgh.

much like a convulsive picture unless one is aware of

Rees Evans, E.T. (1957) *Bacterial endocarditis of cattle. Vet-the signs of prussic
acid poisoning.*

erinary Record, **69**, 1190–202.

The Calf

Chapter 12

Outline of Clinical Genetics

G.B. Young

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Introduction

Deleterious major genes

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Genes and disease in different species

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Genetics in hereditary diseases corresponds to the

Genetic epidemiology

162

microbiology of infections. Before Pasteur's contribu-

Economic loss

162

tion to bacteriology, infections were crudely controlled

Modes of inheritance

162

Dominant genes

162

by isolation, but his work greatly improved preventive

Semidominant genes

162

measures. Similarly, genetics greatly improves the

Recessive genes

163

precision in controlling hereditary diseases.

Sex-limited and sex-linked genes

163

Many genetic problems are complex and exhibit a

Multiple alleles

163

spectrum from being entirely genetic to entirely envi-

Irregular inheritance

163

ronmental. The genetic component may be divided into

Genetic polymorphism

163

major gene and polygenic effects. Polygenes each have

Chromosomal abnormalities

163

a small effect and produce continuous variation. Most

Epidemiology and control

164

disease due to major genes is, however, also influenced

Controlling dominants

164

by minor genes and many diseases currently considered

Controlling recessives

164

polygenic may in reality be affected by relatively few

Controlling an irregularly inherited defect

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Genes exhibiting good and bad effects

164

major genes.

Assessing controls

165

Observations and experimentation indicate that, like

Investigating genetic diseases

165

the well-studied Drosophila, cattle carry a genetic load Pedigree analysis

165

of deleterious major genes producing gross abnormality

DNA analysis

165

and of deleterious polygenes producing subfertility,

Differentiating genetic and infectious diseases

165

increased disease susceptibility and poor physique. With

Genetic counselling

166

current knowledge many polygenic constitutional

Genetic defects and artificial insemination

166

defects can only be studied biometrically, but the final

Genetic defects and egg transplanting

166

aim is to recognize individually every gene and its

Polygenic deleterious genes

166

location on specific chromosomes. Recombinant DNA

Sterility and infertility genes

167

technology now makes this theoretically feasible.

Calving difficulties

167

Susceptibility and resistance genes

167

Metabolic disease genetics

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Deleterious major genes

Disease genetics and cattle improvement schemes

168

Inbreeding depression

168

Intensive single-character selection

169

Every body system is subject to abnormal inheritance.

Balanced selection

169

Indeed, if, as the embryo unfolds, a defect occurs early,

Positive selection for health

169

several subsequent systems may be involved, produc-

Selection and longevity in dairy cows

169

ing a syndrome. Environmental disturbances, or other

Bull mothers

169

genes sometimes inherited differently but affecting

Crossbreeding

169

the same developmental pathway, can produce identi-

Health recording schemes

169

cal diseases or syndromes.

Recent advance

169

Genetic diseases affect all ages from conception

Cloning

170

to senility. Congenital malformations, observed at birth,

The genomics revolution

170

Conclusions

171

may be genetic or due to maternal effects such as

infections, nutritional deficiencies or drugs. Genes

control the synthesis of proteins and, when defective,

generally result in an enzyme reduction or deficiency.

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Molecular genetics has developed the concept of

Few breeders escape genetic disease at some time.

control genes, influencing structural genes, but most

Economic data are not available on either the direct

genetic diseases are still thought of in terms of struc-

loss due to major deleterious genes or the greater indi-

tural or chromosomal mutations.

rect loss from counterselection. Genes capable of pro-

ducing defects are, however, abundant and most cattle

Genes and disease in different species

probably carry several. Only fear of inbreeding and

continual selection against defects prevents more

The basic genetic physical structure of all species from

frequent outbreaks.

worms and fruit flies to humans is similar. For example,

the control homeobox genes, the molecular architects

organizing other genes and switching them on and

Modes of inheritance

off at the correct time, are universally responsible for the division into head, thorax and abdomen. The gene *Inheritance* is duplicate: each individual has two genes that controls eye development in humans, when (units of inheritance) for a particular character or function inserted into a fly, produces normal fly development. tion at each locus on the chromosomes. A parent passes This common, genetic, evolutionary heritage implies one on a random basis to an offspring, the other coming that genetic malformations present in one species will from the second parent. Chemically, genes are deoxyribose nucleic acid (DNA) and the fine structure of the humans and Galloway cattle is an example. Further gene has been elucidated, but for most clinical purposes genes can be transferred from one species to another. the gene may still be regarded as the unit of inheritance. Using transgenics, most known human genetic diseases have been transferred to mice.

Dominant genes

Mammalian species probably have around thirty

In regular dominance every carrier is affected. The thousand genes and yet only three hundred are thought disease is generally inherited from one parent and to separate mice from men. Different species are half its offspring are affected. A new dominant gene therefore likely to suffer from similar infections and producing a severe effect tends to be lethal and produces infestations. Tuberculosis and the influenzas illustrate single isolated effects. Surviving dominants produce the point. Studying genetic disease similarities and dis-relatively minor defects, e.g. notched ears in Ayrshires. similarities between species constitutes comparative Irregular rather than regular dominants are more genomics.

common in cattle. An individual may carry the gene but not manifest it due to intangible environmental effects

Genetic epidemiology

or modifying genes (incomplete penetrance). Half of its offspring also carry the abnormal gene but a proportion All cattle breeds possess several genetic diseases, some similarly do not manifest it, producing irregular segre-

*common to many breeds, others specific to individual
gation ratios.*

*breeds. Several hundred have been described and many
Hereditary ataxia (p. 178) in Aberdeen Angus illus-
more are known.*

*trates this inheritance. Transmitting bulls mated to non-
Most persist at a low frequency. Some appear almost
carrier cows leave around 25–40 per cent of calves
sporadically in many different herds over the years.*

*affected instead of the expected 50 per cent, so pene-
Others are concentrated in a few herds in sudden out-
trance is from 50 to 80 per cent.*

*breaks. Occasionally, a defect increases until individual
Some late-developing defects, such as arthritic con-
or collective action reduces its frequency. If breeders
ditions in bulls (p. 176), could, in theory, be due to dom-
relax, its frequency increases again, producing a cyclical
inants. The bull would not exhibit the disease until old,
pattern.*

*and bred from, and because of culling, the relationship
and ratios between bull and offspring might not be*

Economic loss

easily noticeable.

Calf loss may reach 20 per cent within a herd for several

Semidominant genes

years, as in Galloway tibial hemimelia (p. 176). Maternal

mortality may be high as in prolonged gestations in

These are quite common in cattle. A single gene pro-

many breeds (p. 183). Difficult parturitions reduce yield

duces a defect but a double dose increases its severity.

and late abortions result in long dry periods. In later

In the single dose, the abnormal gene converts a Kerry

developing defects, e.g. hip dysplasia (p. 453), only cull

into the small, more desirable, Dexter, but a double

value is obtained. The bull has to be replaced, the breeding

dose produces monsters. Matings of Dexters produce

programme is disrupted and pedigree sales drop. Coun-

on average 25 per cent Kerry types, 50 per cent Dexter

terselection substantially reduces economic selection.

types and 25 per cent bulldog monsters (see p. 175).

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Many dwarves exhibit this inheritance. Often, as in

Multiple alleles

snorter dwarves in Herefords, the carrier conformation Each locus on the chromosome may have not just two is only slightly different from normal and slightly better but several genes present. Various combinations of these (heterozygote advantage). Selection for carriers then genes may produce a gradation of severity of a con- spreads the disease. At the limits, one-quarter of the dition. For example, a series of multiple alleles reducing calves are defective.

melanism successively dilutes coat colour from normal agouti to albinism. Multiple alleles may contribute to the variation in clinical expression of many diseases.

Recessive genes

They are also a common form of inheritance in many disease-related biochemical variants.

A single gene does not show in a carrier because of the normal dominant gene. The defect only appears with a double dose. Neither parent is affected yet the defect

Irregular inheritance

has come from both parents.

In carrier matings one in four of the offspring are

Many, indeed most, genetic diseases of cattle are

normal (RR), two are carriers (Rr), and one will be

inherited irregularly. They do not provide simple

affected (rr). This is true on average but not for

genetic ratios and are characterized by sporadic

individual or small groups of matings. Moreover, pure

incidence and occasional concentration within families.

recessives giving exact ratios are rare, even with the

Arthrogryposis in Charolais characterized by calves

traditional genetic model of Drosophila.

with twisted limbs (see p. 177), cleft palates and a

The abnormal gene is usually considered neutral in

twisted spine, illustrates the problem. More than half of

carriers but in some cases, probably more than is

all artificial insemination (AI) bulls produce a few

currently understood, it may be detected biochemically.

defective calves but a few (about 5 per cent) leave

Carrier recognition is a major field of genetics and

around five per cent of affected calves.

should always be sought in major recessive outbreaks

Cryptorchids provide another example of non-in cattle.

Mendelian inheritance. Cryptorchidism is frequently Recessive defects may also have reduced penetrance. sporadic (p. 182). Most extensively used sires leave Since the frequency of recessive homozygotes is gener- some cryptorchids. Many cryptorchids leave mainly ally low, if further reduced by incomplete penetrance, normal offspring and most cryptorchids have normal inheritance is best described simply as irregular, and parents. Occasionally, however, affected or normal bulls calculating the penetrance of a recessive is generally sire a higher proportion of affected offspring than unprofitable.

average. Their incidence also increases markedly on Recessive defects often appear following line breed- inbreeding. Cryptorchidism thus has a genetic compo- ing. A breeder obtaining good daughters from one nent and probably both male and female contribute to bull is often tempted to use related bulls on these. its occurrence.

These related males may carry the same recessive gene. Such defects result from unknown environmental factors derived from a common ancestor. Some second matings factors and genetic susceptibility, either recessives or are thus between carriers, and defects appear. The dominants, exhibiting a very sensitive threshold of manifestation, pedigrees of affected animals often reveal common ifestation.

ancestors within a few generations.

Genetic polymorphism

Sex-limited and sex-linked genes

This is a discontinuous variation which persists in a population apparently more or less at random. Cattle Many cattle diseases are limited to one sex, for example, blood groups are an obvious example. Their relative ample, testicular hypoplasia (p. 182). The other sex, proportions are not maintained by a balance between however, plays an equal role in inheritance and the mutation increasing the defect and selection removing defective genes are in the non-sex autosomes. This genes in affected animals. Most, however, will probably

inheritance must be distinguished from sex-linked inheritance. It may ultimately be shown to affect fitness. Many may be recessive inheritance where the abnormal gene is a relic of resistance mechanisms to much earlier plagues. It is carried on the X chromosome and a carrier cow transmits the disease to half her bull calves, and leaves half her heifer calves as carriers; a cow with a double dose

Chromosomal abnormalities

of the harmful gene would show the disease. Since sex-linked recessives are not transmitted by unaffected carriers, they are rare in cattle. Structural chromosomal mutations, such as duplications, deficiencies, inversions, translocations (p. 184) and alterations in chromosomal number, are not uncommon in cattle. Large chromosomal breakages produce complete sterility and small breakages subfertility. Both are inherited like irregular dominants. Bulls with low con-

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ations in chromosomal number, are not uncommon in cattle. Large chromosomal breakages produce complete sterility and small breakages subfertility. Both are inherited like irregular dominants. Bulls with low con-

frequency, and the incidence of affected individuals is
ception rates due to minor chromosomal abnormalities
the square of the gene frequency. Thus, if the incidence
pass the defect directly to their sons.

of a recessive defect is 1 per cent, the gene frequency is
Many chromosomal defects, often difficult to detect,
10 per cent and the proportion of carriers about 20 per
are probably present in early embryos, and account
cent. Even a low incidence of defects thus implies large
for a considerable proportion of early embryonic morta-
numbers of carriers.

lity and some reduction in conception rate. The uterus,
Moreover, this gene frequency will be the average for
however, acts as a clearing house for such defects and
the whole breed and much higher frequencies will occur
few progress to birth.

in farms that have used carrier males recently. Thus
the outbreaks and frequencies will be patchy with
some farms heavily affected and others with few or no

Epidemiology and control

defects. This often renders measurement of the

incidence of a defect in a breed difficult. A useful guide
Breeds are generally organized hierarchically. A few
is that a defect attracts notice when around 1 per cent
top herds supply bulls to less influential breeders who
of calves are affected in the breed so that about 18–20
in turn supply commercial producers. If a harmful
per cent of animals are carriers.

gene spreads in the top strata these bulls spread carriers
Reduction in frequency can be rapid if the initial
through the breed. Similarly, if the defect is eliminated
frequency is high but is slower as the frequency de-
from the top herds then sires free from the defective
creases or if the initial frequency is low. Thus breed-
gene slowly reduce the defect in the other herds. The
ers can rapidly reduce a recessive defect at high levels
origin and increase of a harmful mutant in the top herds
but eliminating it completely is very difficult.

is probably due to mutation and genetic drift.

The main difficulty in selecting against a recessive is
Control is the sum of the control efforts of individual
the large number of carriers that cannot be recognized

breeders. Affected herds select against the gene, and on visual inspection. However, if all bulls and cows breeders soon learn from which herds to reduce their producing defects are culled, experience shows the purchases. The distribution of non-carrier bulls reduces incidence of the defect soon drops to acceptable levels. the incidence.

The desirability of control will vary with the severity and frequency of the defect. A defect causing dystokia justifies considerable counterselection, but a minor

Controlling an irregularly inherited defect

defect such as colobomata very little. Strong selection

In a disease such as arthrogryposis (p. 177) all bulls against a defect may also relax selection for important leaving defects cannot be culled but only bulls trans-economic characters.

mitting most frequently. Similarly, in defects such as cryptorchidism, control is based on not using affected

Controlling dominants

animals or close relatives and this is effective in main-

A regular dominant spreads directly down through

taining a low frequency.

a breed but such direct transmission is rare in cattle

Irregular defects such as cryptorchidism (p. 182)

except for minor defects such as some forms of

would only increase if affected animals were continu-

polydactyly. If required, culling all affected animals

ously used. Why they persist at a low level despite gen-

eliminates the defective gene.

erations of natural and artificial selection is unknown

Dominants exhibiting incomplete penetrance, how-

although carrier advantage may be suspected.

ever, commonly spread directly. The lower the pene-

trance, the greater the likelihood of this occurring. If

penetrance is high a few offspring will pinpoint a carrier

Genes exhibiting good and bad effects

parent and transmitters can be culled. If penetrance is

low, control is more difficult, since many offspring are

Some pleiotropic genes or closely linked gene

required to detect carriers.

complexes produce both desirable and undesirable

effects and selection for the good effects may spread the

gene. For example, some genes producing desirable

Controlling recessives

coat colours also cause infertility. Selection for the coat

At breed level, epidemiology and control of recessive

colour may thus spread infertility or maintain it at a low

genes will depend on the gene frequency. This is simply

level, as in white heifer disease (p. 183).

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A gene may be beneficial when single but harmful

Pedigree analysis

in a double dose as in selection for the desirable

A few generations intensively studied are better than

conformation in American beef breeds resulting in

long pedigrees. A list of normal and affected animals

unconscious selection for carriers.

born during and immediately prior to the outbreak,

Inheritance involving advantageous and disadvanta-

their sex, sire, dam and maternal grandsire is generally

geous effects may be much more common in cattle than

adequate. This list will indicate if simple genetic ratios

suspected.

exist.

With recessives one out of four offspring of carriers

Assessing controls

are affected, but in practice the common ratio obtained is one in eight. This occurs when two carrier males are

Excessive controls, such as compulsory recording of

used successively. The first carrier leaves on average

all abnormalities, are sometimes advocated. Controls

half of his daughters carriers (as well as half his sons).

are essential, particularly where a defect has become a

In mating the second carrier bull to the carrier daugh-

problem, but they should be kept in proportion. They

ters, one out of the four offspring are defective. About

should be kept to the minimum level necessary, so that

one in two \times one in four, i.e. one in eight of the second

selection for efficient production can proceed as rapidly

bull's offspring are defective.

as possible.

Care has to be taken with pedigrees since as many as

10 per cent may be inaccurate. Few pedigree investiga-

tions fail to produce anomalous cases.

Investigating genetic diseases

DNA analysis

Inheritance may be suspected when other factors are excluded, the defect runs in families and previous Almost weekly a new gene for a human disease, located reports implicate genetics. Since environmental defects by deciphering the genome, is recorded and some may simulate genetic conditions and similar clinical breast cancers are already subject to routine screening, defects have different genetic causes inheritance in the particularly in patients with a family history. Although affected herd should be investigated.

mapping the genome in cattle is being pursued in some Inheritance exists when one bull has sired all the laboratories, financial restraints limit progress.

defects and another contemporary bull has left none Moreover, reliable results require great technical skill when mated to similar females in a similar environment. However, such controls are often not available

may involve several hundred genes, to the required and a properly designed experiment may be necessary. gene and to assure the specific gene is involved. Until If line breeding is being practised a new defect is more progress is made reliance for diagnosis and probably genetic. If recessive, both sexes are affected control in livestock has to be placed on the more equally and the incidence within a herd seldom rises standard but still relevant procedures such as pedigree above 15–20 per cent. The disease may disappear as analysis outlined previously.

unaffected males are brought in. Since environmental Predicting future diseases seems to cause substantial changes are often made simultaneously confusion may ethical and insurance problems in humans, but in arise.

livestock, while similar problems will arise, they are Simple recessive defects resemble each other relatively trivial. In contrast, predicting future diseases fairly closely – one dropsical calf tends to be similar in livestock is one major way ahead in reducing disease

to another. Where considerable variation in clinical incidence.

appearance or age of onset is present a simple recessive is unlikely.

Differentiating genetic and

In cattle, and especially in a small herd, establishing infectious diseases

the exact inheritance may be difficult. Often all that can be said is that the disease runs in families and comes

Genetic and infectious diseases may, on occasion, from either one parent only or, alternatively, the genetic be confused. Their epidemiology can be similar, with factors are present in both parents.

deleterious genes or infectious agents radiating out

Breed differences are also suggestive of genetic from heavily diseased foci. Moreover, many infectious disease but not conclusive because of possible con- diseases through close contact are familial and genetic founding between breed and environment. This is par- resistance may be present, enhancing the familial ticularly true where infections or mineral deficiencies

aspects. Pathology may even be similar, since an
are involved.

invading organism can affect the same developmental

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pathway as a deleterious gene. The crucial distinctions

Automatically eliminating bulls leaving three or so

are the isolation of an infectious agent and experimen-

affected offspring is rather rigid. Selection against bulls

tal transmission of the disease or the establishment of

leaving calves causing maternal mortality, late develop-

fairly clearcut genetic ratios, perferably the former.

ing defects, or leaving three affected calves in their first

100 offspring should be more intense than against bulls

producing unimportant defects, or producing a few

Genetic counselling

abnormalities among several thousand normal calves.

Again, a few defects among several thousand normal

calves is tolerable from a bull transmitting efficient

In the event of a genetic disease a breeder should be

production.

advised to change the bull immediately, thus eliminat-

When an undesirable gene is increasing, an AI centre ing the appearance of the defect and reducing the should buy bulls from sources thought to be incidence carrier incidence in offspring. Continuous use of non-free. Even with limited numbers of offspring and a low carrier bulls gradually reduces the number of carriers. gene frequency, carrier bulls should soon be detected.

If a carrier male has had only restricted use all his For a serious condition, bulls might be progeny tested daughters should be culled. However, where the herd on known carrier females.

has many carrier females, culling should be gradual Testing all bulls on their daughters would test for to avoid decimating the herd. Known carriers and any deleterious gene. Since most bulls probably carry low-producing females should be culled first. Where several such genes, the bulls available would be limited. the abnormal gene occurs in a particularly good strain Where older bulls are being used, the test might be its frequency should be reduced rather slowly so as to of some value, although low production in the inbred

preserve the strain intact, i.e. some defects should be daughters would be a disadvantage.

suffered to maintain production qualities.

Generally, AI, because of its scientific basis and mon-

In a serious outbreak a breeder might test a male on

itoring procedures, is an agent for reducing rather than

about 20 of its daughters or half sisters, or on 10 known increasing defects.

carrier females. Generally, however, test mating is expensive and best avoided.

Control of defects inherited as irregular dominants

Genetic defects and egg transplanting

or in a non-Mendelian fashion, follows the same

Routine egg transfer permits intense selection among

principles. The bull should be changed and transmitting

females and potentially concentrates even further the females culled.

genetic base. It thus enhances the risk of spreading

When a defect rises in frequency, the breed society

genetic defects. Provided proper surveillance is insti-

should seek veterinary advice. After ensuring that the

tuted and donors transmitting defects rapidly with-disease is recognizable, both clinically and pathologically, like AI, it should however decrease rather than increase genetic disease.

then explained to breeders so they can counterselect most effectively. Prenatal diagnosis and selective abortion, although feasible, should only have limited

Polygenic deleterious genes

application in valuable animals in cattle practice.

There is very marked individual variation in fertility, in susceptibility to infections and metabolic diseases and

Genetic defects and

in conformation and physique. The genetic part of con-artificial insemination (see Chapter 39)

tinuous variation is considered to be due to many additive genes, each with a small effect. They produce a bell-shaped distribution whose mean can be shifted by of genetic disease because they avoid inbreeding, par-selection.

particularly in the larger units, and rapidly withdraw bulls

Multifactorial inheritance, particularly from field transmitting defects. Although most bulls carry several data, is measured by a heritability estimate – the ratio of defective genes, few leave many defects in their progeny of genetic influences to all influences (genetic and since their cow population is unlikely to have a high environmental). Heritability estimates tend to vary gene frequency for the same defect. While a carrier widely according to the method of calculation and the bull leaves half his daughters carriers, each time a non-particular field data chosen and are commonly averaged to obtain a generalized more reliable estimate. tions, the carrier incidence is halved.

Individual estimates have to be treated with great

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caution, especially when used to predict rates of calving problems. Friesians, for example, have more difficulties than Jerseys or Ayrshires. At the extreme limit

only to the population from which they are obtained in pure, large, continental breeds cows can only produce and their predictive value is limited to a few generations a few calves before becoming sterile. Counterselection at most.

is impossible as long as growth rate is given priority and the only remedy is Caesarean section.

Sterility and infertility genes

Susceptibility and resistance genes

Infertility genetics is little understood. Although there is substantial automatic natural selection present for Susceptibility and resistance genes are widespread and fertility, the marked decline in fertility with inbreeding genetic variation in disease resistance has always been and its restoration on crossbreeding demonstrates the demonstrated when adequately sought. Animals relatively resistant to one disease are often susceptible Difficulties and inaccuracies in statistically measuring to another. Resistance is sometimes polygenic but, fertility, and the different indices used, have produced

with increasing research, it has often been found to be very different estimates of its heritability. Most studies dependent on relatively few genes.

suggest the heritability of pregnancy rate in cattle is In natural epidemics an invading organism frequently low, almost zero. Some based on sire/son comparisons, spreads rapidly causing heavy mortality. Some geneti- however, suggest figures as high as 40 per cent.

cally resistant animals almost invariably survive and Intense selection for yield is thought by some workers subsequently multiply. After initial oscillations, host to lower fertility. It can be argued that a few per cent and parasite settle down to coexist. Initially acute decrease in pregnancy rate might be overestimated in eases tend to become chronic as genetic immunity comparison with high yield. Lengthening calving inter- develops.

vals, however, markedly reduce profitability and selec- Before control, selection for resistance genes must tion for fertility should remain a high priority with some have been intense. This effect is still obvious in tropical

culling of sons of bulls with low pregnancy rate.

countries, for example, where Zebus are markedly more

Semen volumes and sperm numbers, concentration,

resistant to local disease than exotic breeds. In grading

motility and morphology show marked age, breed,

up by crossbreeding, a proportion of Zebu genes has to

weight and individual variation (see p. 604). Semen

be retained and indeed in high-disease areas improve-

characteristics are considered to be moderately herita-

ment of indigenous cattle may be preferable. For many

ble (15–20 per cent) and should respond to selection to

large countries resistance genes dictate a stratified

improve semen quality but progress would be slow. The

breeding programme from pure exotics to pure indige-

correlation between sperm characteristics and fertility

nous cattle. In tropical countries genotype–environment

is, however, not entirely clear and some semen stand-

interactions are of prime significance.

ards may be unnecessarily high, leading to reduced

In European cattle the most obvious example of

selection for other characters. Specific sperm defects

genetic resistance is in mastitis. There are marked breed differences. Heritability estimates vary enormously to single genes and should be strongly selected against. but are probably around 10–15 per cent. Daughters of Testicular size and conformation are also influenced infected dams are more susceptible and some bulls considerably by genes. The difference in libido between transmit substantially more mastitis than others. In one beef and dairy bulls is sufficient to indicate the strong extensive survey most mastitis cases were daughters of genetic influence in libido.

relatively few AI sires.

At the herd level most infertility problems are transient and non-genetic. Selection to raise the frequency of mastitis-resistance genes, even with AI, although feasible, would be difficult. Similarly, repeat breeding in individual cows is usually of environmental or management origins. Many daughters, perhaps 250, would be required to classify the bull. Currently sires' daughters are clas-

of onset of post partum cyclicity, probably has a confounded mainly on first lactation yield when mastitis is less considerable genetic component associated with hormonal frequent and the effect of AI on mastitis incidence is differences.

probably neutral. It is dangerous to assume that selection for high heifer yield automatically implies selection

Calving difficulties

for health, as many diseases occur in later lactations.

Genetic selection for yield and growth rate increases

Calf scours and pneumonia also have a genetic com-

ponent, associated with colostral antibody. This varies

body size and larger cows inevitably have increased

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genetically, as does the vitality of the calf, influencing its suckling ability.

There are also breed variations,

suckling ability.

Channel Island breeds being particularly liable. Breed

BSE is another infectious disease where mouse

variation in hypomagnesaemia and acetonaemia also

research published and some unpublished research

exists.

on scrapie and BSE's epidemiology suggest there are
The anaemia common in high-yielding cows provides
strong genetic and familial factors operating. Research
one of the best experimental examples of the strength
has been hindered by the need to prevent its spread to
of biochemical individuality. In one unpublished twin
humans. Since safety in humans can only be guaranteed
study, despite marked prolonged nutritional and weight
by its elimination in cattle, the genetic factors involved
differences, haemoglobin, red blood cells, packed cell
justify much further research.

volumes and mean haemoglobin concentrations were
There are marked species and individual differences
all so highly determined by individuality that pair
in the severity of foot-and-mouth disease and selection
members closely resembled each other in blood
could probably enforce this resistance, but vaccination
pattern, despite the nutritional differences within and
would seem the best approach in such an infectious,
between pairs. Individual differences overshadowed

rapidly spreading disease involving many viral strains.

the nutritional effect.

Isolating the experimental animals could also prove a

Such individual differences indicate the need for

major, although not insuperable, problem.

caution in interpreting disease status from blood analy-

Parasitic infections, however, are emerging as a very

sis in individuals. Because of individual variation nor-

possible candidate for scientific counterselection. They

mality is difficult to define. A single animal with a low

tend to be chronic, natural selection resistance already

haemoglobin may not be anaemic but merely exhibit-

exists and regular treatment is expensive. This field is

ing a normally determined low value. Blood tests are

likely to develop, perhaps particularly in tropical live-

generally most valuable at the herd level in preventive

stock development centres (LDCs).

medicine.

The greatest progress in artificially selecting for

Selection for increasing yields is putting dairy cows

disease resistance depends on detecting biochemically

under increasing stress and metabolic diseases are resistant and susceptible animals.

Considerable

steadily increasing in developed countries. Both milk progress is being made on this front, centring around fever and acetonaemia are basically diseases of high immune response genes. These control the ability of yielders and almost unknown in less-developed the animal to produce antibody against certain specific countries.

antigens. The Ir genes are on the part of the chromosome that contains the genes controlling the acceptance

of tissue grafts, the histocompatibility genes. These

Disease genetics and cattle

*latter can be detected serologically and both Ir genes **improvement schemes** and histocompatibility genes form a multiple allelic series.*

These are essentially interwoven.

Improvement

As knowledge of this gene complex increases and schemes carry risks as well as rewards. Those based on

the genetic basis of immunity is understood in finer AI and egg transplants steadily to improve rates of detail, positive selection for resistance to disease may progress involve the risk of inbreeding depression. become more feasible. Artificial insemination, egg transplanting and, ultimately, gene transfer are the obvious instruments.

Inbreeding depression

Selection for disease resistance is likely to be profitable, however, only where vaccines are not Since relatively few males are required to avoid available, although in some cases genetically more inbreeding, in theory it presents little problem in resistant animals respond more effectively to vaccines. improvement schemes.

Both approaches may in some circumstances be As technology reduces bull numbers and AI sires are complementary.

followed by their sons more care will be necessary. Using a few score bulls for several million cows should not cause serious inbreeding provided sons are not

Metabolic disease genetics

chosen repeatedly from only the very best sires, causing an undue concentration on very few sires. However, Individual variation in susceptibility to metabolic after many decades, such a system could cause a serious disease is well demonstrated by milk fever. It has a accumulation of inbreeding effects. Remedies would heritability estimate of around 20–25 per cent and a include importations, using stored semen or splitting repeatability of about 20 per cent, indicating how sus- the breed into small units and exchanging bulls. Simi-

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larly, with egg transplanting overconcentration on a few selection for longevity at the end of the first lactation mothers has to be avoided.

is possible but would require too many daughters

In practice, however, more inbreeding may be occur- and measurements to be economically feasible. As bio- ring than is generally suspected and with inadequate chemical individuality is explored in greater depth it recording the risks may be being underestimated.

may become possible.

Intense single-character selection

Bull mothers

Single-character intense selection in all species

A simple approach to improving health, and longevity reduces fertility and frequently produces other undesirable correlated responses. The balanced homeostasis of the animal is upset. At the extreme it often uncovers latent defects, e.g. pygmies in downward selection for weight. This approach is now being implemented.

Intense selection for yield in dairy cattle increases mastitis, udder oedema and metabolic diseases. The

Crossbreeding

genetic, early, high-peak yields in dairy cows, much higher than in any other species, induces stress.

Crossbreeding schemes, even those where the primary

High yielders also suffer increased digestive dis-

objective is blending different maternal and paternal orders, foot problems and calving difficulties. High characters, greatly improve health and vigour.

yielders, although more profitable, thus require expen-

The simplest explanation of hybrid vigour is that sive health care, particularly to maintain mammary the load of recessive deleterious genes, which can function.

be exposed on inbreeding, are covered up by normal

The genetic reduction in fertility associated with high alleles on crossbreeding. Crossbreeding is extensively yield is probably sufficiently serious to reassess current used in beef production, particularly where hardiness selection programmes in dairy cattle. Increased effi- is vital, as in exposed hill areas.

ciency of production, rather than yield alone, should be Systematic crossbreeding schemes are being used the objective.

in some Scandinavian, eastern European and tropical countries but generally have been little developed in western Europe.

Balanced selection

The best schemes of cattle improvement, maximizing both health and efficiency, are probably based on balanced selection. Factors such as growth rate and mature body weight, combining crossbreeding and selection and using selection indices, body conformation and composition, food intake indices. Dairy cattle improvement is only in its infancy and efficiency and perinatal mortality and disease resistance are likely to be built into selection indices. schemes are likely to develop in the West.

Selection will probably also occur under controlled conditions as well as using field data. The aim will be

Health recording schemes

better balanced and healthier animals and hence more profitable dairy cattle.

Another approach to genetically improving health is a greater emphasis on health recording schemes.

Currently, the most effective of these are devoted to

Positive selection for health

improving fertility control, particularly regular calving and improved heat detection and, to a lesser extent,

Selection and longevity in dairy cows

early detection of metabolic breakdown. With improved design, however, they are likely to detect bulls

The very short lifespan of dairy cows (and bulls) is transmitting undesirable qualities such as mastitis susceptibility probably due to poor management and particularly

inadequate fertility control. Genetic selection for health and longevity could, however, contribute substantially to better health. In livestock, positive eugenics is possible.

Recent advances

Longevity has a considerable genetic component independent of yield. The heritability of survival to the sixth lactation may be around 20 per cent. Theoretically, genetic achievements of the last decade, the human

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genome project and cloning, are only now being

is, therefore, untenable and each gene must encode for explored, but the genetic revolution will have a major several proteins – estimates vary from the tens to impact.

several hundreds.

This conclusion leads to a switch in emphasis from genomics to proteomics as the fundamental key to understanding the body and attempts are now being

Cloning

made to produce a comprehensive catalogue and map Livestock cloning, when a relatively reliable success of all the proteins. This will be very difficult since rate is achieved, should greatly advance medical proteins are complex, may interact with other science, enabling the rapid build-up of flocks and herds proteins, once created may be modified and can be containing specific genes for generating pharmaceuti-transient.

cal products, as in various Roslin Institute research This changing emphasis is having a major effect on projects. Investigation of the current reproductive

drug development and therapy. Although in a fluid and malformation difficulties should also enhance our state, the new concepts are already tending to emphasize understanding of cell differentiation, and cell multiplication and division, with implications for genetic diseases and even ageing and cancer.

in different patients and the occasional side-effects

Commercial cloning to achieve rapid once and in block busters and vaccines. The key seems to lie in for all improvement may require greater caution.

genetic individuality producing genetic variation in

Widespread use of identical animals could leave individual proteins and hence different responses to different drugs. Medicine, at present, could be regarded as diseases and without biological diversity, whole a diverse collection of symptoms and empirical treatments could be affected. The Irish potato famine, but is slowly being converted into a science. The

was basically caused by cloned potatoes. Moreover, day when each valuable animal has its own genetic the aim is to improve animals every generation profile and can be treated accordingly, however, rather than stabilize current populations with prize- is a very long way off, but that is the direction in winning livestock. In certain circumstances, however, which genomics and proteomics seem to be leading where scientifically improved animals have been pharmacogenetics.

developed, limited commercial cloning could prove Not only some mammals but many infectious agents, worthwhile. Cloning valuable animals before they such as the cholera bacterium, have been successfully succumb to infertility or disease could be regarded as sequenced, work which should lead to more effective dysgenic.

vaccines. Likewise sequencing biting insects such as the One underestimated benefit would lie in exploring mosquito should ultimately lead to better insecticides. nature and nurture. Currently, field heritability studies

With infectious agents and parasites evolving rapidly to tend to produce lower values than twin studies, and the overcome current therapies, sequencing should provide problems of nature and nurture are far from being a better understanding of the resistance mechanisms satisfactorily resolved. In twin research the existence they are developing (they are fairly well understood in of only two identical twins reduces replication and bacteria already) and this in turn should help provide requires very complex experimental designs. Clones better control systems.

would greatly simplify this. In field studies the intro- In evolutionary terms the reduction in gene numbers duction of clones into different herds and environments seems to imply that the vast variation in life forms, would also greatly improve the measure of genetic variation including the domestic species and their parasites, may ation. Apart from specific genetic studies, readily available due more to control genes, switching genes on and able clones would improve nutritional and disease off, rather than a large number of genes producing dif-

research, raising the whole standard of cattle research, different proteins.

as pure lines have done in mice.

In animal breeding terms it is difficult to over-estimate the significance of genomics and proteomics. Currently improvement is largely based on biometrics, particularly heritabilities. In the future this is likely

The genomics revolution

to be supplemented and possibly finally replaced by

The most significant result from the human genome screening the DNA and looking for genes, particularly project is a reduction in the gene count from an estimated hundred thousand or so to nearer thirty to forty

In the even longer term such genes are likely to be thousand. Since there are probably hundreds of thousands inserted into the genotype – after all, all genes in different species can be regarded as part of one vast

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network – and the future of livestock improvement lies

Further reading

in genetic engineering.

Perhaps after a long latent period, during which the

Brock, D.J.H. & Mayo, O. (eds) (1972) The Biochemical

immense successes of microbiology has dominated med-

Genetics of Man. Academic Press, London and New York,

icine, the old-fashioned subject of medical diathesis or

pp. 1–725.

constitutional medicine, rejuvenated by Mendelism, is

Emery, A.E.H. (1983) Elements of Medical Genetics.

now reasserting itself.

Churchill Livingstone, Edinburgh, London, Melbourne,

New York. pp. 1–283.

Hamori, D. (1983) Constitutional Disorders and Heredi-

Conclusions

*tary Diseases in Domestic Animals. Elsevier Scientific Publishing Company,
Amsterdam, Oxford, New York. pp.*

1–728.

Thus in cattle improvement, the future is likely to see

*Lerner, I.M. (1954) Genetic Homeostasis. Wiley, New York, a greater emphasis
on selection for survival and effi-pp. 1–134.*

ciency as well as production, and genetics will become

Nicholas, F.W. (1987) Veterinary Genetics. Oxford University a core subject in cattle preventive medicine. With gene

Press, Oxford, UK, pp. 1–580.

transfer enabling resistance genes to be built in to

Pirchner, F. (1983) Population Genetics in Animal Breeding.

cattle, a new Pasteurian age is developing and the

2nd edn.

Plenum Press, New York and London, pp.

prospects seem limitless.

1–414.

Chapter 13

Congenital Conditions

A.H. Andrews

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with large herds and limited labour, abortions or

Ovarian hypoplasia

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deformities are often missed. When the condition is

Müllerian duct aplasia

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present in the offspring of bulls used in artificial insem-

Duplication of the reproductive tracts

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ination the frequency may again be hard to determine

White heifer disease

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because of the disinclination of farmers to report the

Prolonged gestation

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problem. An indication as to whether or not a problem

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is of genetic origin may be obtained from the type of

Freemartinism

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defect apparent and the knowledge already available

Pervious urachus

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about the condition. Other evidence may be a sudden

Other defects

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outbreak of a defect following the use of a new sire and

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which only affects calves of his parentage. In some cases

Familial polycythaemia

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there is evidence of a gradually increasing number of

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similar abnormalities that occur over a number of years.

Anomalous twins

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Following an investigation, it may be shown that a

Chromosomal translocations

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defect is confined to a particular family within a herd

or to the progeny of certain dams.

Introduction

In most cases the history will show a much lower inci-

dence of any inherited defect in crossbred animals than

Most farmers will, at times, have calves that show in pedigree ones. Some genetic problems are noted as defects of a varying degree at birth. Such defects, being very common in certain breeds or families within although congenital, may be due to genetic or environmental factors or their interaction. The overall level of condition, e.g. hip dysplasia in Herefords (see p. 453). The incidence of congenital defects ranges considerably in history should be indicative of a relationship between surveys from 0.2 to 3.0 per cent. It should be remembered that genetic defects are not always apparent at birth. The incidence of all specific defects is very small, but in repeat sire–dam matings may also indicate the inherited nature of the condition and its mode of inheritance, as also can sire–daughter or sire–half-sister causes can be inherited on a dominant, recessive or

matings, but these involve a considerable period of time additive basis and often they are influenced by the environment before any mode of inheritance can be suggested or confirmed. In many cases all that can be said about a particular condition is that it is familial. Factors connected

Some of the more common conditions are described with the environment are many-fold. Other problems under generalized headings of the systems involved.

are the result of bacterial or viral infections, nutritional deficiencies, chemical poisoning and physical insults. If the condition occurs in the early stages following fertil-

Cardiovascular system

ization (i.e. up to day 14 after fertilization), death of the embryo occurs and it is resorbed. During the embryo

Ectopia cordis

and organogenesis stage (15–44 days after fertilization)

This is an uncommon congenital abnormality where the effect is variable. In many cases there is death of the heart is present outside the thoracic cavity. The cause is embryo with resorption or abortion, whereas in others

unknown. The heart is usually positioned in the region the embryo remains viable and there is congenital of the lower neck and can be seen pulsating when some absence, deficiency or disturbance in function. Once the distance from the calf. Some cases are displaced into the fetus stage is reached (day 45 to birth) then again death abdominal cavity.

can occur resulting, if early, in resorption, otherwise mummification or abortion and, if later, in stillbirth. In

Ventricular septal defects

other cases the fetus survives and may be normal or suffer growth retardation, reduction in size or organic These are the most common form of congenital heart function, or the animal may become weak.

lesion in calves. They vary in their size, which determines

It is often difficult to diagnose the problem and to the severity of the signs and their location, but they are decide whether a congenital condition is inherited or often high on the septum. In some cases the animal survives for many years with nothing untoward being sus-

essary. Often it is hard to ascertain the frequency of a pected. The defects can be single or combined with problem, as farmers are reluctant to admit its presence abnormalities of the blood vessels. The defect allows and also how frequently it is occurring. In many cases blood to pass from the left to the right ventricle.

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The signs mainly depend on the size of the defect. If five days old. The condition is relatively common and it is small the animal may grow normally, have a normal the cause is unknown. Blood passes from the aorta to exercise tolerance and a normal life expectancy. Such the pulmonary artery. Signs are often limited other than cases are usually only detected when the animal is poor exercise tolerance and lassitude. There is no examined for some other reason. Occasionally, calves cyanosis, but there is a continuous murmur often known will suddenly drop dead with no premonitory signs at a as a machinery murmur as it increases and decreases few weeks to several months of age. In severe cases with each cardiac cycle. The condition can be corrected

there will be some stunting in growth, decreased exercise tolerance and a varying degree of listlessness.

Other calves will remain recumbent at birth and die soon afterwards. In the uncomplicated case there is no

Other calves will remain recumbent at birth and die soon afterwards. In the uncomplicated case there is no

Aortic stenosis

cyanosis, but on auscultation all animals have a systolic

This is very uncommon and is just below or at the aortic

murmur that is very obvious and can be heard on both

semilunar valves' attachments. Some animals show

swelling over a wide area of the chest. At necropsy there

few signs, others show dyspnoea. There is a systolic

is an interventricular defect and this is often just ventral

murmur. Death can occur suddenly with respiratory

distress. In some cases there may be an enlarged

liver.

liver.

Multiple cardiac lesions

Persistence of the right-sided aortic arch

There are many of these but all tend to be uncommon.

This is rare, but when it occurs the oesophagus is

In most cases the animal is born dead, weak or stunted.

encircled by blood vessels, causing constriction. There

Often other congenital defects are also exhibited.

is usually regurgitation of milk after feeding and this

Tetralogy of Fallot is probably the most common and

normally starts at birth or soon afterwards.

involves a ventricular septal defect with pulmonary

stenosis, a dextroposed aorta and a secondary ventric-

Persistent truncus arteriosus

ular hypertrophy. Eisenmenger's syndrome is relatively

similar to tetralogy of Fallot with a ventricular septal

This condition occurs very rarely.

defect, a dextroposed aorta but there is no pulmonary

stenosis. Other multiple cardiac lesions include a

double aortic arch and a double outlet to the right

Abnormal origin of the carotid arteries

ventricle.

This may affect one or both arteries, which may derive

The affected animals usually die. Those that survive

from the pulmonary artery instead of the aorta. This

show a very poor growth rate, severe dyspnoea when

results in weakness of the myocardium in the ven-exercised, and lassitude. Cyanosis is present in many tricle of the affected side due to anoxia, and leads to cases, particularly after exercise, although in Eisen-congestive heart failure.

menger's syndrome, cyanosis may not develop until late. Auscultation of the heart will reveal a murmur.

Aortic coarctation

Patent foramen ovale

This is a constriction at the site of entry of the ductus

This normally takes about seven to ten days to close arteriosus and results in a systolic murmur and poor completely in the normal calf. Patency is relatively pulse.

common. In many cases there is little blood transport, but if it does occur it is usually from left to right and so

Cardiomyopathy

there is normally no cyanosis. Hypertrophy of the right ventricle may sometimes arise. There are usually no

A condition often with polydipsia, hyperpnoea and signs unless other defects are present. Cyanosis is

*dyspnoea for one to seven days before death has
absent unless there is subsequent right ventricular
been described in Australian Poll Hereford calves
hypertrophy.*

*with a tight curly hair coat. At post-mortem examina-
tion there is vascular congestion of the liver, spleen
and lung with diffuse streaking of the entire*

Patent ductus arteriosus

*myocardium. It appears to be a genetic condition,
Although patent during intra-uterine life, the ductus
possibly associated with a simple autosomal recessive
arteriosus closes within a day of birth and at least by
mode of inheritance.*

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Blood disorders

*then it should not be repeated and bulls should be used
which do not carry the BLAD gene.*

Factor XI deficiency

Simmental hereditary thrombopathy

*The main congenital coagulation deficiency reported in
cattle involves factor XI (plasma thromboplastin*

A bleeding disorder resulting from impaired aggregation of platelets has been diagnosed in Simmental-sired recently in Britain. It has been shown to be present in some Holstein–Friesian breed lines and is transmitted although later it causes a prolonged bleeding time after as an autosomal recessive trait. Factor XI protein is concerned at an early stage of the contact or intrinsic activation pathway of blood coagulation. This pathway recorded in Canada, where it is recorded sporadically converges with the extrinsic one due to tissue damage, in the cattle population. The data available suggest that resulting in the activation of factor X. Following activation the factor converts prothrombin to thrombin, two genes rather than a simple Mendelian recessive. which in turn changes soluble fibrinogen to an insoluble fibrin clot. Bleeding problems can vary from minor

Skeletal defects

to profuse with haematuria and post-injection haemorrhage.

Achondroplastic calves (bulldog calves, chondrodystrophia fetalis)

Although it has been associated with the Dexter breed,

Bovine leucocyte adhesion deficiency

it can also occur in the Friesian, Hereford, Jersey and (BLAD)

Guernsey. It is basically a defect of interstitial growth.

This is a genetic problem caused by an autosomal recessive

The condition is mainly a recessive gene except that it

sive gene type of inheritance in Holstein cattle which

is dominant in the Jersey. When Dexters are mated

came into prominence in the 1990s. It is characterized

together 25 per cent of the offspring are bulldogs, 50 per

by a deficiency in the Mac-1 (CD11b/CD18) cell surface

cent are Dexters and 25 per cent are Kerry-type

receptor of the integrin family. The lack of adhesion

Dexters with long legs. Most calves are aborted at about

results in an inability to combat disease effectively.

seven months' gestation. The calves usually have very
The problem is seen in certain genetic lines of
short limbs, flattened skulls with a foreshortened face
Holstein bulls, some of which have been very widely
and short nose. There are often abdominal hernias and
used by AI for their increased milk production
anasarca. In many animals there is hydrocephalus due
characteristics.

to the deformed cranium.

Animals involved are not easy to detect initially as
they are more susceptible to infections and so suffer

Complex vertebral malformation (CVM)

more enteric and respiratory problems and growth

A problem recently discovered by Danish scientists. It
rates are poor. There is usually oral ulceration, which is
involves the Holstein breed and appears to have a
recurrent, and on post-mortem examination some have
genetic basis, possibly with a recessive-type inheritance
peritonitis. Most animals die between two and about
pattern. It appears to have been carried by some bulls
seven months old, although some have survived longer.

widely used for AI across the world. The problem is Many of the signs are similar to BVD infection, BIV, of malformed calves which appear visually to have a mineral deficiencies and parasitic problems including foreshortened neck and thorax; the spinal cord may be coccidiosis, cryptosporidiosis and parasitic gastroenteritis. Diagnosis is often difficult, but if BVD is eliminated appears that there can also be an increase in abortions, and the animal has a marked leucocytosis it is likely to heart and lung abnormalities and low birthweight.

be the cause. This can be confirmed by laboratory examination of blood to show the presence of the genetic known carrier bulls or other bulls descended from the defect and looking at the pedigree to determine the same bloodlines.

presence of certain affected bulls. Many reputable cattle semen companies will indicate the presence of

Congenital joint laxity and dwarfism

the BLAD factor within their bulls. Treatment has been

syndrome (CJLD)

successfully undertaken by bone marrow transplant but

is not recommended as it is helping to perpetuate a

The condition is also known as long bone

genetic defect. If a mating does produce an affected calf

deformity/chondrodystrophy syndrome and in Canada

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‘acorn calf syndrome’, as it occurs at a time when

Mandible and face abnormalities

acorns are around. The condition is seen mainly in the

The terms ‘overshot’ and ‘undershot’ are often used for

calves of suckler cows and very occasionally dairy

these conditions, but they have variable definitions.

heifers. The calves are mainly born in the spring from

Abnormal length of the upper and lower jaws is better

dams which have usually been fed only pit or clamp

termed superior or inferior prognathia and shortening

silage or rarely big bale silage with no other feeds.

of the upper or lower jaw is superior or inferior

Various causes have been suggested but it appears to

brachygnathia respectively. Most newborn calves show

be nutritional in origin, directly or indirectly, as the a degree of inferior prognathia, but this condition addition of another feed such as straw, hay, cereals, resolves. However, persistent inferior prognathia is etc. to the diet usually prevents or greatly reduces its more common than inferior brachygnathia (parrot occurrence.

mouth). The conditions are thought to be inherited. There are various forms of the condition which have Problems can arise from impaction or non-apposition been grouped together and may be variations on the of the molar teeth. Extreme hypoplasia or agnathia same problem or different problems. In the dwarfism are rare. Lateral deviation of the face with normal syndrome animals are born at full gestation but development of the mandible (campylognathia) is have long bone shortening. Other signs can include occasionally seen.

increased (laxity) or decreased joint movement (chondrodystrophy) and some of these can improve. The fetlocks are often involved. The head may be domed or

Vertebral column defects

the face dished, there may be superior brachygnathia.

The fore limbs may show lateral bowing or the hind can

Various abnormalities have been reported including

be sickle hocked. Severely affected calves are unable

spirabifida, lordosis (ventral deviation), kyphosis (dorsal

to rise and may have kyphosis. Post-mortem reveals

deviation) and scoliosis (lateral deviation). Occasion-

the skeletal abnormalities; there may also be hepatic

ally, there is partial or total agenesis of the posterior part fibrosis.

of the spinal column; screwtails are reported in Red Polls

The cause is unknown although silage feeding is crit-

and wrytails in Jerseys and Holsteins. Ankylosis of the

ical. There appears to be an interference in normal bone

intervertebral joints has been recorded.

metabolism: it may be a calcium/phosphorus imbalance,

or vitamin D, A or copper deficiency. Manganese

Defects of the limbs and claws

problems have been suggested but supplementation

does not improve the problem. There appears to be no

Occasionally, duplication of all or part of the limb

genetic link.

occurs and in other animals the whole or various bones of the limb are absent. One problem recently described is tibial hemimelia in Galloway cattle. Polydactyly or

Osteopetrosis (metaphyseal dysplasia)

extra digits have occasionally been seen. A quite frequent abnormality is the partial or complete fusion This has been recorded in black and red Aberdeen of the digits (syndactyly or mule foot). The condition is Angus and Hereford calves. It is thought to be due to reported in the Holstein, Aberdeen Angus, Hereford an autosomal recessive gene. The calf may be born and Chianina. It occurs more commonly in the front prematurely. At birth it is small and of low weight, than the hind legs and the right limbs are more often with brachygnathia inferior (shortened mandible), affected. It can be inherited as a simple autosomal protrusion of the tongue, impaction of the molar teeth, recessive trait. Duplication of the whole limb (poly-misshapen coronoid and condyloid processes, open melia) is very rare. It can be attached to the thigh of the

fontanelle, thickened cranial bones, shortening of the normal limb by soft tissue and a pseudoarthrosis may long bones and a lack of bone marrow. Radiographs develop between the femoral head and the pelvis. show the homogeneous bone shaft.

Osteoarthritis

Atlanto-occipital fusion

Although it can be nutritional in origin, it can also This is rare and is due to a failure of the first cervical be inherited in Jersey and Holstein Friesian cattle. vertebra to separate from the occipit and thereby form There are two main conditions, namely degenerative a joint. It need not necessarily be apparent at birth. The arthropathy, which mainly involves the hips, and degen- main signs are ataxia with inability to coordinate limb erative osteoarthritis, which primarily affects the stifle movements. There are then abnormal flexures of the joints. The latter condition develops in older cattle cervical region and recumbency.

over a period of one or two years. The stifle shows

crepitation and the limb is not raised much off the ground when walking. The articular cartilages show increased salivation.

degeneration.

Atresia ilei

Arachnomelia

This condition has occasionally been reported and there is disruption of patency. The signs are of a distended distal extremities like a spider and the bones are brittle. abdomen and this may lead to dystokia. Some cases are It has been recorded in the Simmental breed. Often due to a recessive gene.

there is spinal curvature and inferior brachygnathia, which often affects other body systems.

Atresia coli

Displaced cheek teeth

This has been recorded in Aberdeen Angus and other breeds. The calves survive only a few days.

The lower mandible tends to be shorter and narrower

than normal with abnormal eruption or impaction of the cheek teeth.

Atresia ani

This may be inherited and is seen in several breeds including the Friesian. The animal is usually born

Lymphatic system

bright, but will usually die within a week unless surgical relief is provided.

Inherited lymphatic obstruction

This has been reported in Ayrshire calves and is caused by the autosomal recessive condition. The lymph nodes

Muscular system defects

tend to be small and the lymphatic vessels are large and tortuous. The condition results in oedema, which varies

Congenital flexure of the pastern joints

from slight to severe. The calves may be born dead. The oedema may be so gross as to cause dystokia. Oedema

This is common and present in most breeds. In the can be of the head, neck, ears, tail and legs. In slight

Jersey it is considered to be caused by an autosomal cases there is oedema of the legs and these animals may

recessive gene. The calves show knuckling over on one survive. Accessory lobes may be present at the base of or both front fetlocks, and occasionally the hind limbs the ears.

are also affected. The condition is usually reversible and most calves recover within about six weeks. In some cases it may be necessary to splint the limbs. Manganese

Alimentary tract defects

deficiency in the dam can also lead to the condition as can locoweed or poison vetch (Astragalus and Oxytrionis spp).

Cleft palate (palatoschiasis)

This can occur as an individual condition but is normally associated with other conditions, particularly

Arthrogryposis ('curled calf disease')

arthrogryposis.

(see pp. 451, 925)

By definition this is a permanent joint contraction. The

Harelip

condition is normally bilaterally symmetrical and the forelimbs are affected more than the hind limbs.

This has been recorded in cattle but its mode of inher-

The muscles show marked atrophy, they are pale

itance is not known. Occasionally, ingestion of lupin

in colour and there is replacement of many muscle

*(*Lupinus sericeus*) can result in the condition.*

fibres with fat. Cleft palate is often also present.

The condition is common in the Charolais breed where

it is a recessive gene. The gene is probably more

Smooth tongue (epitheliogenesis

prevalent than it should be because carrier dams

imperfecta linguae bovis)

appear to have advantages in improved longevity and

This is seen in the Holstein–Friesian and Brown Swiss.

fertility. Infection of the fetal calf by akabane virus can

It is the result of an autosomal recessive gene and leads

*result in the condition, as can lupin (*Lupinus sericeus*) to the filiform lingual papillae being small. The animals*

ingestion.

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Muscular hypertrophy (double muscling,

the Holstein and Hereford and is in-herited. Infection

muscular hyperplasia, culard)

of the fetus with akabane virus can produce the problem and it is thought vitamin A deficiency can contribute to the condition. This is a characteristic with some production potential in that some muscles have increased numbers of muscle fibres. It can result from obstruction to the drainage of cerebrospinal fluid from the ventricles or cranial malformation. In both conditions the Limousin, Charolais and Belgian Blue and the degree animals are born dead or die in a few days. There are of skeletal muscle involvement varies. It occurs most usually ocular defects.

commonly in the hind limbs with a rounding of the hindquarters. The muscles affected have deep grooves along the intermuscular septa and this may be seen in

Cerebellar hypoplasia

the muscles of the shoulder, back, rump and hindquarters. This is also known as Hereford disease, but is seen in others. Many of the animals tend to stand in a stretched posture. The Holstein and Shorthorn and appears to be genetic.

position. The calves tend to be less viable at birth and During pregnancy, infection with BVD or Neospora can because of the increased muscle size dystokia is produce the condition (see p. 900). The cerebellum common. In the Belgian Blue dystokia results partly tends to be small, tough and leathery or even absent. from a narrowing of the dam's pelvis.

Most calves are obviously affected at birth; there is swaying of the neck, with inability to stand and blind-

Multiple tendon contracture

ness occurring in severely affected animals. Less badly affected calves have exaggerated and incoordinated This has been recorded in Shorthorn cattle and results limb movements. The animals are conscious and able to in dystokia due to the calf's limbs being fixed in drink. Some will survive for several months.

extension or flexion. There is a lack of mobility of the limbs and often positioning is abnormal. The problem involves the tendons and there is limb muscle atrophy.

Inherited cerebellar ataxia (see p. 893)

The calves are born dead or are destroyed because they

This condition is described as being due to a single autosomal recessive gene. It is seen in the Holstein, Jersey and Shorthorn and the signs are similar to cerebellar hypoplasia, although they are not apparent at birth. The

gross lesions are minimal and consist of a wet glistening appearance to the cerebellar white matter, which

Joint hypermobility

The cause of the condition is unknown but in Jersey calves it is a single autosomal recessive gene. The joints

are very mobile and can be bent into very abnormal positions with overextension and flexion of all or most

Cerebellar abiotrophy (premature ageing)

of the upper fore and hind limb joints.

This condition is seen in Hereford and Simmental calves and appears when four to eight months old.

Achondroplastic deviation

There is the sudden onset of ataxia, which then progresses slowly. The animals are not blind. Calves remain

Most cases appear to be inherited as a single recessive strong but become recumbent or decline slowly into characteristic and involve the Aberdeen Angus and a spastic ataxia. Histologically, there is ageing or Hereford breeds but cases have been reported in the degeneration of the cerebellar neurones (see p. 893). Holstein and Shorthorn. The calves have short legs, a wide, short head and the mandible protrudes far in front of the dental pad. The eyes bulge and the tongue

Congenital spasms

protrudes. Breathing is stertorous with the forehead The condition has only been reported in the Jersey and protruding and the maxilla distorted.

there is a continual tremor of the head, neck and limbs. The animal cannot walk and it may die in a few weeks.

Nervous system defects (see Chapter 51)

Familial ataxia and convulsions (see p. 893)

Hydrocephalus

These have been reported in Aberdeen Angus calves This is uncommon in calves and can be inherited or and appear to be an incomplete dominance. The signs

congenital. It is often associated with other deformities. are seen within a few hours of birth but can occur when e.g. congenital achondroplasia. The condition is seen in two or three months old with the sudden onset of

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tetanic spasms, which last for three to twelve hours. In an inability to bellow. Histological examination reveals mild cases there is a stiff, exaggerated movement but in accumulations of neurofilaments within the neurones of the severe form there may be convulsions with recumbency, opisthotonus and paddling of the forelimbs. Following the initial signs there is a residual ataxia with a Holstein male calves and is probably caused by a goose-stepping action, which lasts weeks or months. sex-linked recessive mutation. On histology, there Necropsy shows lesions of degeneration of the cerebellar spinal cord changes with spongiform lesions with lar cortex Purkinje cells. Diagnosis depends on age, cavitation.

signs and their remission.

Idiopathic epilepsy

Progressive ataxia (see p. 894)

This condition occurs when the calves are a few months

This has been recorded in the Charolais in Britain and old and mainly involves the Brown Swiss. It is inherited subsequently in France. The signs do not develop until as a dominant characteristic. The convulsions are the animal is about a year old and they are seen as a epileptiform and are seen when the calf is stimulated.

progressive ataxia. The animal has increasing difficulty

They disappear once the animal is one to two years old.

in rising until it may become permanently recumbent.

Histologically, there is a myelin degeneration of the

Lysosomal storage diseases (see p. 892)

white matter of the cerebellum and internal capsule.

There is a generalized glycogen storage problem in beef

Shorthorns with muscle weakness, incoordination of

Bovine progressive degenerative

gait and eventual recumbency. In the Friesian there is

myeloencephalopathy

a GM1 gangliosidosis where there is an accumulation of
(Weaver syndrome)

ganglioside (GM1) in the nervous tissue due to reduced activity of the enzyme β -galactosidase. At about three months old the animal begins to grow more slowly, is high milk production and occurs in the Brown Swiss blind and has a staring coat.

breed, although a similar condition has been described in the Murray Grey. Signs usually develop between five months and two years, with the occasional one starting

Mannosidosis

after that time. The problem is one of a progressive
The condition has been recorded in Aberdeen Angus (it often stretches over one to one and a half years)
and Murray Grey cattle in New Zealand, Australia and bilateral hind limb weakness with the animal showing recently in Britain (see p. 892). It is inherited as an autosomal recessive trait and is a deficiency of a specific ataxia and dysmetria. Thus the animal gradually has

lysosomal hydrolase enzyme, α -mannosidase, and this increasing difficulty in rising, a weaving action and often causes the accumulation of mannose and glucosamine goose stepping of the fore limbs while starting to drag in secondary lysosomes. The signs develop from one to its hind legs. Although there are proprioceptive deficits, fifteen months old and most animals die by one year.

limb reflexes are normal. The animal is bright and alert

There is, at first, slight hind leg ataxia, then a fine lateral throughout the condition. At post-mortem examination

head tremor, slow vertical head nodding, aggression and

there are lesions in the white matter of the cerebellum

loss of condition. Diagnosis is based on reduced tissue

and at all levels within the spinal cord. The lesions

and plasma levels of α -mannosidase. Histologically,

include axonal degeneration and vacuolation of the

accumulations of mannose and glucosamine are seen

white matter due to large intercellular spaces and

in the nerve cells, fixed macrophages and epithelial

axonal spheroidal degeneration.

There may be

cells of the viscera. Tissue and plasma levels of α -muscular wasting but without muscular dystrophy. mannosidase are about half the normal level in heterozygous animals and can thus be detected.

Inherited neurodegenerative disease

Spastic paresis (see p. 458)

(shaker calf syndrome)

There is an extension of the stifle and tarsal joints of This syndrome, which has been described in Canadian one or both hind limbs. The condition is seen in the horned Hereford cattle, is evidenced by a severe muscular tremor and shaking of the head, body and tail. The Friesian, but other breeds can be affected. Signs are usually present until the animal is several weeks or animal has difficulty in rising, a wobbly spastic gait and months old, when they start to progress. Contraction

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of the Achilles tendon, gastrocnemius and superficial recumbent, often in lateral recumbency and some are flexor tendons overstraighten the hock joint, so that the

unable to move their head. There is extension and
os calcis is moved cranially towards the tibia. Usually,
crossing of the hind limbs with hypersensitivity to noise
one leg is more affected than the other and this limb
and touch. Often when animals are encouraged to stand
may appear shorter. In the later stages of the severe
there are myoclonic spasms with the body becoming
cases, the leg may swing backwards and forwards like a
rigid. At necropsy there is usually damage to the hip
pendulum. The condition is considered to be inherited.
joints, probably secondary to myoclonic contractions.
However, the genetic influence is considered to be
There is no oedema of the central nervous system. The
small, and it is thought that the phenotypic expression
main differential diagnosis is maple syrup urine disease.
as a probably multifactorial recessive genotype depends
No treatment is possible and the same mating pattern
on mostly unknown environmental factors. Where only
should not be used again.
small numbers of an AI bull's offspring are affected and
the animal is of high genetic merit, it has been sug-

Maple syrup urine disease

gested that it should still be used as a sire (see p. 166).

(branched-chain ketoacid

Surgery can relieve the condition but the animals

decarboxylase deficiency, BCKAD)

should not be used for breeding. Recently, analysis of

cerebrospinal fluid concentrations of homovanillic acid,

This condition is possibly inherited as an autosomal

the main metabolite of dopamine, has shown levels to

recessive gene. It is very uncommon but may be seen in

be lower in spastic paresis calves than normal contem-

polled Hereford calves (see p. 893). There are higher

poraries. The possibility of a disorder in dopamine

than normal concentrations of branched-chain amino

metabolism has therefore been suggested as a possible

acids in plasma and/or serum, urine, cerebrospinal fluid

cause of the condition.

and formalin-fixed cerebral tissue. It has been sug-

gested that the condition is analogous to branched-

chain ketoacid decarboxylase deficiency or maple syrup

Neonatal spasticity

urine disease. There is dullness, opisthotonus and
This condition involves a single recessive characteristic
recumbency and a poor response to touch or auditory
in the Jersey and Hereford. The animal is born normal
stimuli. At post mortem there is severe stratus spon-
but within the first week it develops convulsions of the
giosus. The main differential diagnosis is inherited con-
head, neck and limbs, preceded by neck deviation and
genital myoclonus.
bulging eyes.

Congenital pastern paralysis

Periodic spasticity

The defect is lethal due to prolonged recumbency and
in Red Danish cattle at birth can take the form of
This has been recorded in the Guernsey and Holstein
opisthotonus–muscle tremor with spastic extension of
breeds. It appears to be a single recessive character with
the limbs and exaggerated tendon reflexes. There is
incomplete penetrance and is often not noticed until
neuronal degeneration in many parts of the brain and
the animals are adult. Early signs involve the hind end,

spinal cord. In the Norwegian Red Poll a similar condition is seen but only involving opisthotonus and backwards and the back depressed. The back muscle tremors.

may fasciculate and the condition progresses from a few seconds duration to last up to 30 minutes. The animal cannot walk during the attack.

Perosomus elumbis

This occurs very occasionally in ruminants. There is aplasia or hypoplasia of the spinal cord caudal to the

Inherited congenital myoclonus

thoracic area. This results in rigidity of the hind limbs, The condition does not involve oedema of the central there is muscle atrophy and no joint movement. Most nervous system and is therefore described as inherited cases are born dead.

congenital myoclonus (see p. 893). It is inherited as an autosomal recessive gene and is seen in America, New Zealand and Australia to affect Hereford and polled

Ocular defects (see p. 914 onwards)

Hereford-cross calves. It has also been reported in Britain in the Hereford, Jersey and South Devon. Reports of ocular defects in cattle are few. Anophthalmia and microphthalmia occur infrequently. Animals are usually produced after a shorter than normal gestation period. They are bright and alert but Entropion is also very rare. Dermoids can occur on the

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eyelids, conjunctiva and cornea, but they are common-

Persistent hyaloid vessels

est on the third eyelid.

These are quite common and are the vestige of the earlier development of the eye. They have no practical significance.

Exophthalmus with strabismus (see p. 918)

This has been recorded in the Hereford and Holstein is combined with strabismus in Shorthorns and their

Skin defects

crosses in Britain and Jerseys in America. The signs, particularly in the Shorthorn, are usually delayed until

Symmetrical alopecia

a year old, although occasionally young calves are
This is apparently inherited as a single autosomal recessive
affected. The condition is progressive and defective
sive characteristic in Holstein cattle. It involves animals
vision is observed first, followed by protrusion and deviation
born with a normal hair coat but which is then lost in a
ation of both eyeballs medially with difficulty in focus-
symmetrical pattern over the body. It occurs between
ing. The condition is considered to be inherited as a
six weeks and six months of age and affects both
recessive gene, but some occur in cases of cerebellar
pigmented and non-pigmented areas.

hypoplasia or mucosal disease.

Congenital hypotrichosis

Colobomata (see pp. 923, 924)

Several forms are recorded that vary both in inheritance
These problems appear to have a high prevalence in the
ance and degree. There may be partial or complete loss
Charolais. There is an absence of part of one or more
of hair and the condition is present at birth. In some
of the structures of the eye. The condition occurs during

instances the animals will grow satisfactorily provided early gestation, when the eye is developing. Although there is sufficient shelter.

always bilateral, it may not be symmetrical and it is usually found associated with the optic disc and the

Epitheliogenesis imperfecta

tapetum nigrum below the disc. The retina is involved and in some animals the choroid and sclera are also

This condition can occur in either sex and reports affected. The condition is present at birth and does not include Holstein, Ayrshire and Jersey calves. Most progress. The mode of inheritance has been debated, animals die within a few days of birth. It is considered but a dominant gene with incomplete penetrance, auto-somal recessive or polygenic inheritance have all been normally areas of varying size devoid of skin or mucous suggested. Signs are not usually apparent although an membrane. The defects are often distal to the tarsal and ophthalmoscopic examination will reveal the lesion. carpal joints. Lesions may also occur on the muzzle,

The very severely affected animal can be blind, and a tongue, hard palate, cheeks and nostrils.

few others are considered to be hyperexcitable due to the defective vision.

Keratogenesis imperfecta (baldy calves)

The condition appears a few months after birth and is

Congenital cataract

lethal. It has been observed in the Friesian and is due (see p. 923)

to an autosomal recessive gene. The skin tends to
Lens opacity is present from birth. The condition has
develop alopecia, there is a loss of body condition and
been recorded in the Friesian, Hereford, Jersey and
the horns do not grow. The skin then becomes scaly,
Shorthorn. Some cases in the Holstein and Jersey are
thickened and folded, particularly on the neck and
considered to be due to an autosomal recessive gene or
shoulders. There is alopecia and raw areas may develop,
infections. A form of nuclear cataract in Friesian and
particularly on the knees, hocks, elbows, axillae and
Friesian-cross calves appears to be more common in

flanks. The joints tend to be stiff and there is
calves born in the summer months and is considered to
overgrowth of the hooves.

be of environmental origin. The condition is bilateral
and not progressive. In most animals the degree of

Inherited parakeratosis (lethal trait A46)

involvement of each eye is similar. In severe cases
blindness is apparent. It is not always possible to

This is possibly an autosomal recessive condition. It is
examine the fundus of the eye, but in most affected
seen in Friesian cattle and is thought to be due to an
calves there is no abnormality of the retina or optic disc
increased zinc requirement. The signs are usually seen
(see p. 923).

about four to eight months after birth with alopecia and

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parakeratosis of the limbs, muzzle and under the jaw.

umbilical herniae are missed unless a conscious effort

The animal becomes stunted and, if untreated, it dies in

is made to look for the defect. This is due to the hernia

about four months. At necropsy there is thickened skin

occurring just anterior to the prepucial orifice. If the with thick crusts over the skin lesions. The spleen shows hernia is small it may not need to be treated. Larger hypoplasia. Diagnosis depends on low serum zinc levels hernias may need surgical repair (see p. 1122) by suturing across the hole or surgical webbing may need to be parakeratosis. Therapy must continue for the rest of the introduced. Many cases are inherited and the animal animal's life and as a calf about 0.5 g zinc oxide or 1 g should be recorded as often the hernia is hard to detect zinc sulphate daily is required. The dose should be in the adult. Do not breed from affected cattle. increased as the animal grows older (see p. 260).

Inguinal herniae

Interdigital hyperplasia (see p. 429)

These occur far less commonly than umbilical herniae. This condition is particularly seen in the Hereford and Little is known about aetiology. it is considered to have a genetic predisposition. The condition tends to be present in the older animal and

Scrotal herniae

can be surgically removed, but has a tendency to recur.

Like inguinal herniae, these are rarely seen but can occur in the Sussex and Friesian breeds. Little is known

Albinism

about the aetiology, but there is a familial trait.

Varying types occur. In partial albinism the coat colour is normal for the breed or a dilute colour, but the iris is

Schistosoma reflexus

blue and white centrally, with a brown border. Incomplete albinism is characterized by a white or mainly This is a group of conditions in which there is longitudinal fissure in the body wall. The cause is unknown, but condition is inherited by an autosomal dominant gene.

it may be due to the failure of the somatopleure of the

In complete albinism the coat is pure white and the iris blastodermic vesicle to close. At birth the vertebral white or pink. The condition is inherited as a simple column is angulated with the head and tail showing autosomal recessive trait.

approximation dorsally. The abdominal and thoracic organs lie free in the dam's uterine cavity.

Familial acantholysis

Diaphragm defects

This has been recorded in Aberdeen Angus calves.

There is a loss of skin at the carpal and metacarpal

These occur occasionally and, depending on size and joints and coronet, where there is horn separation. The position, they may or may not involve abdominal organ defect is one of defective collagen in the basal and herniation.

prickle layers.

Reproductive system defects

Congenital ichthyosis

This is also known as fish scale disease in that there is

Testicular hypoplasia (see p. 618)

alopecia and the presence of a horny epidermis.

This occurs sporadically in all breeds of bull. The problem can be bilateral or unilateral as well as being partial or complete. The left side is more commonly

Body cavity defects

affected. Work on gonadal hypoplasia in Swedish Highland cattle showed an inherited origin and this is **Umbilical herniae (navel ruptures)** probably also the case in British breeds.

These are found with a low frequency in several breeds, but especially the Friesian and Holstein. It would

Cryptorchidism (see p. 482)

appear in some cases to be due to the environment, following infection, to a dominant gene with incomplete

There is incomplete descent of the testicles into the scrotum and this may be unilateral or bilateral, of the progeny of Holstein bulls, more cases occurred although the former is more common. Bilateral cryptorchidism usually produces a sterile animal. The

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condition occurs in most breeds including the Friesian and Hereford. Although studies of aetiology are few, it usually first stage labour is minimal, necessitating a Caesarean section. The calves tend to be heavy, have

is considered to have an inherited basis.

well-erupted teeth and a good coat growth. The adrenals of the calves are hypoplastic and following delivery most are weak and die in hypoglycaemic crisis. The

Wolffian duct aplasia

condition is the result of an autosomal recessive gene.

This is normally seen in the area of the epididymal

(2)

Prolonged gestation with adrenohypophyseal head.

hypoplasia. This is due to a recessive gene and it is mainly recorded in the Channel Island breeds. The gestation length is increased by weeks or months and par-

Ovarian aplasia

turition occurs about seven to fourteen days after the

This occurs occasionally with or without other

calf's death. There is again no udder development and reproductive abnormalities.

few signs of parturition. The calf in this case is small and ceases to grow after the seventh month of gestation, so it can often be delivered by manual traction. It may

Ovarian hypoplasia

show disproportionate dwarfism, craniofacial defects

As with testicular hypoplasia, this has mainly been that may cause hydrocephalus, alopecia and abdominal distension. There is no or only partial development of inherited.

the adenohypophysis.

It should be remembered that the gestation period of some of the larger beef breeds such as the Charolais,

Müllerian duct aplasia

Simmental and Limousin is longer than for the Friesian

Various forms can occur and all are uncommon, but the or most British beef breeds.

main form is uterus unicorni.

Hermaphroditism

Duplication of the reproductive tracts

Both true and pseudohermaphrodites occur. In the true

This can occur as a deficient union or exaggerated form there are gonads of both sexes, although they may union of the Müllerian ducts. They can result in a partial

be combined into an ovo-testis. In the pseudohermaphrodite, complete duplication of the cervix or uterine body, cattle are genetically female with female or vaginal septa.

gonads but they have partial masculinization of the external genitalia. The animals usually exhibit normal oestrous behaviour and on investigation have normal

White heifer disease

ovaries. However, the vulvar orifice is usually small and The condition used to be common in the white Short-displaced ventrally.

horn with up to 10 per cent of them being affected. It is now rare, but can be found in other breeds. There are

Freemartinism

varying degrees of involvement. In all cases there is partial or complete persistence of the hymen. This may This is seen in twins where a female is developing with be the only abnormality, but in other animals there are a male and this affects about 11 out of 12 such twins. abnormalities cranial to the hymen. These defects may The calf contains both normal 60 XX chromosomes and

include the absence of the cranial vaginal cervix, uterine a few 60 XY (male) chromosomes. Occasionally, a body or horns. The ovaries are functional and in most female calf will be born singly with this abnormality and cases there is a distension of the normal organs due to this is due to the death of the other twin. Freemartins the accumulation of the products of secretion. are usually sterile.

The external genitalia resemble a female, but the vulva is smaller than normal. Later a tuft of hair devel-

Prolonged gestation

ops at the vulva and in many cases an over-sized This has been recorded in most dairy breeds and in clitoris is found. Internally, there is a varying amount some cases it has an inherited origin. There are two of agenesis or hypoplasia of the Müllerian system and main forms of the condition.

stimulation of the Wolffian system. The ovaries tend to (1)

Prolonged gestation with fetal giantism. In these be hypoplastic and the vagina is usually non-patent,

animals the fetus continues to grow in utero before par-which allows diagnosis of the condition in the calf by

turition 21 to 100 days late. The cow usually calves with the passage of a probe. However, occasionally the no udder development or ligament relaxation and vagina is tubular and terminates at the normal position

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of the cervix, which is absent. The uterus tends to be

Congenital goitre

two thick cords and there are two thin ducts that extend

This is an inherited condition but animals can be kept from the gonads to the intrapelvic urethra. There are alive although mortality is high. Goitre also occurs often seminal vesicles present and sometimes testicles. when iodine deficiency affects the gestating dam (see pp. 257, 301)

Urinary system defects

Anomalous twins

Pervious urachus

There is faulty division in monozygotic twinning and

The defect occurs occasionally in calves. However, it

this results in various abnormalities. There are varying degrees of conjunction, but double-headed monsters are most often seen. Others have a single head and the urachus. All urine may be passed via the urachus posterior part of the body is divided. In some cases although in some cases passage is also via the urethra. The separation of the vertebral column may be almost complete, resulting in Siamese twins. Occasionally, does not heal, there is often omphalophlebitis, septi-small amorphous monsters called amorphous globosus caemia and polyarthrititis. Surgical correction of the or acardiac twins occur. They are related to double condition is possible and can be successful. monsters and identical twins. They are found attached to the fetal membranes of the other calves and comprise an outer skin enclosing adipose tissue.

Other defects

Congenital porphyria

Chromosomal translocations

There are always 60 chromosomes present in the

This is very rare. It is mainly seen in the Friesian and

normal bovine. Translocation is the fusion of two mor-

Holstein. It is caused by a simple recessive gene and

phologically distinct chromosomes. The most common

occurs more frequently in the female than the male.

is 1/29 translocation where there is fusion between

There is increased porphyria in the blood and urine

number 1 and 29 pairs; it is also referred to as the

leading to the accumulation of the product in the

Robertsonian translocation. It has been recorded in the

tissues. The teeth tend to be pink or brown in colour

Swedish Red and White breed, Charolais, Red Poll,

and this can be seen in the newborn. The urine is red

British White and other breeds. The condition appears

or purple and animals will develop cutaneous photo-

to be of importance in that there is reduced fertility in

sensitization and anaemia. There are high levels of

such animals due to early embryonic death.

uroporphyrins and coproporphyrins in the blood.

Familial polycythaemia

Further reading

The condition is seen in the Jersey and is inherited as a

Blowey, R.W. & Weaver, A.D. (2003) Congenital disorders. In simple autosomal recessive trait. There are early deaths.

Colour Atlas of Diseases and Disorders in Cattle, 2nd edn.

Those alive have poor growth, congestion of the

Mosby, Edinburgh, pp. 1–9.

mucosae and dyspnoea. There is a reduced ery-

throcyte count, packed cell volume and haemoglobin

concentration.

Chapter 14

Calf Diarrhoea

P.R. Scott, G.A. Hall, P.W. Jones and J.H. Morgan

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permits a logical approach to disease control. Appro-

Causative mechanisms in diarrhoea

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priate advice on nutrition, colostrum feeding, vaccina-

Altered ion transport

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tion, hygiene and the use of antibiotics can only be

Passive malabsorption

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given if it is clear which infectious agents are present

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and what their contribution to the disease process

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might be.

Tissue hydrostatic pressure and increased permeability

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The digestive tract may be regarded as a high fluid flow

Bovine rotavirus

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system in which 80 per cent of the fluid contained within

Bovine coronavirus

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it is secreted into it and 20 per cent is ingested. Secreted

Calici-like virus (Newbury agent)

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fluid originates from salivary glands, gastric mucosa,

Astrovirus

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pancreas, liver and small and large intestinal mucosa.

Breda virus

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Of the water that enters the digestive tract, 95 per cent

Escherichia coli

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is absorbed. Diarrhoea may be defined as an increase

Campylobacter spp .

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in faecal water loss due to increased faecal water

Cryptosporidium parvum

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content or to increased volume of faeces excreted or to

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a combination of both. The occurrence of diarrhoea

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indicates an imbalance between absorption and secre-

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tion of water and electrolytes. Only a slight imbalance

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in the equilibrium between secretion and absorption,

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in favour of secretion, may lead to severe diarrhoea

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because very large volumes of fluid are fluxing in

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both directions. There are several possible causes of

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imbalance.

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Diarrhoea due to altered ion transport is caused by reduced absorption of sodium ions by villous entero-

Introduction

cytes (Fig. 14.2), by increased secretion of chloride ions by crypt cells (Fig. 14.3) or by both mechanisms acting

Diarrhoea in the neonatal calf is a serious welfare problem and a cause of economic loss due to mortality, by bacterial enterotoxins produced by enterotoxigenic treatment costs and poor growth. Calf diarrhoea is an

Escherichia coli (Fig. 14.4). These bacteria, held on the example of a complex or multifactorial disease, result-gut surface by fimbrial adhesins, release enterotoxins.

ing as it does from an interaction between the calf, its

The subunit A of the heat-labile toxin activates adeny- environment and nutrition and infectious agents (Fig.

late cyclase located on the basolateral membrane

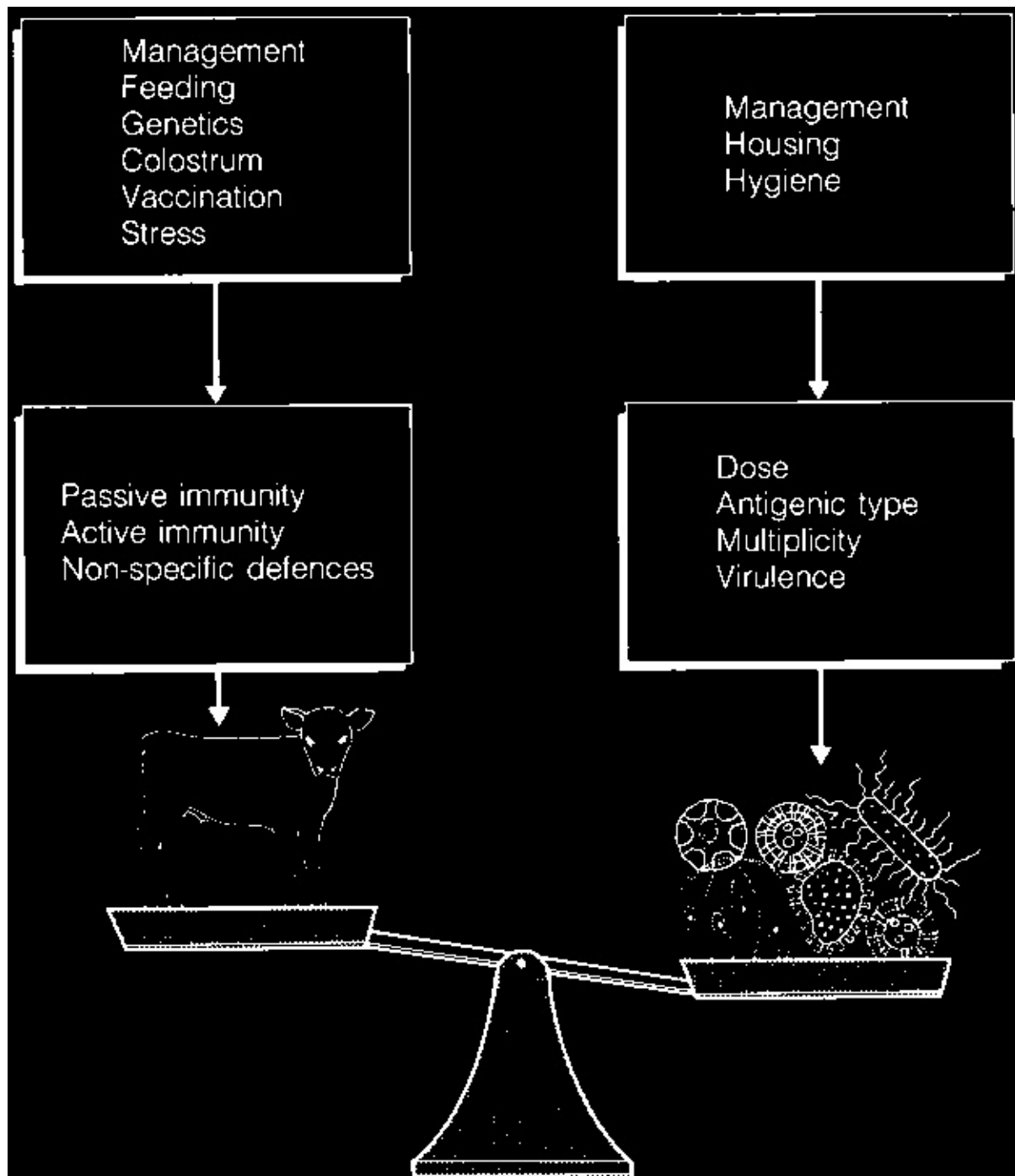
14.1). Successful control of an outbreak will depend on

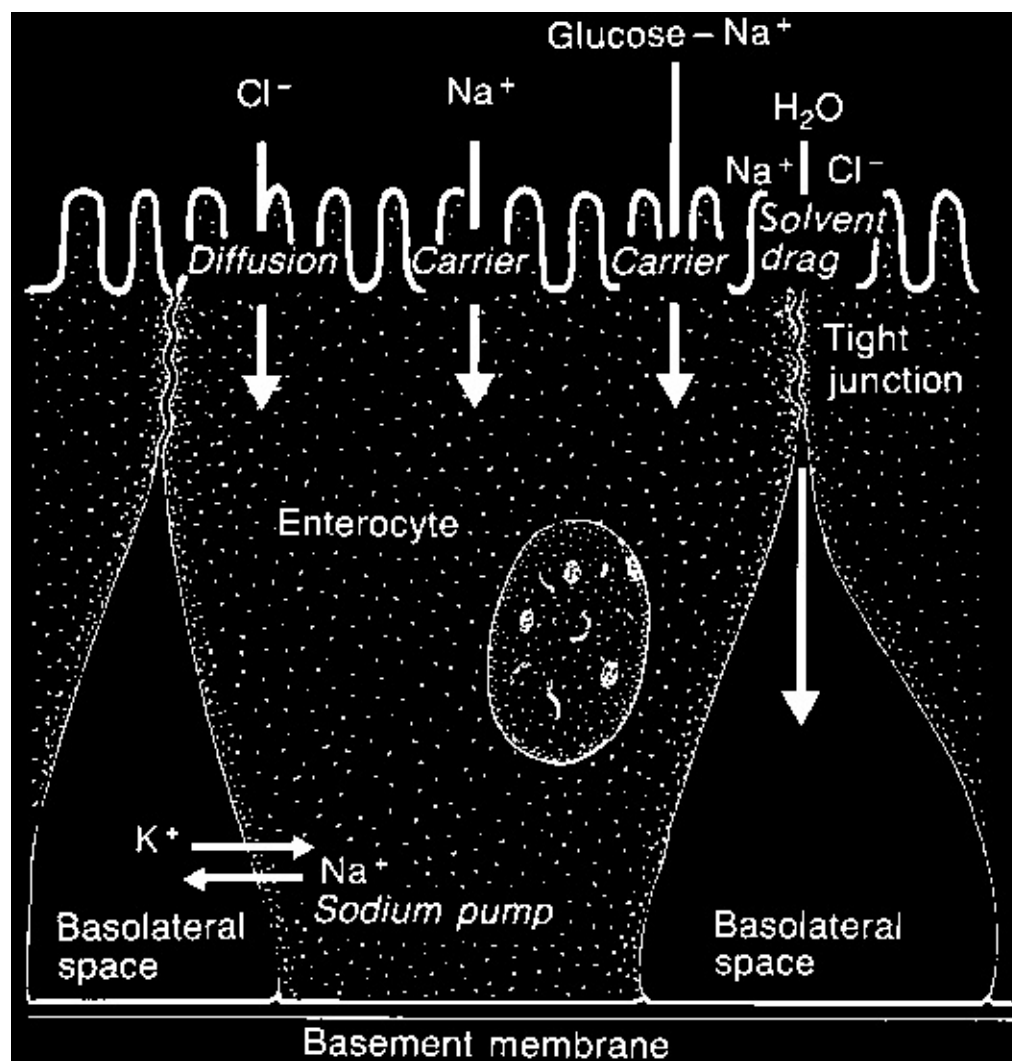
which, in turn, raises the production of intracellular recognition of the important factors in that outbreak cyclic adenosine monophosphate (cyclic-AMP).

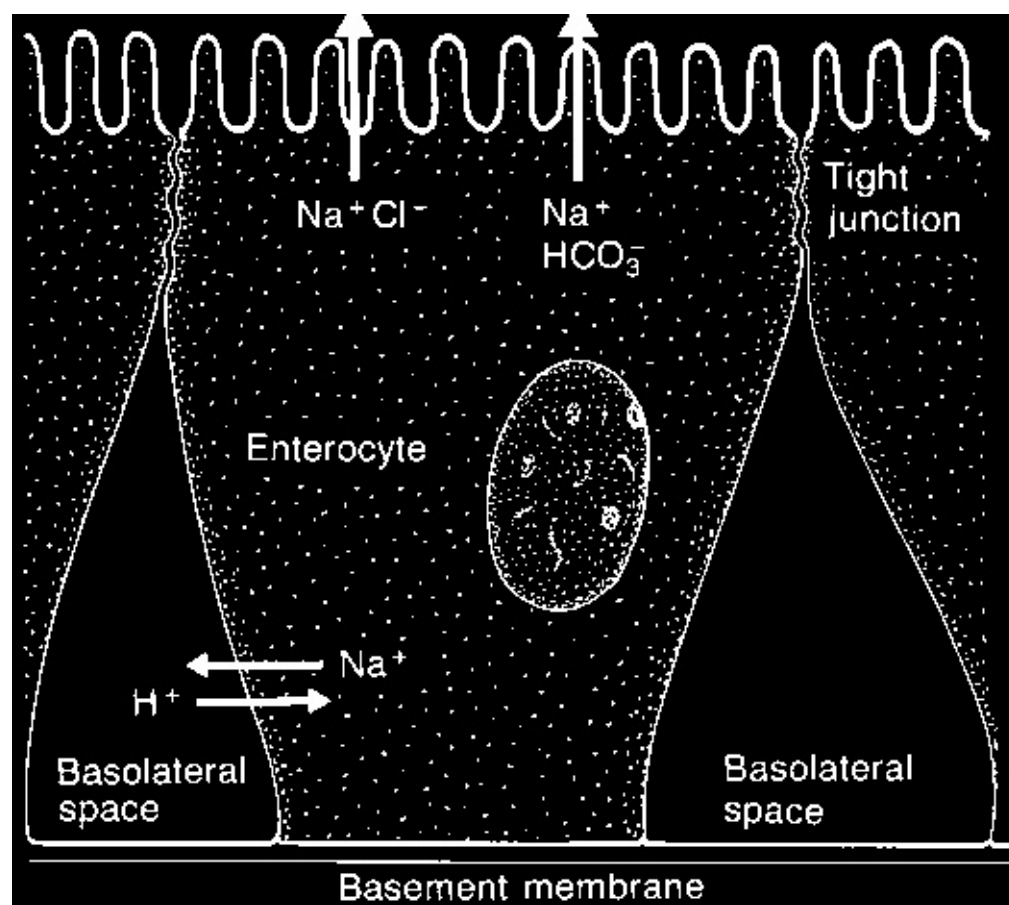
and correction of the problems. Identification of the

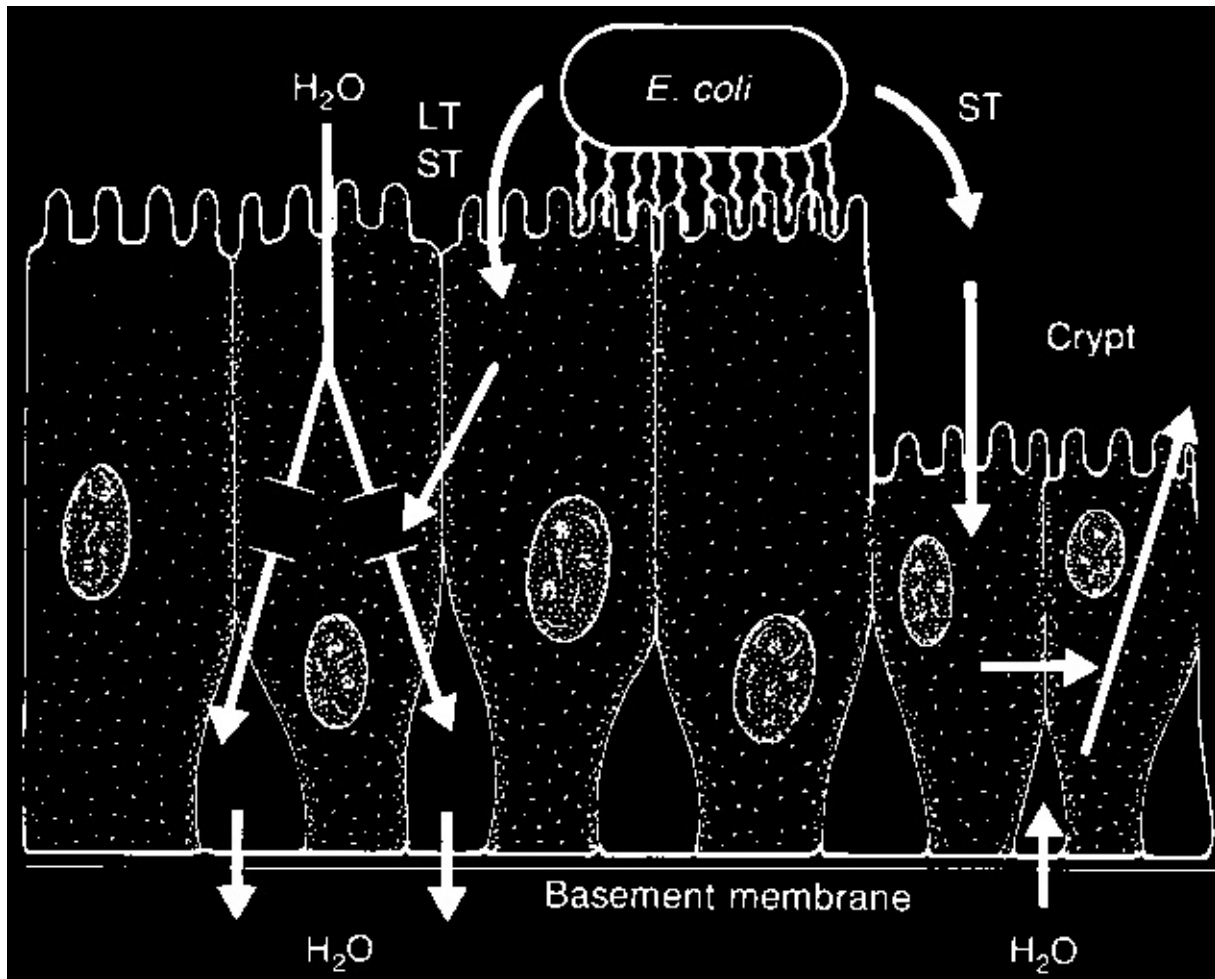
*Increased production of cyclic-AMP reduces sodium
infectious agents involved is important because it
ion absorption by villous cells (Fig. 14.5) and*

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Fig. 14.3

Line drawing illustrating some mechanisms of sodium secretion by crypt enterocytes. Sodium ions are secreted through the luminal surface and chloride and bicarbonate ions and water are transported within sodium ions.

Fig. 14.1

Interactions between management, the calf and enteric agents. Reproduced from Morgan (1990).

Fig. 14.4

Line drawing illustrating the mechanisms by which

enterotoxigenic E. coli cause diarrhoea. Bacteria are attached to the enterocyte surface by fimbriae and secrete heat-labile

enterotoxin (LT) or heat-stable toxin (ST), which act on metabolic pathways in villous enterocytes to block water absorption and

on metabolic pathways in crypt enterocytes to stimulate water secretion.

Fig. 14.2

Line drawing illustrating some mechanisms of move-

consequently water absorption is reduced. At the same

ment of sodium and chloride ions and water from the intestinal lumen secretion of chloride ions, and therefore water, is

lumen into basolateral spaces. The sodium pump expels sodium

stimulated in crypt cells (Fig. 14.6). The heat-stable

ions from the villous enterocyte into the basolateral space.

enterotoxin activates guanylate cyclase, stimulating

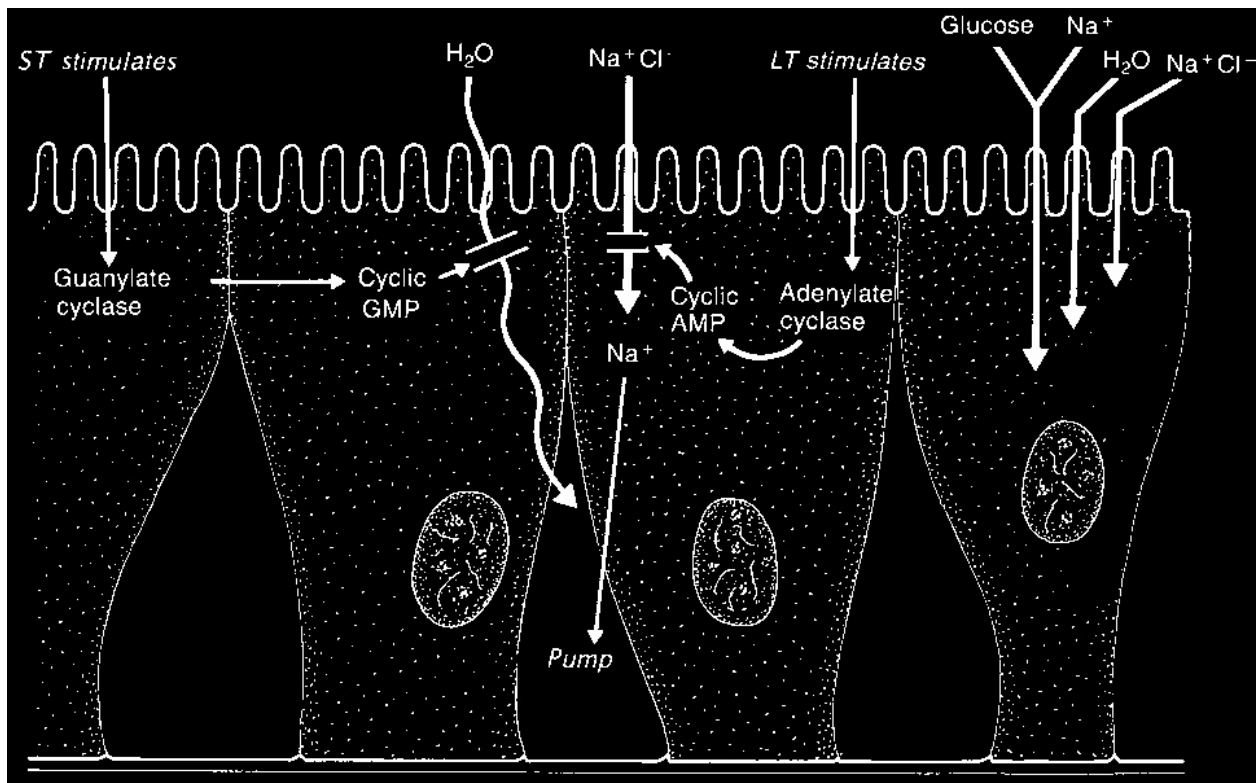
Sodium ions diffuse along the electrochemical gradient created intracellular synthesis of cyclic guanosine monophosphate by the sodium pump, through the enterocyte luminal surface

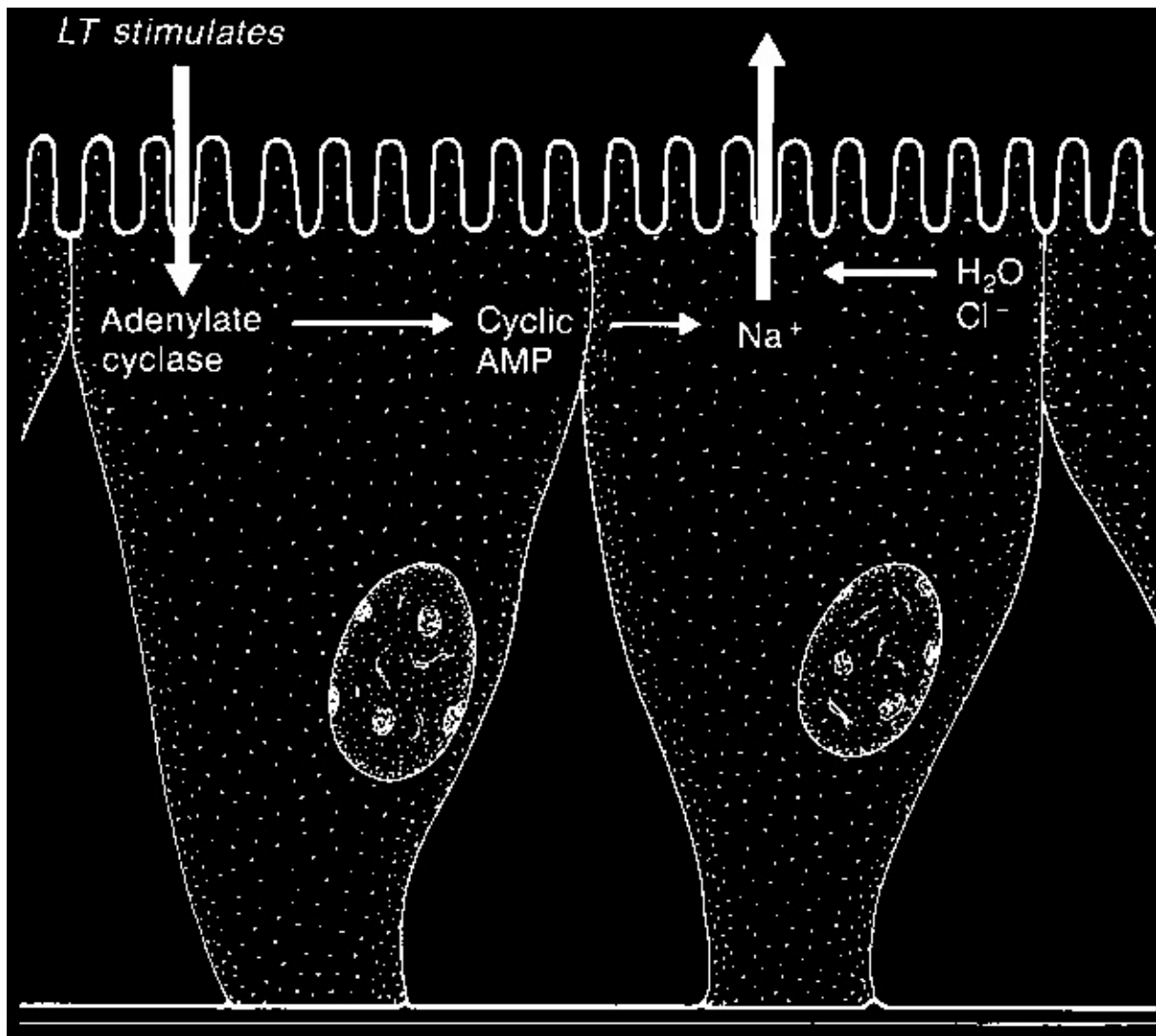
membrane, where passage is assisted by glucose-dependent

phosphate (cyclic-GMP),

which probably stimulates

and glucose-independent carrier systems. Chloride ions and secretion and reduces absorption, although the precise water follow the movement of sodium ions. mechanisms are not known (Fig. 14.5).





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Fig. 14.5

*Line drawing illustrating
the effects of heat-stable entero-
toxin (ST) and heat-labile entero-
toxin (LT) on movement of water
through villous enterocytes into*

basolateral spaces. ST inhibits water absorption via cyclic-GMP and LT, acting via cyclic-AMP, blocks absorption of sodium ions and water along the glucose-independent carrier system. The glucose-dependent carrier system is not affected by either toxin.

lumen. Malabsorption also occurs when morphological changes reduce the absorbing surface area. The best example of such morphological change is villus stunting produced by viral enteritis. The mature villous enterocytes can be regarded as the functional compartment of the small intestine, with absorption as one of the principal functions. A substantial reduction in the number of villous enterocytes causes a corresponding loss of function. Furthermore, crypt hyperplasia occurs in viral infections and immature secretory cells migrate onto the villus, increasing secretory capacity. In addition to loss of absorptive capacity there is also loss of digestive capacity, therefore maldigestion occurs, which

can lead to an osmotically induced diarrhoea.

Intestinal motility

Increased intestinal motility may contribute to the

Fig. 14.6

Line drawing illustrating the effects of heat-labile

development of diarrhoea, resulting in decreased

enterotoxin (LT) on the secretion of sodium ions by crypt cells, transit time and insufficient time for normal absorption.

rocytes. LT increases production of intracellular cyclic-AMP and this stimulates secretion of sodium ions, which carry with them The role of motility is now considered to be less

chloride ions and water.

important than was thought previously.

Osmotic effects

Lactose, the major sugar in cows' milk, is split into

Passive malabsorption

glucose and galactose by the enzyme galactosidase

Diarrhoea may be the consequence of water mal-

(lactase), which is located on microvilli of jejunal ente-

absorption (Fig. 14.7). If malabsorption occurs then

rocytes. These monosaccharides are rapidly absorbed

normal secretory processes and fluid loss due to tissue

and therefore have little osmotic effect. Viral hydrostatic pressure will continue and will cause diarrhoea. Water malabsorption will follow a direct reducing a transient deficiency of galactosidase. Consequence of active uptake of sodium ions from the intestinal lumen, lactose passes undigested to the colon together

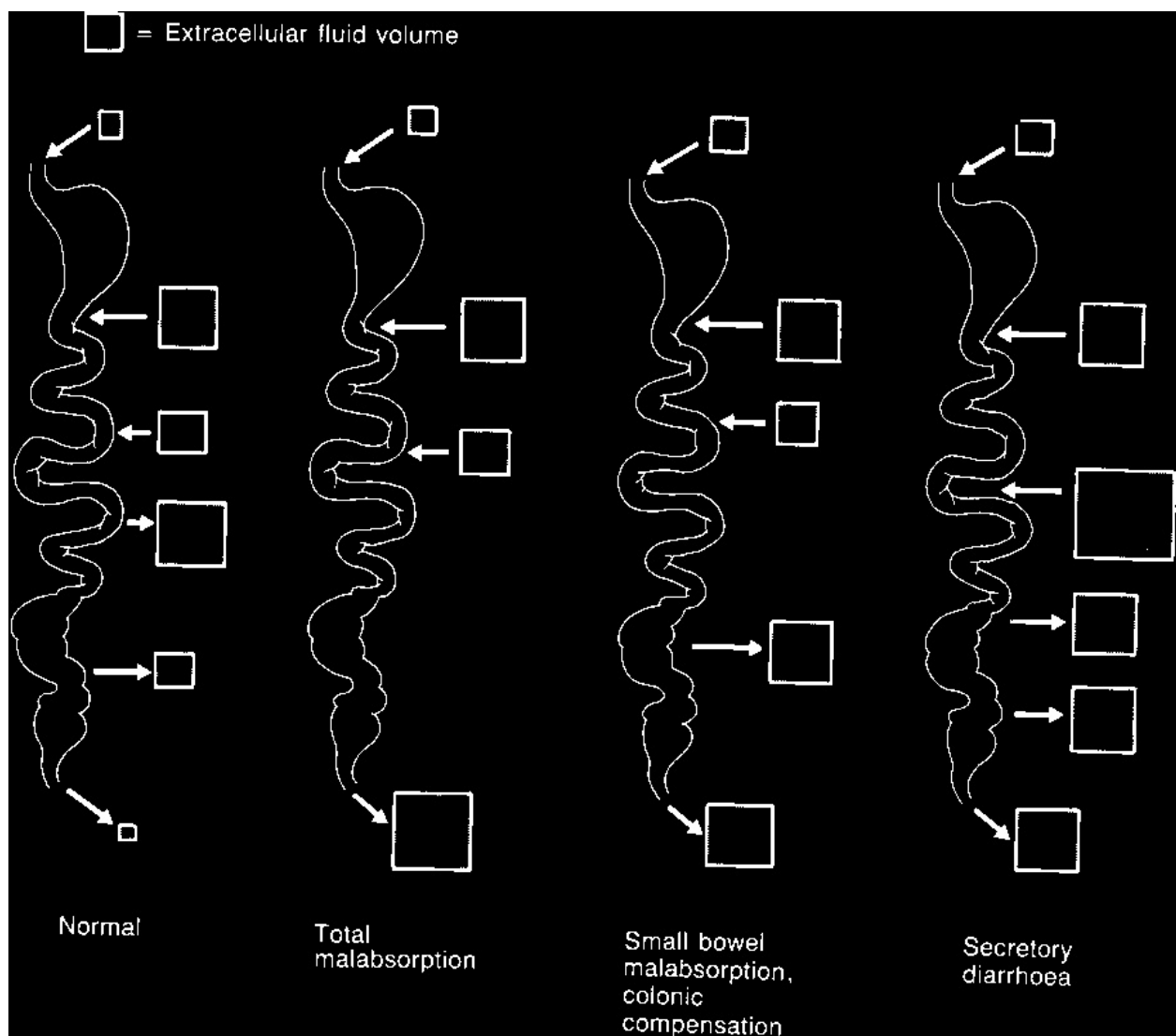


Fig. 14.7

Fluid fluxes in normal growing pigs (40 kg) and pigs with diarrhoea. Volumes of fluid entering and leaving the intestines are drawn in proportional size to the extracellular fluid volume shown at the top. Most water is absorbed by the distal small intestine and colon in pigs of this age and colonic compensation is likely to be much less in neonatal pigs.

Source: Argenzio (1984).

with an osmotic equivalent of water. It may be inferred

Tissue hydrostatic pressure and

from studies of pigs, goats and horses that the conse-

increased permeability

quence of increased lactose in the colon depends on the

Tissue hydrostatic pressure results in a continual

balance between input of lactose and the fermentative

seepage of water from the mucosa into the lumen and

capacity of the colonic microbial flora. If the calf possesses a well-developed colon and colonic microbial malabsorption is present. Seepage may be increased by flora and the amount of lactose entering is not excessive because of restricted dietary intake and/or slight inflammation of the mucosa, which allows greater leakage of fluid between enterocytes. In very severe intestinal damage, then the lactose will be fermented to inflammatory conditions, for example acute salmonellosis, the epithelium may be so extensively damaged as the colonic mucosa, a process that facilitates the absorption to allow erythrocytes to leak from capillaries, through the epithelium into the lumen; clearly other blood constituents will be lost also.

young animal, with a poorly developed colon and colonic microflora, there may be little fermentation of lactose that remains in the lumen, holding water and

contributing to the diarrhoea (Fig. 14.8). In an animal

Types of diarrhoea

with a well-developed colonic microflora, dumping of large amounts of lactose into the colon as a result of ad

Having examined the various pathophysiological

libitum feeding and severe intestinal damage results in processes that can give rise to diarrhoea, two general

hyperfermentation and the production of lactic acid

types of diarrhoea can be recognized.

rather than short-chain fatty acids. Lactate is poorly

absorbed and draws water into the colon by osmosis,

Secretory diarrhoea

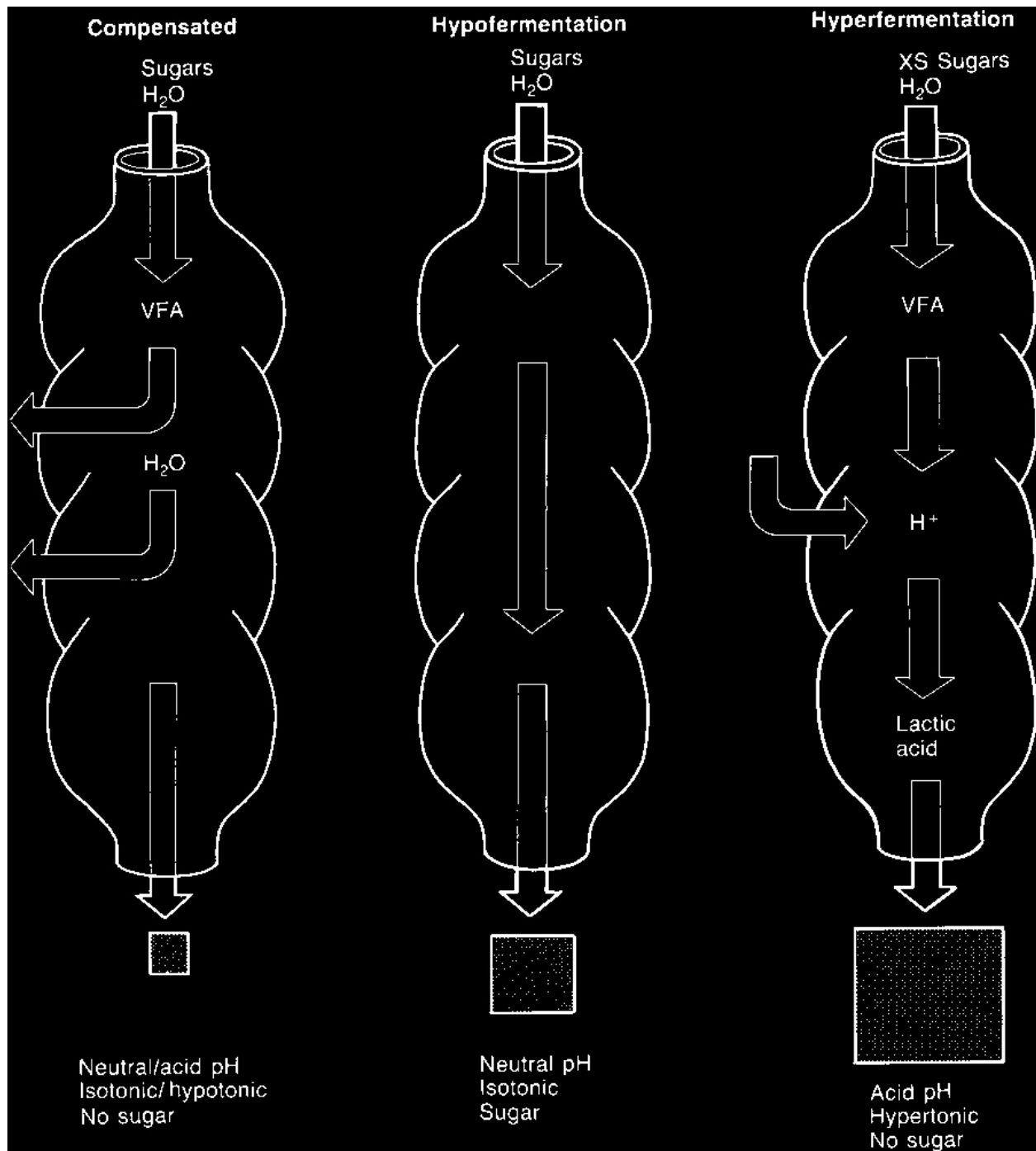
exacerbating the diarrhoea. These mechanisms explain

why withholding milk from calves with rotavirus scour

A diarrhoea resulting from net movement of fluid

reduces the severity of the diarrhoea (Fig. 14.8).

into the gut lumen despite fasting. Faeces are



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Fig. 14.8

Three alternative consequences of a small intestinal maldigestion-

tion and malabsorption, as seen in rotavirus infection. In the 'compensated' situation, the colonic flora is well developed and microbial fermentation of the moderate amounts of malabsorbed carbohydrate yields volatile fatty acids (VFA). These are rapidly absorbed, stimulating water absorption and promoting colonic compensation so that diarrhoea does not occur. If the colon and colonic flora are poorly developed there is hypofermentation and consequently no compensation, and diarrhoea occurs. If the colonic flora is well developed and is overloaded with sugar, hyperfermentation occurs and lactic acid is produced that promotes osmotic diarrhoea. Source: Argenzio (1984).

characteristically isotonic with plasma, watery and alka-

Role of the large intestine

line, and the volumes produced are usually large. The Most diarrhoea in the calf originates in the small intestine and as a result small intestinal function has ions are secreted by the ileum. In compensation, the received most study. It is evident that the large intestine, colon may be exchanging potassium ions for sodium particularly the colon, is a very important site of ions. Acute secretory diarrhoea is always caused by a water absorption and consequently may contribute to bacterial infection.

the development of diarrhoea if its functional capacity is overwhelmed or impaired by the fermentative

Osmotic diarrhoea

mechanisms described above. Similarly, its function may be impaired by infection with *Cryptosporidium*, coron-

A diarrhoea where the faeces may have high osmolal-

avirus or enterohaemorrhagic *E. coli*. Ingesta entering ity due to unabsorbed molecules with osmotic activity,

the colon from the small intestine may be damaging.

usually of dietary origin. Faeces may contain undigested

lactose and faecal pH will vary, depending on the amount of lactose fermented to short-chain fatty acids or lactic acid. Osmotic diarrhoea may also be thought

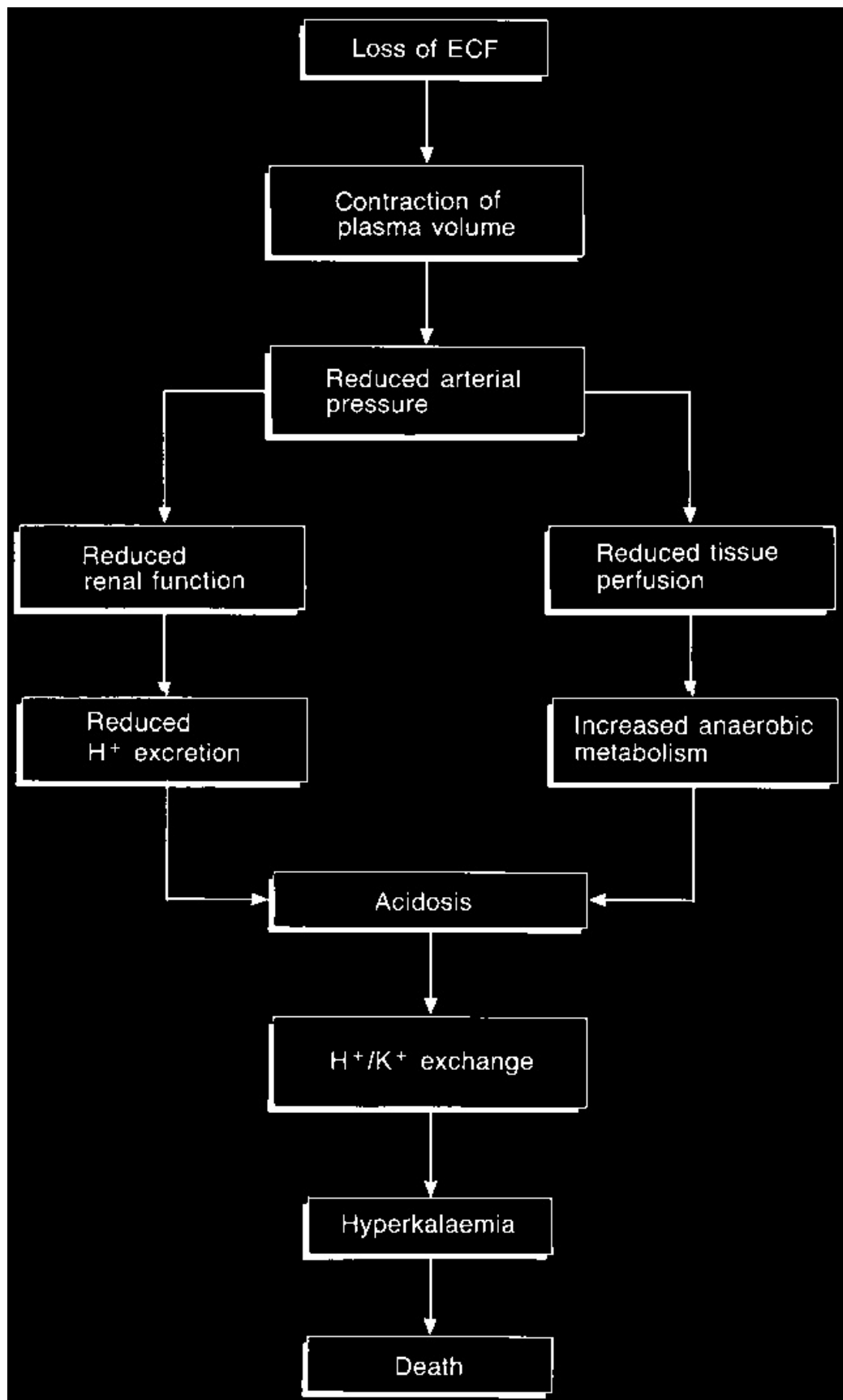
Effects of diarrhoea and

of as a diarrhoea caused by malabsorption and

their clinical signs

malabsorption. Faecal volume is smaller than in secretory diarrhoea and the diarrhoea is reduced or abolished by

The systemic effects of diarrhoea, which eventually culminate in death, are precipitated by a single event, the



sodium and chloride ions and considerable losses of bicarbonate and potassium ions. There was loss of body weight, 12.7 per cent between onset of diarrhoea and death. Plasma volume decreased by 40 per cent leading to a 39 per cent increase in haematocrit and a 33 per cent increase in plasma protein concentration. The increase in plasma protein concentration was lower, possibly because of protein loss by catabolism or by leakage into the intestinal lumen. Both these values vary widely between individuals and are of little value in assessing severity of fluid loss.

Contraction of plasma volume gives rise to the clinical signs of sunken eyes and 'tenting' of skin folds; it leads to a fall in arterial blood pressure, which stimulates peripheral vasoconstriction. Peripheral vasoconstriction, in its turn, leads to poor tissue perfusion with blood, localized ischaemia and lower metabolic activity so that the temperature of peripheral tissues falls, approaching ambient temperature prior to death; the

extremities, the ears and mouth, feel cold. Rectal temperature increases until near to death when it falls rapidly to below normal.

Acidosis is an important consequence of diarrhoea and a number of factors contribute to its development. A major factor is loss of bicarbonate ions in faeces and, additionally, there may be absorption of acids produced by microbial fermentation of lactose in the large intestine. Loss of extracellular fluid (dehydration) causes decreased perfusion of the kidney with blood, causing reduced renal function, which leads to decreased excretion of hydrogen ions by the kidney. Finally, lactic acidosis may develop because of increased production of lactate following peripheral hypoxia, and decreased uti-

Fig. 14.9

*The systemic consequences of diarrhoea. Reduction
lization of lactate due to decreased delivery of lactate*

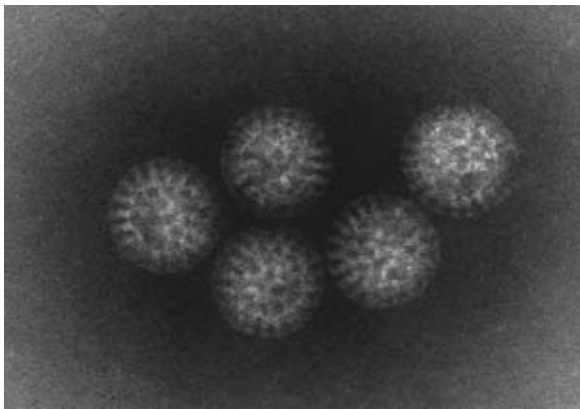
of extracellular fluid volume leads to acidosis, an exchange of to the liver. The ability of the liver to use lactate for glu-extracellular H^+ for intracellular K^+ leading to hyperkalaemia coneogenesis may be impaired because of increased causing cardiac failure. Source: Argenzio (1984).

intracellular concentration of hydrogen ions. Indeed,

the liver may become a lactate producer rather than a lactate utilizer. The calf attempts to reduce metabolic acidosis by panting to increase exhalation of carbon dioxide. Intracellular acidosis occurs in parallel with the loss of extracellular fluid. Endogenous secretion into fall in the blood pH. Intracellular production of hydro- the gastrointestinal tract in 24 hours may equal the gen ions increases and hydrogen ions move into cells. extracellular fluid volume. Loss of 7 per cent of the This movement of hydrogen ions into cells forces potas- extracellular fluid volume leads to clinical signs and loss sium and sodium ions to be lost and hyperkalaemia of 30 per cent results in death.

develops. However, potassium is also lost in the faeces, Diarrhoea causes changes in plasma constituents that so that the plasma concentration may, theoretically, be are similar regardless of the cause of the diarrhoea (Fig. increased, normal or decreased depending on the rate 14.9). When neonatal colostrum-fed calves were experi- of loss from plasma into faeces, and from cells into mentally inoculated with coronavirus, the water and

plasma. In general it is increased. The loss of potassium electrolyte losses were severe (Lewis & Philips, 1978). ions from cells into the interstitial fluid causes levels of Faecal water loss increased 28-fold, faecal volume intracellular potassium to fall and levels in interstitial increased 22-fold and faecal water content increased fluid and plasma to rise and, consequently, adjacent to from 73 to 94 per cent. Renal water loss was reduced to cells, the ratio of extracellular potassium : intracellular 30 per cent of normal and there were severe losses of potassium is reduced. This redistribution of potassium



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causes a reduction in the resting potential of cell mem-

Bovine rotavirus

branes, which has serious and eventually lethal effects

The agent

on cardiac muscle function. As the concentration of potassium ions in plasma rises, heart rate falls and there is a decreased amplitude, or loss of the P wave. The virus contains double-stranded RNA in 11 segments. Particles have a wheel-like appearance; a wide hub formed by the core, spokes formed by 20 outer capsomers and an often ill-defined outer rim (Fig. 14.10). Incomplete particles lacking the outer capsid layer are to be due to potassium cardiotoxicosis. death from acute severe diarrhoea in the calf appears frequently seen in faeces. Almost all bovine rotaviruses share a common antigen and have been classified as group A rotaviruses.

Metabolic and hormonal changes

Hypoglycaemia frequently occurs in acute severe diarrhoea of calves, especially young calves near death.

Epidemiology

Bovine rotavirus is universally present in all cattle. Anorexia, decreased absorption of nutrients, minimal glycogen reserves, inhibited gluconeogenesis, increased suckled beef calves than dairy calves because of the glycolysis due to reduced tissue perfusion and anoxia much greater risk from cross-contamination between and insulin-like effect of bacterial endotoxins on liver calves, the seasonal nature of the calving pattern and may contribute to the hypoglycaemia. Signs of hypoglycaemia are weakness, lethargy, convulsions and during late gestation.

coma. Hypoglycaemia stimulates corticoid secretion; The incubation period varies from 15 hours to five plasma concentration of corticosterone and hydrocortisone are elevated in calves with diarrhoea and are mences in the second week of life. Many infections are higher in calves that die. Theoretically, corticosteroids subclinical but a proportion results in severe diarrhoea

help counter hypoglycaemia by stimulating gluconeogenesis and even death if not treated promptly. The peak incidence of diarrhoea occurs at about 10 to 14 days of age. Initially, calves are dull and reluctant to suck. The dam's udder becomes distended with milk and may be the first indication to the diligent stockperson that the calf is unwell. Diarrhoea develops which is pale yellow or white and may contain mucus; dehydration and metabolic acidosis develop in more severely affected calves which limits the helpful actions of aldosterone.

Infectious agents

The elucidation of the infectious causes of calf diarrhoea has been a major area of progress over the last 30 years. For many years, salmonellas were the only

known cause, but in 1967 it became clear that a small

number of strains of *E. coli* caused a watery diarrhoea and subsequently these came to be known as enterotoxigenic *E. coli* (ETEC). Rotavirus was the first viral enteropathogen to be recognized, followed by coronavirus, and more recently Breda virus. Approximately

20 years ago, *Cryptosporidium parvum* was found to

cause diarrhoea in calves. Most recently, *E. coli* have been identified that cause diarrhoea without producing

enterotoxins; these include strains that infect the large

Fig. 14.10

Transmission electron micrograph of a cluster of

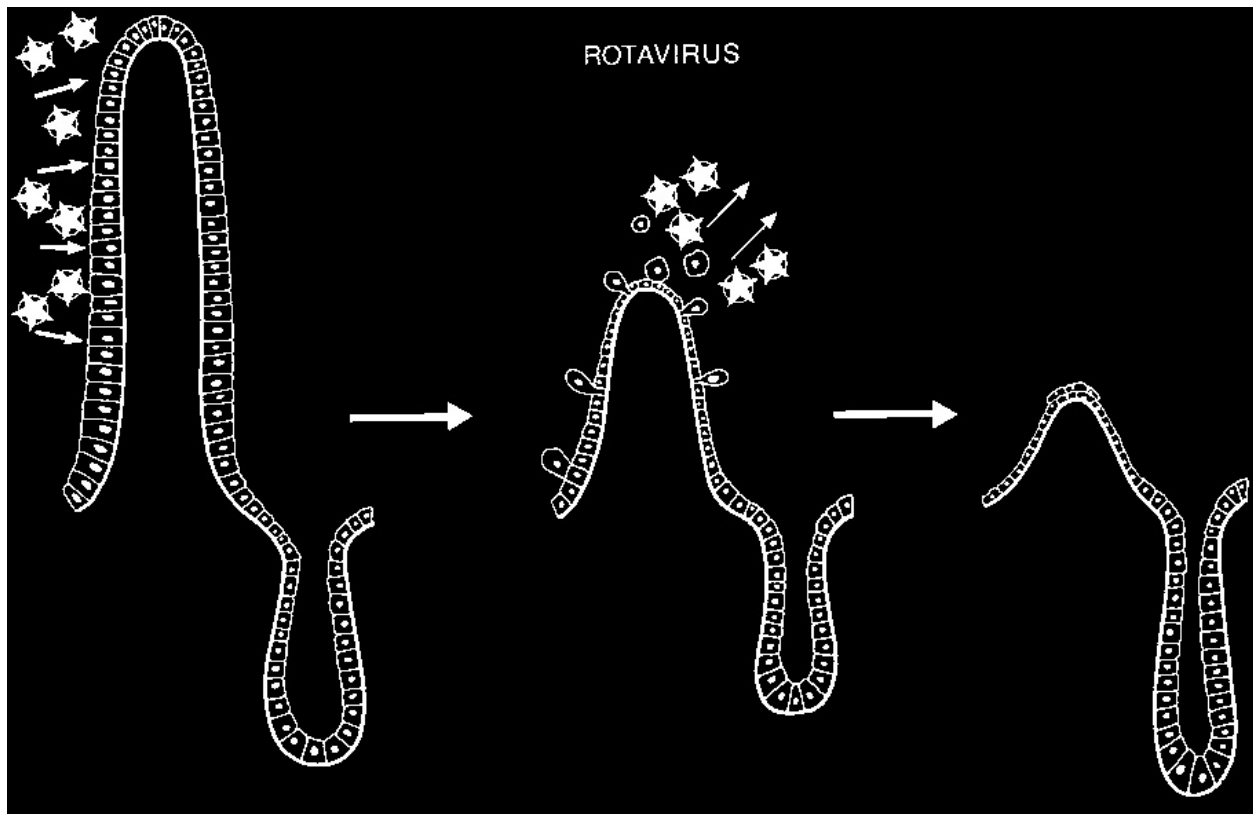
intestine and cause mild dysentery, the enterohaemor-

five bovine rotavirus particles. Visible are the wide hub, spokes rhagic *E. coli* (EHEC), and strains comparable to

formed by the capsomeres, and the outer rim. (Courtesy of Dr

human enteropathogenic *E. coli* (EPEC).

J.C. Bridger, Royal Veterinary College, University of London.)



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and may be fatal. At this stage the calf may have

et al., 1971). There is variation in pathogenicity between abdominal distension due to fluid sequestration in the

strains; whilst some strains may only damage a limited

small intestine. Excretion of copious amounts of

length of upper jejunum, others may damage the entire

yoghurt-like faeces is typical of rotavirus infections that

length of the small intestine. Some poorly virulent

are uncomplicated by other enteropathogens and the

strains are able to infect enterocytes, replicate within

faeces are thought to have this appearance because them and cause enterocyte loss, but not at a sufficiently they represent the passage of partially digested or rapid rate to outpace repair mechanisms and cause undigested milk.

lesions and diarrhoea. The existence of strains of low Occurrence of pyrexia is variable, but when it occurs virulence has implications for accurate diagnosis of it is usually mild ($<39.5^{\circ}\text{C}$) and may be more suggestive rotavirus diarrhoea because it may be assumed that of a focal bacterial infection such as omphalophlebitis such strains could be excreted by calves with diarrhoea or polyarthritis.

caused by other enteropathogens. To be confident in a diagnosis of rotavirus diarrhoea, it is necessary to identify rotavirus in the faeces of significantly more diar-

Pathogenesis

rhoeic calves than in age-matched, clinically normal Rotaviruses infect mature enterocytes, located on the calves on the same farm. On the basis of present knowledge of villi. Particles are detected in the cytoplasm,

edge, groups of four affected and four normal calves are usually in dilated cisternae of the endoplasmic reticulum.

Masses of granular or finely fibrillar virus precursor (viroplasm) containing virus cores are present

The changes in intestinal structure affect its function.

Loss of mature enterocytes with their lactase, and their

outside the cisternae. Particles released from viroplasm replacement with immature enterocytes containing less

pass into the cisternae; some bud through the membrane of the endoplasmic reticulum and acquire an

envelope. Viral multiplication within the cells initiates surface area of the small intestine is reduced, there is

degenerative changes, the cell exfoliates and the rapid reduced ability to absorb the glucose and galactose that

loss of large numbers of cells leads to fusion and stunting of villi. The columnar epithelium is replaced by

accumulates in the large intestine, where by virtue of its

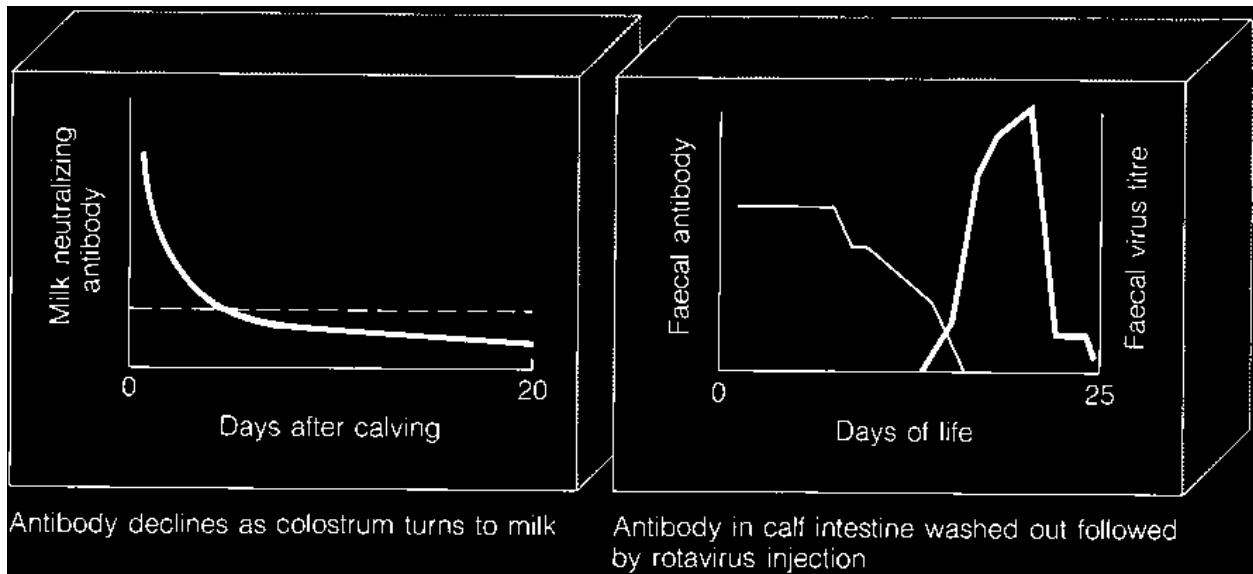
enterocytes that are cuboidal or squamous and the hypertonicity it prevents absorption of water from epithelium contains increased numbers of immature faeces and contributes to the development of water loss cells from the crypts (Fig. 14.11). In very severe cases and dehydration. Bacterial fermentation of the lactose the villi may be obliterated leading to a totally flat may increase the osmotic effects. As a result of entero-epithelium. The pathogenic process may commence in cyte loss, the population of enterocytes on the villi the upper jejunum and progress along the small intestine from mature cells with digestive and absorptive function to the ileum, producing a wave of damage (Mebus

Fig. 14.11

Line drawing illustrating the development of the small intestinal lesion in rotavirus infection. Mature enterocytes are infected and virus replication causes enterocyte death and sloughing. Villi stunt and the surviving

enterocytes are cuboidal or squamous.

Mitosis increases in the crypts, which elongate. The star within a circle represents a virus particle.



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secretory function. The number of cells in the crypts calves by vaccinating dams with commercial combined (secretory cells) also increases. Thus the functional inactivated vaccines containing rotavirus, coronavirus balance may change from absorption to secretion.

and E. coli K99 one to three months before the anticipated calving date. Because rotavirus is endemic in

herds, cows have antibodies to rotavirus in their serum

Prevention

and parenteral administration of a single dose of inactivated virus absorbed onto aluminium hydroxide gel, serum antibody to rotavirus, which can be detected by and the whole emulsified in a light mineral oil, boosts a number of tests. These antibodies, however, do not these pre-existing serum antibodies. Consequently, the protect against infection and disease because for protective purposes neutralizing antibody is required in the increased and antibody is present in milk for longer gut lumen. This is because the entire disease process than in unvaccinated animals. Thus, dam vaccination is occurs on the mucosal surface and if antibodies are to essential on those farms which have experienced losses interfere with the pathogenic process they must be due to viral diarrhoea in young calves. This system is present in the gut contents that bathe the mucosal suitable for both suckled calves, and for artificially surface; luminal antibodies neutralize virus and prevent reared calves where hyperimmune colostrum is saved

initial infection and spread of infection from enterocyte from the first six to eight milkings and stored in a cool to enterocyte. There is recent evidence, however, suggesting that serum antibodies, which originated from for the first two weeks of life.

the colostrum of hyperimmune cows, can be transferred It is essential that all calves ingest sufficient good to the gut where they are protective.

quality colostrum within the first six hours after birth Following natural infection, calves are immune to with values equivalent to between 7 and 10 per cent of disease; this is thought to be the result of an active bodyweight commonly quoted. Factors that may reduce mucosal immunity provided by IgA and cell-mediated the level of specific antibody accumulation in the mechanisms. Repeated episodes of reinfection without colostrum differ between beef and dairy cows because signs occur throughout life, maintaining herd immunity of the lower nutritional and management inputs in beef and the virus in the population and environment.

herds and the higher prevalence of metabolic disease in
Colostrum contains antibodies to rotavirus that help
high-yielding dairy cows.

protect the calf against infection and disease. The con-
Problems with sufficient colostrum accumulation in
centration of antibody in colostrum and early milk from
the udder of beef cows include debility of the dam, poor
unvaccinated cows declines rapidly, reaching levels that
nutritional status during late gestation, intercurrent
are thought to be non-protective three to four days
disease such as fascioliasis and chronic mastitis caused
after parturition (Fig. 14.12).

by *Arcanobacterium pyogenes* (summer mastitis)

Under farm conditions where rotavirus is always
(Chapter 24). Dystokia is more common in beef calves
present, feeding colostrum only immediately after birth
leading to extended intervals to standing and sucking.
delays, rather than prevents, rotavirus excretion and
As a general rule, those calves born following an
calves are susceptible to infection and disease in the
assisted calving should receive 2 litres of colostrum

second week of life. Protection of the calf has been within the first two hours of life and be closely super-achieved by providing enhanced passive protection to vised to ensure normal sucking behaviour.

Fig. 14.12

Graphical illustration of changes that occur in neutralizing antibody to rotavirus in cows' milk after calving and faecal antibody to rotavirus and faecal virus in the early days of a calf's life. Reproduced from Morgan (1990).

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Recumbency resulting from hypocalcaemia, over-which alter the clinical presentation, whereby profound crowding of the calving accommodation, slippery acidosis is the main presenting feature rather than underfoot conditions and poor mothering ability, especially in dairy heifers, may delay the ingestion of and the abomasum and intestines are distended with

sufficient colostrum by the newborn dairy calf.

fluid and 'slosh' when succussed.

Dam vaccination may not prevent all episodes and

Calves treated with oral rehydration solutions by the

rotavirus diarrhoea may follow stressful events such as

farmer using an oesophageal feeder often have normal

housing and disbudding if undertaken before the calves

hydration status but become severely acidotic, causing

are six weeks old.

profound weakness and depression extending to stupor.

There is no relationship between the degree of dehy-

dration and the severity of the acidosis. The eyes are not

Diagnosis

sunken and the skin tent duration is normal. Fluid dis-

Rotavirus infection produces the complete range of

tension of the abdomen is obvious. The respiratory rate

clinical signs from no observed abnormality through to

is increased above 40 breaths per minute with increased

severe diarrhoea and dehydration with high mortality

depth of expiration. The heart rate is often less than 90

even in treated calves. The incubation period is one to

beats per minute.

three days depending upon the level of viral challenge.

A thorough clinical examination is essential in all

Isolation of rotavirus from faecal samples does not nec-

young calves with diarrhoea because of the likelihood

essarily indicate a diagnosis of rotavirus diarrhoea

of other focal infections such as early bacterial menin-

because rotavirus can be isolated from a high propor-

goencephalitis, respiratory disease, omphalophlebitis

tion of normal healthy calves without signs of diar-

and possible associated peritonitis, hypopyon and pol-

rhoea. Comparison of faeces samples collected from

yarthritis. Indeed, the diarrhoea reported by the client

healthy and diarrhoeic populations of calves may be

may be the agonal stage of a septicaemia, although such

necessary to determine the precise aetiology but such

peracute infections usually occur in calves less than six

sampling will prove cost-prohibitive in most farm

days old.

situations. Determination of the age of affected calves,

In normal calves more than two days old the um-

and vaccination status of their dams against infectious
bilical remnant is dry and shrivelled, hastened by its
causes of neonatal diarrhoea, will assist in determining
immersion in strong veterinary iodine solution imme-
diately after birth and again six to 12 hours later.
infections are common. For example, *Cryptosporidium*
Omphalophlebitis manifests as a swollen (>3 cm diam-
eter), wet and painful umbilicus which may discharge
pus under digital pressure (see p. 249). Localized peri-
Calves with rotavirus infection during the early
tonitis associated with omphalophlebitis may result in
stages of disease typically show signs of mild depression
the formation of fibrinous adhesions and associated
and salivation, reluctance to stand and suck and profuse
ileus which proves difficult to detect on clinical exami-
nation alone. Abdominocentesis is difficult to perform
rapidly spreads among young calves present on the

on such recumbent calves because of the fluid-distended intestines and the scant fibrinous nature of the adhesions. Similarly, abdominal ultrasonography fails to identify such early adhesions. Calves with peritonitis blood or mucosal casts. The rectal temperature is fail to respond to standard fluid therapy and antibiotic usually within normal limits during the early stages therapy regimens, with the adhesions identified at unless there is evidence of focal bacterial infection. post-mortem examination.

Without oral rehydration therapy the affected calf Polyarthrititis manifests as hot, distended and painful becomes dehydrated, hypothermic and weak, and joints, typically the fetlock, hock and carpal joints, remains in sternal recumbency. The eyes are sunken and resulting in the tendency for calves to adopt lateral the skin becomes tight and inelastic, consistent with 8 recumbency rather than sternal recumbency associated

to 10 per cent dehydration within 48 hours, unless
with weakness (see pp. 249, 455).

treated appropriately. As hypovolaemic shock develops,
peripheral vasoconstriction causes the calf's extremities

Laboratory diagnosis of rotavirus

to feel cold. At this stage the calf is often hypothermic
($<38.0^{\circ}\text{C}$). Without treatment, death may ensue within

Enzyme-linked immunosorbent assays (ELISA) have
72 hours of the onset of diarrhoea.

been developed for rotavirus and a plate ELISA is used

In most situations diarrhoeic calves have been

by many diagnostic laboratories. Some diagnostic

treated by farm staff with oral rehydration solutions

laboratories use the polyacrylamide gel electrophoresis

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method, which has proved to be equally satisfactory.

Example: treatment of diarrhoeic calf under

The diagnostic kits available to practice laboratories in
field situations

the UK are a dot ELISA, a latex method and a plate

The objectives of intravenous fluid therapy include the

ELISA system. The plate ELISA system requires a correction of extracellular fluid volume, plasma pH, minimum of equipment and also tests for bovine coronavirus and K99+ Escherichia coli; a modification to the of sodium and potassium, restoration of cellular system has permitted the detection of Cryptosporidium potassium and the provision of nutrients. spp.

A 10 day-old, 45 kg single suckled beef calf is pre-The identification of rotavirus particles or antigen sented with profuse diarrhoea with faecal staining of in the faeces of calves with diarrhoea does not the tail, perineum and hind legs. The calf is weak, unable automatically lead to a diagnosis of rotavirus disease to rise or remain standing when lifted. It is markedly in the outbreak. Rotavirus antigen was detected in depressed with cold extremities. The skin tent is about 50 per cent of faeces from healthy calves aged one three seconds. The eyes do not appear sunken but to two weeks in a study in which an ELISA test was

the scleral vasculature is pale. The heart rate is 85
used. Thus, in order to reach a sound diagnosis, and
beats/minute and the respiratory rate is 45
using that test to identify excretors, it was necessary to
breaths/minute. Two litres of an oral rehydration solution
show that significantly more than 50 per cent of diar-
hoeic calves were excreting rotavirus. This was
daily for the past two days.

achieved by examining faeces from four calves with
diarrhoea and four matching normal calves on the farm.

The number of calf faeces that will need to be exam-

Typical laboratory values: Typical laboratory values are ined in an investigation
of an outbreak will depend on

listed below for this calf but such analyses are rarely
the ability of the diagnostic test in use to detect
possible under field situations. Excellent clinical recov-
excretion by normal calves.

ery rates for such cases are achieved in general practice
using estimations of the base deficit based upon the
clinical examination

Treatment (see p. 209)

Packed cell volume = 31%

Arterial blood gas

analysis:

Veterinary treatment of calf diarrhoea is generally

Total plasma protein = 68.0 g/l pH = 6.9

limited to collapsed animals which have failed to

Sodium = 128 mmol/l

pCO =

2

46 mmHg

respond to oral rehydration solutions. Administration

Potassium = 7.2 mmol/l

HCO =

3

7 mmol/l

of oral rehydration solutions is frequently made by the

Chloride = 105 mmol/l

Base deficit = 20 mmol/l

farmer using an oesophageal feeder. While this proce-

dure has considerable time savings, it is frequently used

*The calf is estimated to be only 5 per cent dehydrated.
for weak recumbent calves without a strong suck reflex
The serum potassium concentration is elevated because
which serves to further delay veterinary involvement to
intracellular K^+ ions exchange with H^+ and pass into the
the stage where calves are profoundly acidotic when
extracellular fluid. However, the intracellular (and total
presented. In veterinary practice therefore, treatment
body) K^+ ion concentrations will be lowered, but this
of calf diarrhoea involves the correction of acidosis
deficit is more safely replaced by the oral rehydration
rather than simple correction of hydration status,
solution rather than the intravenous fluid. Diarrhoeic
although both problems may have to be tackled at the
calves are not markedly hypoglycaemic and nutrients
same time.
are better supplied in the oral rehydration solution, and
Basic animal husbandry is frequently overlooked in
then milk, than in the intravenous fluids.
many modern beef production systems. The diarrhoeic
calf should be isolated in a warm, dry and well-bedded*

Intravenous fluid therapy: The fluid requirements are pen. In beef herds, the dam should be separated from calculated as:

the calf by a gate which allows visual contact.

45 kg \times 0.05 = 2 litres

Hypothermic calves should be warmed and this is best achieved with convector heaters rather than heat lamps. The daily requirement of 75–150 ml/kg should be added

to this deficit. The fluids should be warmed to body

Intravenous fluids are essential because most temperature before administration and insulated if left severely affected diarrhoeic calves are profoundly acid-suspended in a cold environment.

otic (see example below), with variable degrees of dehydration depending upon previous oral rehydration

Base deficit: The base deficit is estimated from the therapy.

clinical findings whereby profoundly weak and

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depressed/stuporous calves, in the absence of sepi-

rate information on the precise severity of the acidosis, caemia or bacterial meningoencephalitis, have a base the farmer can be instructed to spike the intravenous deficit in the region of 20 mmol/l. As an alternative fluid with materials left on the farm.

practical guideline, calves able to maintain sternal recumbency are reported to have a base deficit of Treatment regimen: Under practical conditions on farm 15 mmol/l, whilst those in lateral recumbency have a a 14 gauge (or 16 gauge) catheter should be stitched deficit of 20 mmol/l (Naylor, 1996). However, such dis- into a jugular vein as this makes fluid administration tinction is not necessary to achieve excellent results, much easier because the calf can be left unattended. A even under less than ideal practice situations.

5 cm by 5 cm area of skin overlying a jugular vein is The total base deficit (or negative base excess) can shaved and aseptically prepared. A stab incision be calculated as:

through the skin overlying the jugular vein using a 15T blade greatly facilitates catheter placement. If the calf

base deficit \times bicarbonate space \times dehydrated calf
is severely dehydrated and cannot be catheterized
weight = 20 \times (0.3 to 0.6) \times 45 = 270 to 540 mmol
easily, either the first litre of fluid can be administered
bicarbonate

through a 16 gauge hypodermic needle or the calf is
There is considerable debate as to which value should
suspended by its hind legs to distend the jugular veins,
be employed for the bicarbonate space because this
thereby facilitating catheterization.

value is higher in neonates than adults. In practice
A typical treatment regime for a recumbent acidotic
situations, where blood gas analysis is rarely available,
calf would commence with 1 litre of isotonic saline
an estimate of 20 mmol/l for base deficit in recumbent
spiked with 200 mmol bicarbonate (16 g sodium bicar-
stuporous calves is commonly used in conjunction with
bonate) administered intravenously over 20 to 30
an estimate of 0.5 to 0.6 for the bicarbonate space.
minutes. This solution can be administered while the
While it is accepted that there will be errors in such a

veterinary surgeon remains on the farm suturing the simplified approach, this regimen has proved very reliable in general practice where the rapid response to set, and possibly attending further calves with diarrhoea intravenous fluid therapy spiked with bicarbonate is the or collecting faecal samples as appropriate.

most valuable indicator of success of the fluid therapy A further 3 litres of isotonic saline solution contain-regimen.

ing the balance of the estimated bicarbonate deficit Typically, stuporous calves should be much improved (approximately 400 mmol) should then be administered after three to four hours, and be able to stand six to over the next four to six hours, by which time the calf's eight hours after the start of intravenous fluid therapy. demeanour will be much improved. The calf should be A thorough initial clinical examination will eliminate able to stand and suck a teat enthusiastically at this focal and septicaemic infections from the list of different stage. An oesophageal feeder should be used only as a

ential diagnoses. Re-evaluation of the diagnosis and/or last resort to administer oral fluids because the calf's treatment is necessary if the calf's condition has not demeanour and willingness to suck are valuable indicators of the calf's recovery. Indeed, early cases of intravenous fluid therapy. The two most common bacterial meningoencephalitis may be difficult to differentiate from profound acidosis, but acidotic calves the extent of the base deficit has been underestimated should respond well within six hours to spiked and insufficient bicarbonate has been infused or, intravenous fluid therapy and suck, whereas those with less commonly, that the calf has bacterial meningoencephalitis will not and may progress to encephalitis (see pp. 251, 901).

opisthotonus with spontaneous nystagmus. Alternatively, in cases where meningoencephalitis is suspected to determine total CO₂, which is a useful index of the

during the veterinary examination, lumbar CSF can be severity of the metabolic acidosis. However, determined under local anaesthesia and inspected visually for turbidity caused by white cell infiltration.

blood gas analysis necessitates hospitalization of the calf with the attendant risks of contaminating the facilities described above has been employed in many instances with a range of enteropathogens, some of which are farm animal practices because the calf remains on the serious zoonoses. While blood samples can be transported to the practice laboratory for blood gas analysis, removing the risk of introducing enteropathogens, such retrospective information may not be so helpful

including *Salmonella* spp., into the practice hospital because financial considerations frequently preclude

facilities with the potential for disastrous consequences.

a second veterinary visit to correct any estimations

Often only one veterinary visit is possible for each diar-

based upon clinical findings. Alternatively, with diarrhoeic calf because of cost limitations, but this system

Calf Diarrhoea • 197

of intravenous fluid administration works well in whereby sodium is transported across the gut wall into practice.

the extracellular fluid and water follows along the Most diarrhoeic calves are not profoundly hypoglycemic and the addition of glucose to the intravenous propionate, glycine and glutamine, also play an important role in sodium transport. Energy supply is

better given as a component of the oral rehydration

The original World Health Organization (WHO) oral solution therapy (high energy rehydration solution), rehydration solution was an equimolar solution containing approximately 100 mmol/l sodium and glucose the start of intravenous fluid administration. Recently and was the basis of the first generation oral rehydra-

introduced oral rehydration solutions have a high
tion solutions introduced into veterinary practice
energy content designed to counter the energy deficit
during the 1970s. Potassium was present to assist in
state caused by withholding milk from the calf's diet. As
restoring any deficits. These solutions contain no bicar-
soon as the suck reflex returns, the calf should be
bonate or precursor and are therefore unable to correct
offered either one litre of oral rehydration solution or
a significant acidosis, but are effective in many cases of
milk at alternate feeds every two hours.

calf diarrhoea associated with dehydration. Viral causes
of neonatal diarrhoea were not reported in the United
Other intravenous fluids: Clinical evaluation of the
Kingdom until the mid-1980s (Snodgrass et al., 1986),
alkalinizing effect of various bases in diarrhoeic calves
and these oral rehydration solutions proved effective
suffering from severe dehydration and acidosis (Kasari
in the treatment of bacterial and nutritional causes of
& Naylor, 1986) clearly demonstrated the advantages in
calf diarrhoea which were not usually associated with

*both speed and extent of administering isotonic saline
severe metabolic acidosis.*

solutions containing bicarbonate compared to those

*The widespread occurrence of rotavirus-induced
solutions containing either lactate or acetate.*

diarrhoea and associated metabolic acidosis led to the

Lactated Ringer's solution should not be used to

development of oral rehydration solutions containing

treat severe acidosis because it contains only 30 mmol/l

80 to 120 mmol/l bicarbonate as a precursor, whether

of lactate, which would necessitate in excess of 15 litres

lactate, propionate or citrate. These solutions have been

to counter severe acidosis in a calf. Furthermore, lactate

referred to as 'second generation' oral rehydration

must be metabolized to yield bicarbonate but the young

solutions. While these solutions are very effective in

calf cannot fully utilize D lactate in the racemic mixture.

meeting the primary objectives of correcting hypo-

Hypertonic (7.2%) saline solutions are not indicated in

volaemia, moderate metabolic acidosis (and associated

the treatment of acidotic calves because of their lack

hyperkalaemia) and hyponatraemia, they fail to
of alkalinizing effect, although successful treatment of
provide sufficient calorific support and treated calves
mild acidosis in experimental calves has been reported
lose considerable body condition during treatment
using a hypertonic saline solution containing sodium
when milk is withheld from the diet.

bicarbonate (Dupe et al., 1993).

‘Third generation’ oral rehydration solutions contain
The administration of sterile isotonic saline solutions
much higher concentrations of glucose (up to 375 mmol/l)
spiked with bicarbonate described above has obvious
and sodium (up to 133 mmol/l) to counter the energy
advantages over non-sterile fluids made using tap
deficit which results when milk is withheld from the calf’s
water; however, solutions prepared using small water
diet. Such hypertonic solutions do not worsen diarrhoea
purification plants are reported to work well under
due to osmosis or produce hypernatraemia and, when
practice conditions. A typical mixture of salts available
compared to standard oral rehydration solutions, these

in the UK (Electrolyte ED; Vetoquinol Ltd) added to 'third generation' solutions have reduced the liveweight loss which results from calf diarrhoea.

5 litres of water yields 141 mmol/l sodium, 4 mmol/l potassium, and 35 mmol/l bicarbonate, which can then be spiked with additional sodium bicarbonate. Extra generation') include the addition of the amino acid glutamine to promote enteric sodium uptake and sustain villus form and function. A high-calorie oral rehydration solution with glutamine was more effective in correcting plasma, extracellular fluid and blood volume to the 175 mmol already present in the 5 litre solution than solutions without (one WHO-type solution and (Grove-White, 2000).

two high-glucose but glutamine-free solutions) using an *Escherichia coli* model (Brooks et al., 1997).

Oral rehydration solutions: There are a large number
In summary, an oral rehydration solution should be
of oral rehydration solutions available but all utilize
judged on the composition of the solution in mmol/l and
the active glucose/sodium-linked gut transport system
not on a dry matter percentage basis. The sodium

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concentration should not exceed 130 mmol/l. The bicar-
increased glucose content as energy source should be
bonate concentration (usually as a precursor such as
chosen if the calf is fed only oral rehydration solution
citrate, acetate or propionate) should be in the region
for more than 24 hours (third or fourth generation oral
of 80 to 120 mmol/l. The role of glycine may be over-
rehydration solutions).

stated with respect to sodium transport but it aids
palatibility. High-energy-containing oral rehydration

Antibiotics: Antibiotics are frequently employed in the solutions, with glucose
concentrations up to 375 mmol/l,

treatment of diarrhoeic calves despite the fact that
limit liveweight loss during diarrhoea and should be

significant bacterial causes are limited to age-specific
considered if milk is withheld for more than two days.
periods within the first four days (enterotoxigenic *E.*
The addition of glutamine to the oral rehydration solu-
coli) and at over three weeks old (*Salmonella* spp.).
tion appears to offer numerous advantages over other
Unsupervised widespread use of antibiotics prescribed
solutions (Brooks et al., 1997).

by veterinary surgeons for the treatment of calf diar-
rhoea has been claimed to result in an increased preva-

Treatment with oral rehydration solutions: As detailed lence of muliple resistant
enteropathogens in humans,

above, there is an almost bewildering list of oral rehy-
which cautions against their use except for specific
dration solutions which can be used to treat diarrhoeic
bacterial causes (see also p. 210).

calves, each supported by its own experimental and
Enterotoxigenic E. coli do not display a high degree
clinical database. For veterinary practitioners presented
of antibiotic resistance and the commonly used anti-
with a recumbent calf with severe metabolic acidosis,

biotics such as trimethoprim-sulpha are effective. Such the most critical treatment is appropriate intravenous calves do not develop a bacteraemia and oral antibiotic fluid therapy. No oral rehydration solution will correct administration is preferable. Many *Salmonella* spp. the moderate to severe base deficit in these profoundly infections result in bacteraemia with localization in the weak and recumbent calves. Within six to eight hours lungs, joints and growth plates. Antibiotic selection of the start of intravenous fluid therapy the calf should should be guided by bacterial isolation and antibiotic be much improved, with return of the suck reflex. This sensitivity testing. Improved biosecurity and hygiene, clinical improvement indicates that the acidosis has and possibly dam vaccination, should all be considered been largely corrected, but to counter ongoing losses 1 to control salmonellosis in neonatal calves, thereby litre of a high-calorie oral rehydration solution with a reducing the need for antibiotic therapy. The use of high alkalinizing ability should be offered eight times fluoroquinolone antibiotics to treat salmonellosis in

daily (note: this may be twice the recommended rate calves should be carefully considered and employed detailed in the product data sheets). The presence of a only where there is no alternative antibiotic.

vigorous suck reflex is the best indicator that the calf is The use of antibiotics in rotavirus diarrhoea is not recovering and is a more important factor than the con- indicated, but care must be taken not to overlook focal tinued production of fluid faeces.

bacterial infections. Indeed, it is possible to overlook The calf should be returned to a milk diet as soon as early cases of bacterial polyarthritis and meningoen- possible, and this should be a complete change. The cephalitis, but such infections generally appear in calves milk must not be diluted with an oral rehydration solu- less than six days old whereas rotavirus infection tion. Typically, when milk is added to the diet, the calf causing recumbency and stupor occurs in calves more will continue to have profuse diarrhoea for a few days than eight days old with a history of diarrhoea for the but it will possess a strong suck reflex, be bright and

past two days or more.

alert with no signs of weakness. Assessment of the calf's

Following intravenous fluid therapy it is important

recovery based upon demeanour, activity and appetite

that the catheter is removed within 24 hours to reduce

is more important than the negative aspect of the

the risk of thrombophlebitis; parenteral antibiotics

continued production of fluid faeces.

should not be necessary simply because the jugular vein

Traditionally, milk was withheld from diarrhoeic

has been catheterized.

calves until solid faeces were passed, which often did

not occur until after four or more days of administra-

tion of only oral rehydration solutions. This calorific

Non-steroidal anti-inflammatory drugs: Non-steroidal

deprivation led to considerable body condition loss and

anti-inflammatory drugs (NSAIDs) such as flunixin

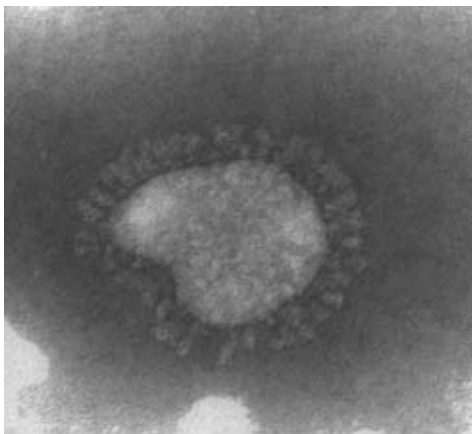
a protracted convalescence. Some clinicians recom-

me glumine and ketoprofen have been recommended

mend that milk and an oral rehydration solution are

for diarrhoeic calves but have not been fully evaluated

alternated every two to four hours during the early convalescent phase, thereby largely overcoming the calorie deficit but still retaining the benefits of the oral rehydration solution. Oral rehydration solutions with an NSAID must not be given NSAIDs.



Calf Diarrhoea • 199

Protectants: Protectants, e.g. kaolin and pectin mixtures, are used as an adjunct to treatment but their value between batches, steamed, disinfected and allowed to dry, in serious cases is doubtful.

Disinfectants that have been reported to be effective against rotavirus are 0.25 per cent formaldehyde, 2 per

Convalescence

cent phenol, 1 per cent sodium hypochlorite, 0.25 per

Convalescence from rotavirus-induced diarrhoea is

cent propiolactone, quaternary ammonium compounds

often protracted because of physical loss of absorptive

and iodophores. Mixing calves of different ages should

capacity of the small intestine. The villus epithelial cells

be avoided and stress should be limited by staggering

are lost, resulting in stunted villi (Fig. 14.11). These

management interference such as castration, disbud-

lining cells are initially replaced with undifferentiated

ding and changing of accommodation.

squamous and cuboidal cells, and only later replaced by

specialized absorptive columnar cells. Extended oral

Bovine coronavirus

administration of certain antimicrobials may prolong

this delay to normal absorptive capacity.

The agent

Bovine coronavirus (BCV) is a member of a group of

Management

viruses with a characteristic morphology seen in the electron microscope (Fig. 14.13). The virions, which contain single-stranded polyadenylated RNA, are once it has been diagnosed, should be based on the twin pleomorphic spherical particles 70 to 220 nm in diameter with a corona of widely spaced club-shaped surface projections (peplomers), which are 20 nm long. Bovine coronavirus has two types of peplomer of different length. There is only one serotype of bovine coronavirus. The management of an outbreak of rotavirus diarrhoea, should be based on the twin pleomorphic spherical particles 70 to 220 nm in diameter with a corona of widely spaced club-shaped surface projections (peplomers), which are 20 nm long. Bovine coronavirus has two types of peplomer of different length. There is only one serotype of bovine coronavirus. Good hygiene will not be successful in controlling spread of infection because coronavirus.

rotavirus is endemic and highly infectious, but it will help to reduce the build-up of infection throughout the calving period and thus reduce the infectious challenge

Signs

to which calves are exposed; several studies have

Calves usually become infected with coronavirus when recorded beneficial results from good hygiene.

they are between one and three weeks old, although

Unlike dairy herds, most beef cattle have a seasonal disease may occur in calves up to three months of calving pattern with over 90 per cent of the cows calving age. Transmission of infection is by the faecal–oral during a nine week period, which presents problems route. Calves develop diarrhoea in a manner virtually with stocking levels in farm buildings and calving accommodation. Beef cattle should calve outdoors wherever possible. Newly-calved cows and their calves should be moved to clean pastures as soon as possible after birth. The stocking density on these pastures should be kept as low as practicable to prevent environmental contamination and build-up of infection.

Problems with rotavirus diarrhoea usually appear during the second half of the calving period and are often associated with adverse weather conditions during early spring or late autumn, when young calves

are crowded in sheltered areas of the field facilitating build-up of infection. Infection builds up rapidly in housed beef herds where poor building design and lack of sufficient straw bedding material aggravate overstocked conditions. The provision of a dry, well-bedded creep area for calves may reduce the level of challenge but is not sufficient to prevent disease.

To assist good hygiene, calving accommodation and

Fig. 14.13

Transmission electron micrograph of a single particle of bovine coronavirus. The particle has an irregular shape cleaning and with adequate drainage. An 'all-in, all-out' policy should be adopted where possible, especially in

Bridger, Royal Veterinary College, University of London.)

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identical to rotavirus; studies of experimental inoculations suggest that the incubation period is 20 to 30

The existence of bovine coronavirus excretion by clini-

hours. Generally, coronavirus diarrhoea is more watery than normal calves is recorded, although it is not seen and of greater severity than rotavirus diarrhoea, leading as commonly as is rotavirus.

more rapidly to dehydration and acidosis. The severe diarrhoea also leads to substantial losses of sodium,

Management

potassium, chloride and bicarbonate ions. Fluid yellow faeces are passed at first and these turn to a yellow

An outbreak of BCV diarrhoea should be managed in the liquid containing milk clots and mucus.

same way as an outbreak of rotavirus diarrhoea; that is, aim to reduce exposure to infection and enhance resistance to infection by vaccination of the dam and ensuring

Pathogenesis

adequate passive antibody transfer (see p. 1006).

Bovine coronavirus infects mature enterocytes located

Sick calves should be moved to isolation facilities to

on the surface of villi and epithelial cells of the upper

help prevent a build-up of infection and to facilitate the

respiratory tract. The small intestine is severely

provision of rehydration therapy. Diarrhoea induced damaged due to sloughing of infected cells, especially by BCV is often more watery than that caused by the ileum, and cells on the surface and in the crypts of rotavirus, possibly because the colon is less able to the large intestine are also killed. Infection of the upper absorb water because it is damaged by the infection. respiratory tract does not appear to cause lesions. Virus The watery diarrhoea is likely to lead to severe particles are assembled in the cytoplasm by a budding dehydration and rehydration therapy as outlined for process through the rough endoplasmic reticulum. They rotavirus is indicated (see pp. 195–6).

are subsequently transported through and accumulate in the Golgi complex and are released from the cell by

Calici-like virus (Newbury agent)

lysis. The range of lesions seen in the small intestine is identical to those produced by rotavirus and repair can Particles of calf calici-like virus measure approximately occur rapidly because small intestinal crypt cells are 33 nm in diameter and have an indefinite feathery

largely unaffected. Virus damage to colonic crypt cells outline with dark hollows on the surface. Some parts results in atrophy of the mucosal ridges and dilated crypts containing dead exfoliated cells. The virus was first detected in faeces of calves with diarrhoea and the by which the intestinal damage caused by bovine coronavirus results in diarrhoea are thought to be the same as described for rotavirus. The presence of severe unweaned calf, although a gnotobiotic calf aged 60 days lesions in the ileum, caecum and colon, areas of the was susceptible to infection and disease. In gnotobiotic small and large intestine that absorb water, may calves, clinical signs of enteric disease are identical to account for the more watery nature of the diarrhoea those seen in rotavirus infections and the lesions are seen in BCV infections. It is not clear whether or not comparable to those produced by rotavirus and BCV. strains of BCV vary in virulence, as is seen in bovine

Two antigenically distinct isolates of Newbury agent rotaviruses.

have been identified and infection with either virus did not protect against clinical illness following infection with the other virus three weeks later. There are no spe-

Prevention

cific diagnostic tests for the calf calici-like viruses, other There are two inactivated vaccines available in the UK

than electron microscopic examination of faeces concentrated and purified by ultracentrifugation. They to provide protective antibody in colostrum and early might be suspected as the aetiological agent in cases milk. The vaccines are administered 12 to 13 weeks that appeared clinically similar to rotavirus, but are before the anticipated calving date.

negative for rotavirus and other enteropathogens.

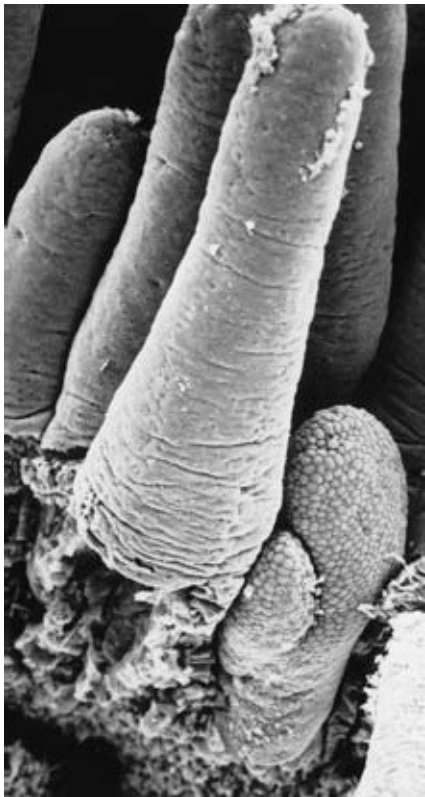
Calves with diarrhoea caused by a calici-like virus may be managed as though they were infected with rotavirus.

Diagnosis

Bovine coronavirus is universally present on all cattle

Astrovirus

farms, as indicated by the presence of antibodies in all adults. Diagnosis of coronavirus disease, like that of Astrovirus is a descriptive name for viruses with a star-like pattern on their surface in the electron microscope. virus antigen in intestinal tissue or faeces. Assays using They have a diameter of 28–30 nm and contain single-ELISA have been developed for detection of bovine stranded RNA. Astroviruses have also been detected in



faeces of lambs with diarrhoea where the virus infected

Breda virus

enterocytes causing mild villus stunting in the mid small

This virus was first described as causing calf diarrhoea

intestine. Astroviruses have been detected in calf faeces

in the USA in 1982. Lesions were similar to those pro-

in association with diarrhoea but experimental infec-

duced by coronavirus. Jejunal and ileal villi were

tion of gnotobiotic calves with astrovirus did not cause

stunted and there were crypt abscesses in the small

diarrhoea. Nevertheless, antibodies to astrovirus are

intestine and particularly in the colon. There were areas

widespread in cattle sera, having been found in 11 out

of necrosis of the surface epithelium of the colon.

of 22 herds in the UK and in 30 per cent of cattle sera

There are no tests available commercially which

examined in the USA. Recently, astroviruses were

detect Breda virus in faeces. Electron microscopy of

shown to infect and damage the specialized epithelium

faeces and thin sections through ultracentrifuge pellets

of the dome villi (Fig. 14.14) in the Peyer's patches of

may be used in the research laboratory and, because all calves. These calves did not develop diarrhoea, but isolates of Breda virus possess common antigens, anti-severe diarrhoea occurred in mixed infections of astrobodies to these can be used in an immunofluorescent virus and rotavirus or astrovirus and Breda agent. It is test of intestinal sections and in an ELISA test. Ex-probable that astrovirus are not pathogenic on their amination by ELISA of over 200 faeces from UK calves own, but may increase the severity of the disease produced by other enteropathogenic viruses. There are no in a separate study, antibodies to Breda virus were routine tests to detect astroviruses in faeces, but in the detected in 55 per cent of cattle sera.

research laboratory they can be identified by electron microscopic examination of faeces concentrated by centrifugation.

Escherichia coli

Three groups of *E. coli* have been identified that appear to cause diarrhoea in calves. Strains that elaborate a

heat-labile enterotoxin or a heat-stable enterotoxin are called the enterotoxigenic *E. coli* (ETEC). A second collection of strains exists that colonize the small intestinal mucosa and cause diarrhoea, but do not elaborate enterotoxins. Characteristically, these strains attach closely to the luminal surface of enterocytes, often in cup-shaped depressions or on cytoplasmic protrusions, described as 'pedestals'. This attachment of bacteria to the enterocyte surface usually results in the microvilli being effaced and the lesion has been named the 'attaching and effacing' (AE) lesion (Fig. 14.15). Comparable strains cause diarrhoea in children and are called enteropathogenic *E. coli* (EPEC). It is appropriate to use this term for equivalent calf strains, but care is required because the term enteropathogenic *E. coli* has been used in the veterinary literature for many years to describe strains associated with diarrhoea, regardless of their pathogenic mechanism.

A third group of strains colonizes the surface of the large intestine and causes a mild dysentery. These strains also cause AE lesions but because they cause

blood to be lost into the lumen of the large intestine,
they are called enterohaemorrhagic *E. coli* (EHEC);
once again comparable strains cause a similar disease
in children. Some EPEC and EHEC, but not all,
produce a toxin that kills Vero cells in vitro and is called Verocytotoxin (VT) or
Shiga-like toxin (SLT). This

Fig. 14.14

Scanning electron micrograph of calf small intestine

toxin does not cause AE lesions, but is probably

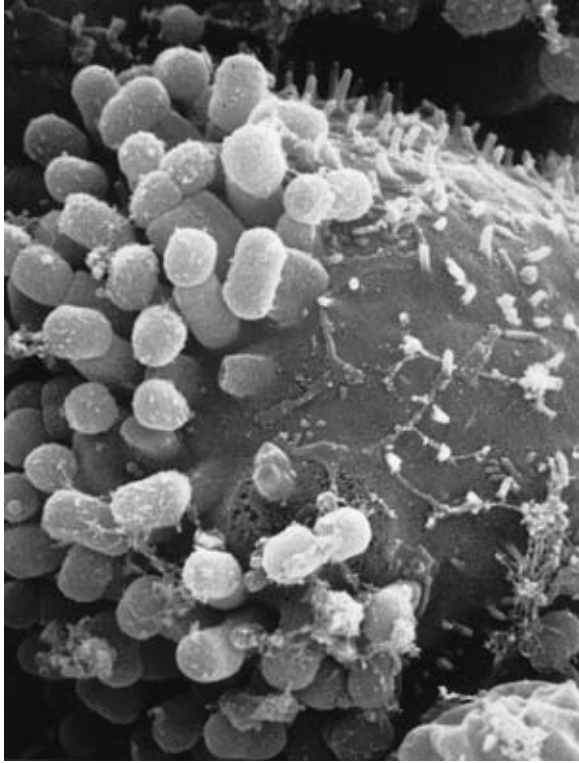
illustrating absorptive villi and a dome villus covered by a characteristic
epithelium, which is thought to be specialized for

involved in the pathogenic process. *Escherichia coli* that antigen uptake and
which has been shown to be susceptible to

produce VT are referred to as VT+ *E. coli* or VTEC.

infection and damage by bovine astroviruses.

VTEC have been isolated from calves with diarrhoea,



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Enterotoxigenic E. coli possess two virulence attributes (determinants) that distinguish them from non-pathogenic strains: ability to adhere to the mucosal surface of enterocytes and, as mentioned previously, ability to produce enterotoxins (see Fig. 14.4).

Adhesion is mediated by filamentous protein structures called fimbriae, sometimes called pili or adhesins, which bind to specific receptors on the enterocyte cell membrane. Adhesion mediated by fimbriae does not bring the bacterium in close contact with the enterocyte

luminal surface as is the case of EPEC and EHEC.

The microvilli are unaltered and ‘cups’ and ‘pedestals’ are not seen. The presence of receptors is affected by age, and susceptibility to infection is greatest in the newborn calf when expression of receptors is greatest. These fimbriae are antigenic and so many of them are known by their antigenic name (e.g. K99, F41). Their adhesive ability allows ETEC to overcome the peristalsis of the small intestine by sticking to the mucosal surface. The adhesion antigens commonly found in ETEC of calves are K99 and F41; they often occur together but may be present independently. K99+ strains are also capable of inducing diarrhoea in pigs, foals, lambs, goats and possibly other ruminants. Adhesion of the bacterium to the enterocyte surface confers another advantage to the bacterium because entero-

Fig. 14.15

Scanning electron micrograph of an enterocyte in toxins are released close to their receptor sites. The the colonic mucosa of a gnotobiotic calf inoculated experimentally of ETEC to produce fimbriae and enterotoxins

tally with an enterohaemorrhagic *E. coli* (strain S102–9). Bacteria is determined by genes that are usually carried on a

plasmid. Bacteria are attached closely to the surface of the enterocyte, often single plasmid and thus occur together. It is adequate,

on 'pedestals' (arrow) and the microvilli have been effaced.

therefore, to diagnose ETEC infection by detecting

either the enterotoxin or the adhesin.

Detection of adhesins forms the basis of the diag-

nostic tests for ETEC because they are easier to detect

but they may also be present in the faeces of healthy

calves. Special culture media may be

required to encourage ETEC to express adhesins

be as common in the faeces of healthy calves as in the

faeces of calves with diarrhoea. Therefore, designation

by bacteria in vitro, although they are readily produced of an *E. coli* as a VTEC may not necessarily define it as by bacteria growing in the gut. Once expressed they can

an isolate capable of causing diarrhoea and demon-

be detected using specific antisera in agglutination,

stratification of a VTEC in the faeces of a calf or calves with

haemagglutination or ELISA tests.

diarrhoea does not constitute a diagnosis of the cause

It is helpful to understand the mechanisms of absorption of the diarrhoea.

tion and secretion in the small intestine before considering how the enterotoxins produced by ETEC cause diarrhoea. Absorption occurs through the activity of Enterotoxigenic E. coli

the mature villus enterocyte. Sodium ions are trans-

Enterotoxigenic E. coli most commonly cause diar-

ported out of enterocytes through the basolateral cell

rhoea in calves under four days old. In experimentally

membranes into the basolateral spaces by an energy-

infected newborn calves the incubation period is 12 to

dependent mechanism, the sodium pump. This creates

18 hours. Calves are depressed and anorexic and rapidly

a concentration gradient extending from the lumen into

dehydrate and die. In calves ETEC diarrhoea can be

the enterocyte. Sodium ions, followed by chloride ions,

diagnosed clinically because it causes disease in very

diffuse along the concentration gradient from the

young calves and because it produces a very profuse

lumen through the microvillous surface, assisted by a and much more watery diarrhoea than any other of the brush border carrier system. The osmolality in the calf enteropathogens.

basolateral spaces increases and water passes from the
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lumen along the osmotic gradient (see Fig. 14.2). There because adhesion is an important component of the are several carrier systems that assist the entry of pathogenic process.

sodium ions; one system does not utilize the products It is possible to recognize clinically an outbreak of calf of digestion, another utilizes glucose, which is specific diarrhoea caused by ETEC because of the severity and cally absorbed through the luminal surface of entero-age group of calves affected. An outbreak characterized cytes and each molecule of glucose carries with it a by the rapid onset of severe, very watery diarrhoea, molecule of sodium. Chloride ions and water follow and which quickly causes dehydration and collapse and this flow of water traps additional sodium and chloride

which is frequently fatal and occurs in calves that are
ions by solvent drag (Fig. 14.2). Carrier systems for
less than 96 hours old, is likely to be caused by ETEC.
amino acids and citrate also transport sodium ions and
In some circumstances, calves may die before there is
water through the intestinal mucosa. These carrier
evidence of profuse diarrhoea. There is rapid and severe
systems, particularly the glucose carrier system, are par-
dehydration. The calf quickly becomes recumbent.
ticularly important in oral rehydration therapy. Secre-
Sequestration of fluid in the abomasum and intestines
tion occurs from crypt cells; sodium passes into the
gives the abdomen a bloated appearance which 'sloshes'
intestinal lumen taking chloride and bicarbonate ions
on succussion. The rectal temperature may be elevated
and water with it (Fig. 14.3). Cyclic-AMP stimulates this
in the early stages but rapidly falls to subnormal.
secretory activity.
Affected calves are rarely acidotic. Faecal bacteriology
The heat-stable enterotoxin (ST) is the enterotoxin
will confirm the diagnosis. ETEC diarrhoea is not

usually produced by ETEC strains from calves.

common in the UK (less than one per cent of calf diar-

*Two forms of ST occur (STA, and STB), but only STA
rhoea outbreaks), but outbreaks have been associated
occurs in calves. Both forms stimulate guanylate
with poor housing and management at calving.*

cyclase activity within enterocytes, causing increased

*There are a number of possible causes of recumbency
levels of intracellular cyclic-GMP, which inhibits
in neonatal calves which must be excluded from the
absorption by villous enterocytes but has no effect on
differential diagnosis list, including congenital heart
secretion.*

defects, rupture of liver/spleen following dystokia and

*The heat-labile enterotoxin (LT), rarely produced by
bilateral femoral nerve paralysis. Atresia coli/ani cases
calf ETEC strains, is made up of two subunits. Subunit
present with increasing abdominal distension, progres-
A stimulates adenylate cyclase activity within entero-
sive weakness and dehydration leading to recumbency.
cytes. Subunit B assists entry of subunit A into entero-*

Septicaemia and bacterial meningoencephalitis are also
cytes. Adenylate cyclase raises the levels of cyclic-AMP
commonly encountered in calves less than four days
in mature villous enterocytes and crypt cells. In mature
old. Congenital neosporosis has also recently been
villous enterocytes, cyclic-AMP inhibits the independ-
reported as a cause of recumbency in neonatal calves.
ent pathway for the absorption of sodium ions and
The immediate response must be rapid isolation and
therefore of chloride and water (see Fig. 14.5). In crypt
treatment of sick calves by rehydration with an elec-
cells, it stimulates sodium secretion, which takes with it
trolyte solution. Acidosis is not a feature of diarrhoea
chloride and water (see Fig. 14.6).

in calves less than six days old and spiking intravenous
Although the main mechanism for the pathogenesis
fluids with bicarbonate is not indicated in calves with
of diarrhoea is attributed to enterotoxins, which do not
enterotoxigenic E. coli infections. Rehydration may
cause visible damage to the gut, experimental infections
be given by intravenous infusion if the calf is unable

of gnotobiotic and conventional calves, both colostrum to feed; intravenous infusions may be changed to oral fed and colostrum deprived, have revealed a pathology administration as the calf recovers strength. Less associated with ETEC infections that probably conservatively dehydrated calves may be treated only by oral tributes to the development of diarrhoea. Villi become rehydration. The biochemistry of oral rehydration stunted and fused together and the enterocytes change has been discussed earlier (pp. 197–8). Oral antibiotics shape from columnar to cuboidal. These cells are probably should be administered to all calves at birth in the face ably immature enterocytes and these changes indicate of an outbreak until the vaccine administered to all an increased rate of enterocyte loss.

remaining pregnant cattle can provide protective Immunoprophylaxis against ETEC-induced diarrhoea is based on the use of immune colostrum. Dam immunoglobulins in the colostrum. Antibiotic resistance is not a problem in such outbreaks thus antibiotic vaccination is used to stimulate high levels of antibody

selection is not critical to a successful outcome. Fluid in the colostrum and this approach is particularly successful because calves are susceptible to this disease for only the first few days of life when colostrum is fed. from antibiotic therapy.

Vaccines containing the K99 fimbriae, or dead bacteria Following immediate management of an outbreak with the K99 antigen expressed on the surface, are used with fluid therapy and antibiotics, it is essential to

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introduce dam vaccination (see p. 1006). It should be is little risk of spinal meningitis. Selection of a narrow possible to eliminate the infection from the herd with gauge hypodermic needle, and removal of only 1 to 2 the use of antibiotics, dam vaccination and good ml of CSF, will greatly reduce any risks associated with hygiene. ETEC with the K99 antigen are absent from sudden release of CSF pressure.

most farms and disease prevention should concentrate

on exclusion of the infection by good herd biosecurity.

Campylobacter spp. (see p. 852)

The related campylobacters, Campylobacter jejuni and

Enteropathogenic E. coli , enterohaemorrhagic

E. coli, have been recognized recently as important

E. coli and verocytotoxin-producing E. coli

enteric pathogens in humans. They are the most

common cause of diarrhoea in developed countries and

There are no specific clinical signs of diarrhoea caused

they are one of the three most common causes of

by EPEC and VTEC although a yellow watery diar-

human diarrhoea in developing countries; E. coli and

rhoea has been reported. The mean age of calves with

rotavirus are the other two enteropathogens of major

diarrhoea caused by EHEC was 15 days. Calves main-

importance. Recognition of the importance of campy-

tained a normal appetite and did not develop pyrexia,

lobacters followed marked improvements in culture

but a mild diarrhoea containing blood was seen. In pro-

techniques that improved isolation and identification

longed cases there was dullness, signs of abdominal

*from faeces. They have been isolated from healthy and
pain, dehydration and weight loss.*

*diseased domestic animals and poultry for many years
Although the EPEC, EHEC and VTEC are capable
and, not unexpectedly, the most common sources of
of causing diarrhoea in calves and have been associated
infection in human outbreaks are fresh poultry, minced
with outbreaks of disease, it is difficult to diagnose their
meat and unpasteurized milk.*

*specific involvement. Presence of frank blood in the
The recognition of animal products as the source of
faeces is suggestive of EHEC, especially if salmonellas
human infection has prompted a reassessment of the
and cryptosporidia are not isolated from faeces. The
importance of *C. jejuni* and *C. coli* in causing enteric presence of these
enteropathogens will be suggested by
disease of farm animals. Since the 1930s, campylobac-
the failure to find other agents and possibly by the
ters have been thought of as the cause of an enteric
finding of *E. coli* in association with the mucosa of the disease of housed adult
cattle in the winter-syndromes
small or large intestine.*

referred to as 'winter dysentery', 'winter scours', 'vibronic enteritis'. The above association has, however, been questioned and more recently a coronavirus has

Clinical signs of septicaemia

been implicated as the cause of winter dysentery (see

In many situations it can prove difficult to differenti-

p. 852). The association of campylobacters with calf

ate the cause of the calf's depression, lethargy and

diarrhoea has been examined in experimental infec-

weakness between profound acidosis or early

tions and in case-control studies of field outbreaks. The

septicaemia/bacterial meningoencephalitis. Most septi-

results have shown that *C. jejuni* is present on all farms caemic calves are less than six days old and this age

and most case-control studies have failed to demon-

prevalence differs from the typical occurrence of viral-

strate an association between excretion of this organ-

induced diarrhoea and acidosis (more than eight days

ism and calf diarrhoea. In the majority of experimental

old). Septicaemic calves usually present with episcleral

studies, inoculation of calves with *C. jejuni*, even using injection, toxic mucous membranes and evidence of

high doses, resulted in colonization without diarrhoea.

multi-organ system involvement such as joints, respira-

Campylobacter jejuni may be detected in up to 80 per

tory tract and eyes. Pyrexia above 39.5°C is not a con-

cent of weaned calves, indicating that isolation of this

sistent finding in septicaemic calves while diarrhoea is

organism from calves with diarrhoea does not consti-

often only present during the terminal stage of illness.

tute a diagnosis. *Campylobacter coli* is less commonly Lumbar cerebrospinal fluid (CSF) collection and

isolated from cattle and experimental inoculations and

visual inspection of the turbid sample on farm enables

case-control studies indicate clearly that it is non-

immediate diagnosis of bacterial meningoencephalitis

pathogenic. *Campylobacter fetus* subsp. *fetus* and *C.*

and appropriate aggressive antimicrobial therapy

hyointestinalis are also commonly isolated from cattle (Scott & Penny, 1993).

This sampling procedure is rec-of all ages, but most evidence suggests that they are not

ommended in those cases where the clinician is unsure

a cause of intestinal disease.

of the diagnosis. On-farm collection of lumbar CSF in

depressed calves is a simple procedure which only

Cryptosporidium parvum (see p. 286)

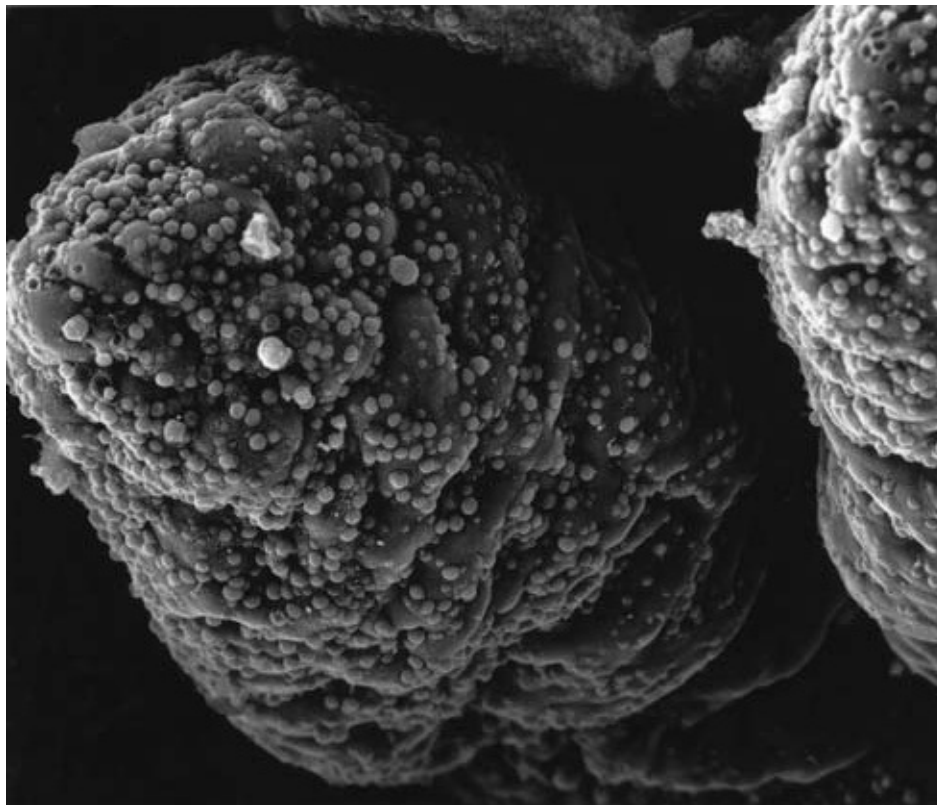
requires a knowledge of the bony landmarks and an

appropriate length hyopdermic needle without stylette

Cryptosporidium parvum is an enteric coccidia recog-

(see p. 896). With appropriate aseptic precautions, there

nized commonly in calves and lambs and is a zoonosis.



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The organisms are spherical or ovoid parasites that

similar to that seen with rotavirus and not similar to

adhere to the microvilli of enterocytes, particularly in

that seen in *Eimeria* spp.

the ileum, but also in the large intestine. Cryptosporidia
Cryptosporidiosis is seen in neonatal calves, usually
exhibit three important differences from other enteric
when they are aged one to two weeks (peak 11 days old),
coccidia: excreted oocysts are directly infective to new
at about the same time as they develop rotavirus diar-
hosts, cryptosporidia are not host specific so that infec-
rhoea. Most calves appear to become infected, but not all
tion can spread between mammalian species, including
develop diarrhoea. Subclinical infections are not thought
man, and finally they are unaffected by most existing
to be the result of passive protection by colostral anti-
anticoccidial drugs (see pp. 206, 1029).

bodies because experiments have shown that antibodies

The life cycle commences with the ingestion of
that are fed are not protective; subclinical infections
oocysts containing sporozoites, which become tropho-
remain unexplained. Experimental infections indicate
zoites. In the asexual phase of the life cycle the tropho-
an incubation period of two to five days and close associ-

zoites mature to schizonts containing eight merozoites, ation between occurrence of diarrhoea and excretion of which are liberated and infect new enterocytes to form oocysts. Depression and anorexia accompany the a second generation of merozoites. In the sexual phase, profuse watery green diarrhoea, which contains mucus macrogametes fuse with microgametes and give rise to and occasionally blood. Diarrhoea may be intermittent zygotes, which form oocysts. Sporozoites form within and lasts two to 14 days (usually about seven) and causes oocysts in the intestinal lumen or whilst they are still dehydration which often requires treatment with oral attached to the surface of enterocytes so that oocysts rehydration solutions. Morbidity is usually high and mor-excreted in the faeces are directly infective to new hosts tality low,although some outbreaks have been associated without an obligatory period of maturation outside the with high mortality.

host body. Consequently, infection can spread rapidly Infected mucosae (Fig. 14.16) are congested and villi within a group of calves and the pattern of spread is

are stunted in the ileum. Enterocytes in the small and

Fig. 14.16

Scanning electron micrograph

of the ileal mucosa of a calf naturally

infected with Cryptosporidium parvum. The

surface of the villi is heavily infected and

partially exfoliated enterocytes are present

at the tip of the villus.

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large intestines become cuboidal or squamous and

and before treatment commences and should be exam-

ined for rotavirus, coronavirus, Cryptosporidium and

cells and neutrophils. The lamina may become infil-

Salmonella spp. Samples from very young calves with

trated with mononuclear inflammatory cells, neu-

watery diarrhoea should be tested for K99+ E. coli.

trophils and eosinophils. Cryptosporidia appear to

Faeces should be collected from affected calves and

cause diarrhoea by destroying mature enterocytes, but

age-matched healthy calves on the farm, a minimum of

by an unknown mechanism. The population of mature four calves in each category. Faecal collection should enterocytes is reduced in size and numbers of immature continue if the outbreak persists to monitor for changes cells are increased. Mucosal lactase activity is markedly in enteropathogens being excreted.

depressed and there is reduced ability to digest and Where a problem persists, or no diagnosis has been absorb food. Intestinal secretion may be enhanced due reached, samples may be sent to a research laboratory to the increased numbers of secretory cells in the for examination by electron microscopy or for the mucosa. The self-limiting nature of natural and experi- detection of EPEC, EHEC and VTEC. A post-mortem mental infections and the widespread detection of anti- examination of a calf submitted alive to a laboratory bodies indicate that immune responses occur that are may help to elucidate the significance of results of protective. No further information is available on faecal examination and indicate the presence of other immunity.

agents not detected by faecal examination. Laboratory Cryptosporidiosis can be diagnosed by detection

ries are frequently asked to assist in the diagnosis of of oocysts in faecal smears but more reliable results the aetiology of calf diarrhoea outbreaks on the basis are obtained from stained gut sections. However, such of samples submitted from one or two calves only. material is rarely available due to the low mortality. Clearly this is pointless, because diagnosis of disease

Several staining methods are available, of which the caused by an endemic agent is based on the detection Giemsa stain, the modified acid fast method and of the agent in a higher percentage of the faeces of auramine are the most widely used. Alternatively, calves with diarrhoea than in healthy calves on the same mucosal smears of ileum may be stained with Giemsa farm. Where data are not available from healthy calves or histological sections of ileum stained by haema- on the same farm, results from other studies may be toxylin and eosin. The histological method is of little considered. Rotavirus and cryptosporidia have rarely

value if autolysis is advanced because the enterocytes been found in more than 50 per cent of randomly will have sloughed from the mucosal surface. The most selected healthy calves and coronavirus is usually reliable method is to make and stain smears of oocysts detected in less than 25 per cent. Figures above these concentrated from faeces using flotation techniques. values may be taken as a tentative diagnosis.

Management of an outbreak presents problems;

Salmonellas, which may be suspected clinically, are although a large number of chemotherapeutic agents often only detected qualitatively. Merely isolating these

have been tested for efficacy against *Cryptosporidium*, bacteria is relatively meaningless; quantitative bacteri-only halufiginone has been found to be effective. Most

ological techniques should be used to demonstrate workers recommend high levels of hygiene as the best excretion of large numbers of bacteria from calves with approach to control, but oocysts are extremely resistant diarrhoea. It is only worth looking for K99 adhesins to a variety of disinfectants, including iodophor, cresylic where the outbreak is suggestive of ETEC, i.e. if calves

acid, sodium hypochlorite, benzylkonium chloride, under four days of age are affected and there is severe sodium hydroxide and aldehyde-based disinfectants. watery diarrhoea.

Ammonia and formalin are effective disinfectants. The organism is susceptible to freezing and thawing and to temperatures over 50°C. Steam cleaning is strongly

Epidemiology

recommended.

On a farm basis, the infectious agents that have been associated with neonatal calf diarrhoea can be divided

Investigating an outbreak of

into three groups: those infections that are usually

calf diarrhoea

absent but may be introduced and cause an outbreak of

disease (ETEC, Salmonella spp.); the ubiquitous agents The investigation should start with a farm visit to

(rotavirus, coronavirus, and cryptosporidium) that are

examine the calves clinically and establish a picture of

invariably present on every farm; and those agents for

calf husbandry and of the outbreak. Enterotoxigenic E.

which inadequate epidemiological data are available

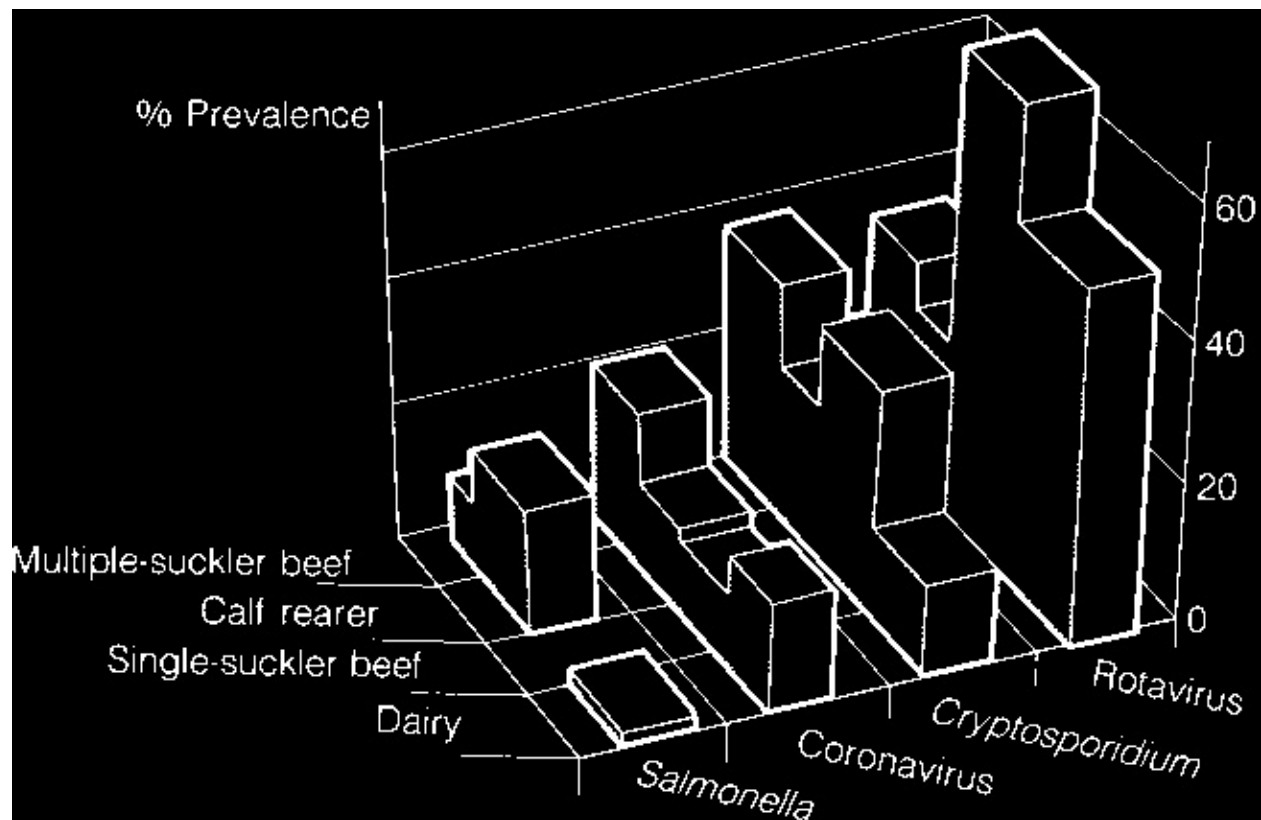
coli diarrhoea can be suspected on the basis of age of (Breda virus and VTEC). Although there is evidence to

affected calves. Faeces, not rectal swabs, should be col-

support the view that all the above infectious agents are

lected as soon as possible after the onset of diarrhoea

able to cause diarrhoea in calves, it is clear that their



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detected rarely in clinically normal calves and it was

Cryptosporidium

Rotavirus

Salmonella

invariably associated with disease, usually in outbreaks with high mortality. Results of two surveys show that

VT+ E. coli

diarrhoea caused by ETEC was uncommon in the UK

No diagnosis

at that time; this may still be the situation. Intermittent

Coronavirus

excretion of ETEC may occur throughout life, often

K99+ E. coli

unassociated with diarrhoea, making interpretation of diagnostic findings difficult and emphasizing the need

Calici-like virus

for quantitative bacteriology.

Fig. 14.17

Prevalence of pathogenic agents in calf diarrhoea.

These survey data have been reproduced by numerous monitoring organizations over the past 15 years which clearly indicates the important role of rotavirus and coronavirus in neonatal enteritis. Vaccination is highly effective against these viral causes of diarrhoea

but the need for annual vaccination and perceived high cost have resulted in many beef calves remaining unprotected.

Mixed infections

With the recognition of enteropathogenic agents and the development of diagnostic tests, it has been possible to look at outbreaks of diarrhoea and at individual animals to assess the importance of mixed infections.

Results of surveys of faeces of calves with diarrhoea indicate that although single infections may result in

Fig. 14.18

Correlation of farm type with prevalence of pathogens in calf diarrhoea. Reproduced from Morgan (1990).

diarrhoea, the likelihood of diarrhoea occurring increases with the number of enteropathogens present.

When 21 moribund calves with diarrhoea were examined in detail by necropsy, two or more presence in the intestinal tract does not inevitably lead enteropathogens were detected in 19 calves (Hall et al., 1988). The most common combination of

aetiology of calf diarrhoea.

enteropathogens revealed by these studies has been

The prevalence of these agents in calves with diar-

rotavirus and cryptosporidia and the way in which these

rhoea in southern Britain, as determined from 45

infections can overlap in rearing pens is illustrated in

outbreaks of calf diarrhoea, is shown in Fig. 14.17

Figs 14.19 and 14.20. Studies of distribution of infection

(Reynolds et al., 1986); another survey examined out-

of the gut mucosa with these two agents and the sever-

breaks in northern England and southern Scotland

ity of the lesions have shown that either agent, as a

(Snodgrass et al., 1986). Rotavirus was associated most single infection, might not cause sufficient intestinal

frequently with calf diarrhoea and was most common

damage to cause diarrhoea, but together they could. A

on dairy farms and single-suckler beef units (Fig. 14.18).

study of the intestinal pathology of normal calves that

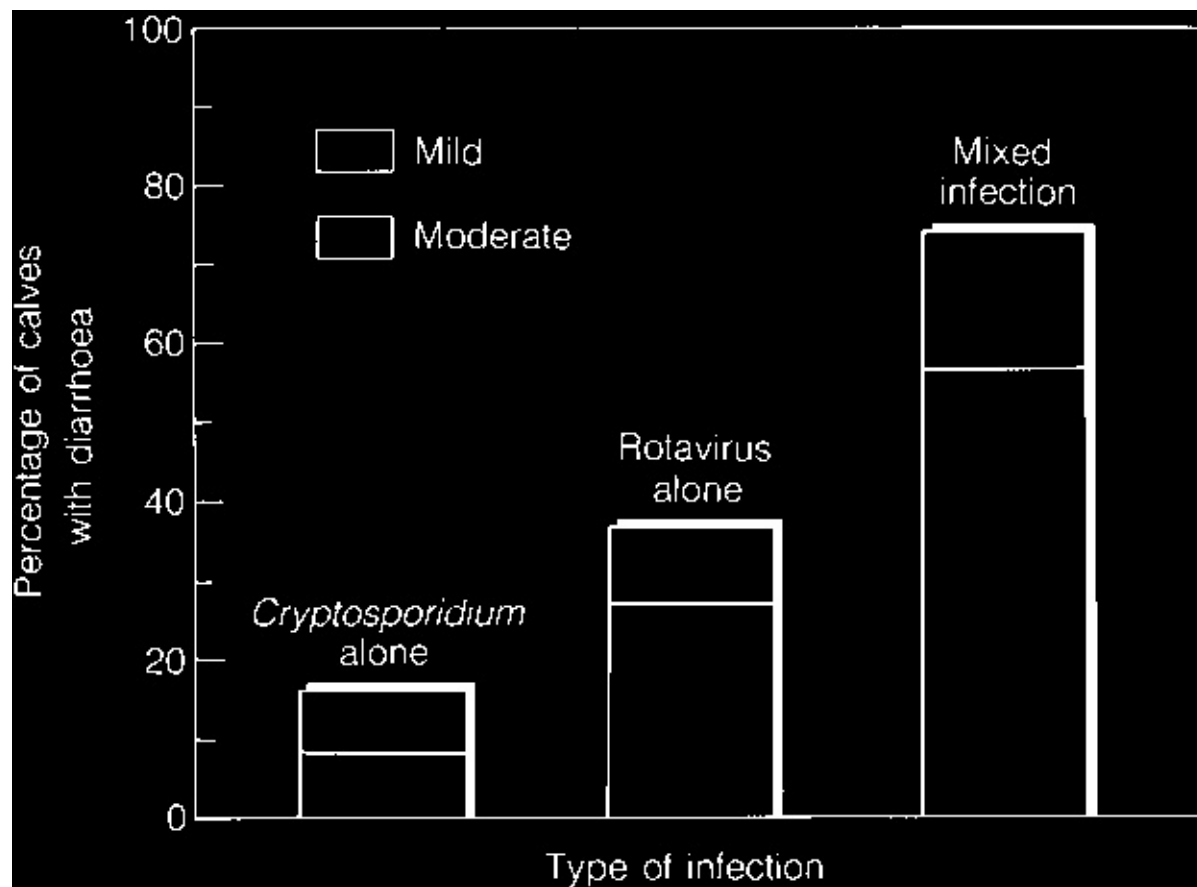
Cryptosporidia oocysts were associated with calf diar-

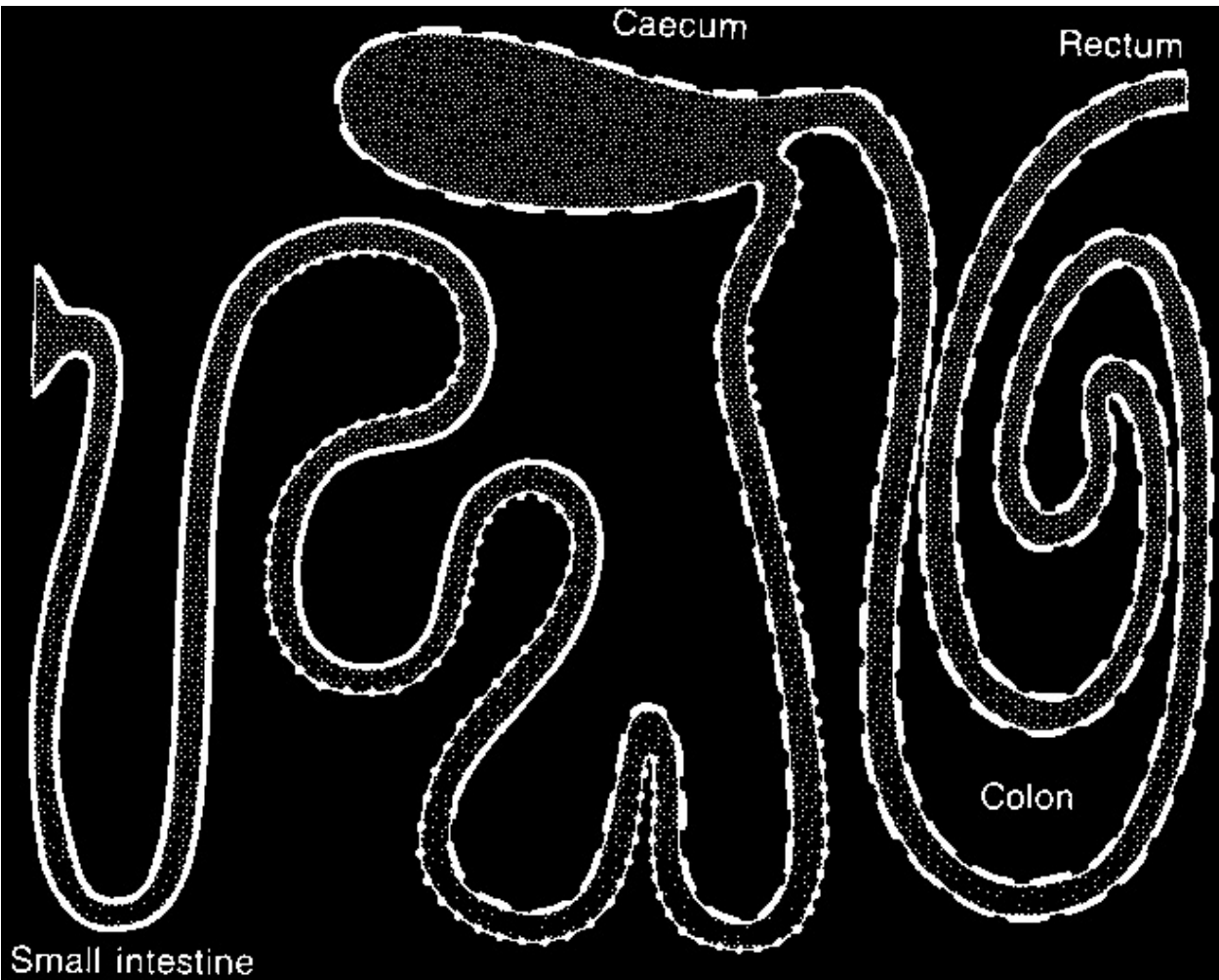
were excreting rotavirus showed that there were severe

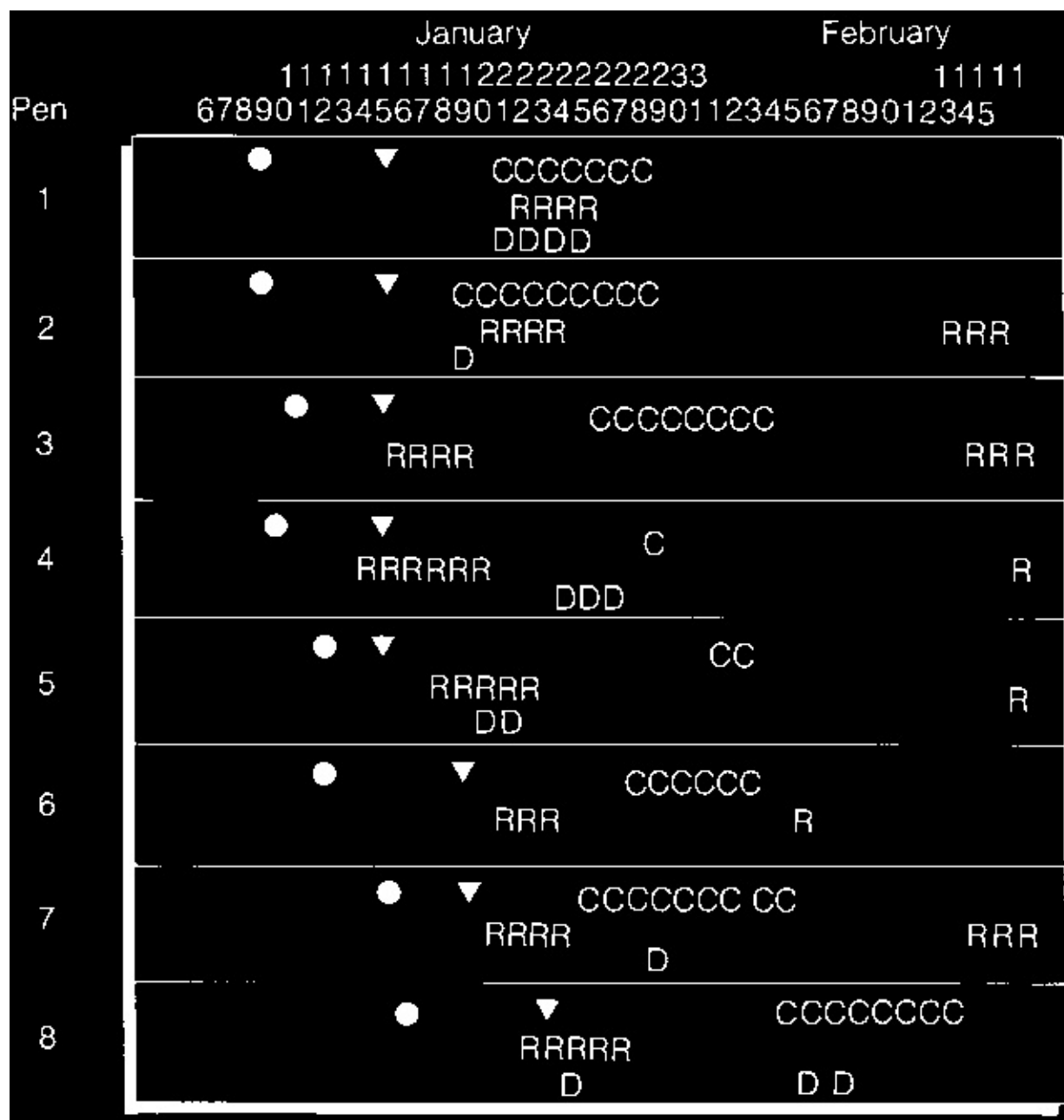
rhoea slightly less often than rotavirus and were more

lesions in the anterior small intestine and mild lesions common in beef suckler units than in dairy farms. In the mid small intestine, but the lower small intestine southern England, salmonellas occurred in 25 per cent and large intestine were normal (Reynolds et al., 1985). of outbreaks and were most often a problem for rearers Cryptosporidia, however, infect and damage the of calves bought in from markets. Enteropathogens lower small intestine and the large intestine, so that in were not detected in 31 per cent of faeces of calves with a combined infection the cumulative damage would be diarrhoea. Rotavirus and cryptosporidia were excreted sufficient to cause diarrhoea (Fig. 14.21). Similarly, if by up to 50 per cent of normal calves, and on the basis simultaneous infection with coronavirus and either of this evidence an outbreak of rotavirus-induced or rotavirus or calici-like virus occurred there would cryptosporidia-induced diarrhoea should only be diagnosed if more than 50 per cent of calves with diarrhoea damages the lower small intestine and large intestine,

are excreting either enteropathogen. Coronavirus was
whilst the latter infects and damages the upper small







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Fig. 14.21

Line drawing illustrating the small and large intes-

tines of the calf and indication of those parts susceptible to infection by rotavirus (. . .) and those susceptible to infection by Cryptosporidium spp. (---). Reproduced from Hall (1989).

Fig. 14.19

The spread of rotavirus and Cryptosporidium

between calves in adjacent pens. (糞) Day of birth, (糞) day of

Many of the recognized enteropathogens infect

move to rearing pens. C, Cryptosporidium excretion; R, rotavirus and damage the small intestine and coronavirus, Breda

excretion; D, day of diarrhoea. Reproduced from Morgan (1990).

virus, EHEC and cryptosporidia also damage the large

intestine. However, assuming that diarrhoea is more

likely to occur when both the small and large intestines

are damaged, there is a need to study the infectious

agents that damage the large intestine. Diarrhoea was

often associated with infections and lesions throughout

the small and large intestines in a study of the pathol-

ogy of 21 calves with diarrhoea in southern Britain

(Hall et al., 1988); coronavirus and bacteria adherent to the mucosal surface were associated with the lesions in

the large intestine. The results indicate that these bac-

teria, some of which were EHEC, were contributing

to the development of diarrhoea. Evidence from field

studies also suggests that mixed infections are impor-

tant in the development of diarrhoea. In a longitudinal

study of calves, from birth to weaning, only 15 per cent of calves that excreted *Cryptosporidium* sp. and 37 per **Fig. 14.20**

Occurrence of diarrhoea in farm calves infected with cent of calves that excreted rotavirus experienced diarrhoea either rotavirus or *Cryptosporidium* and in mixed infections.

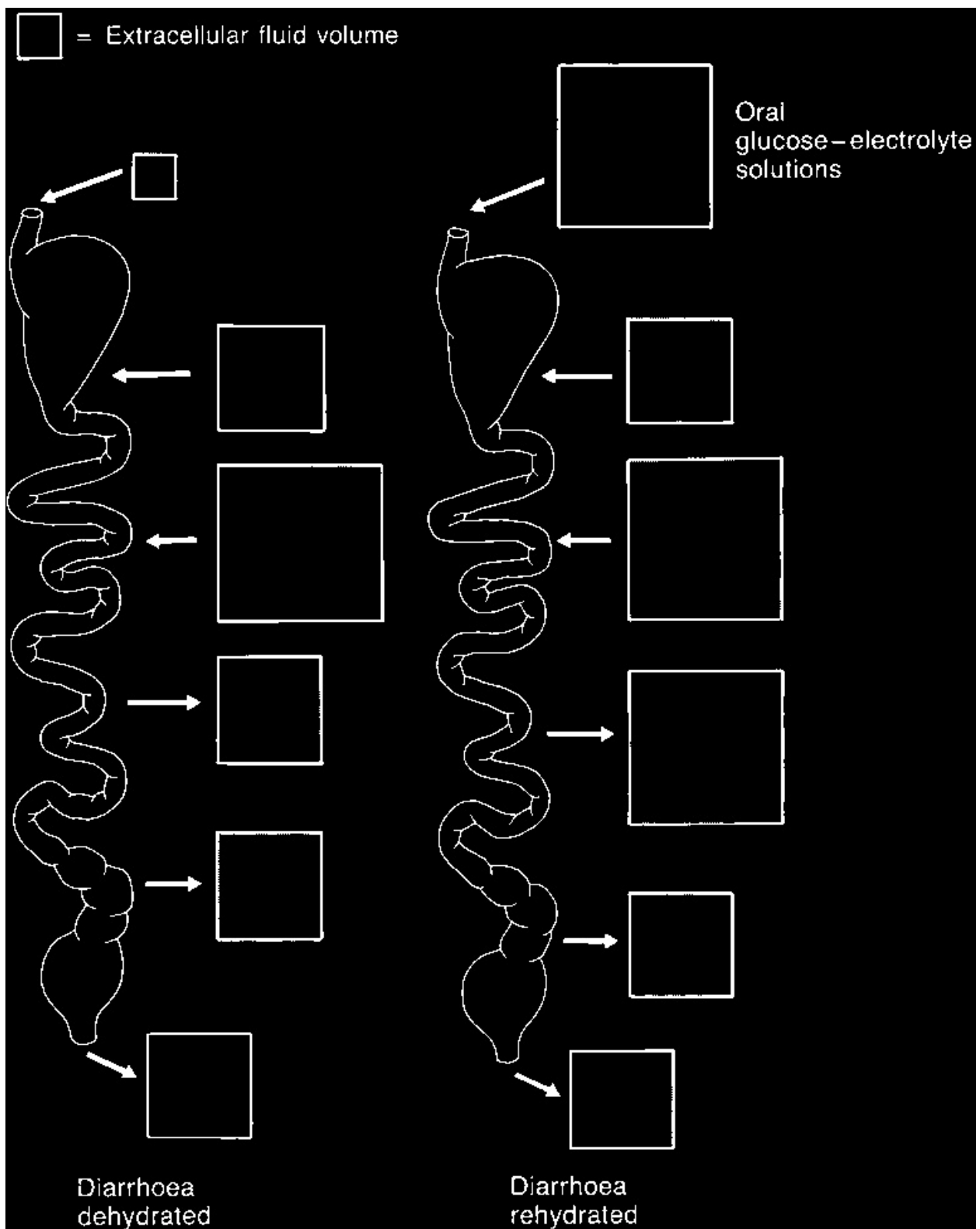
rhoea, but in calves in which the two infections occurred together, 75 per cent developed diarrhoea (Fig. 14.20).

Rotavirus and ETEC were the infectious agents first intestine. The concept that mixed infections involving identified as able to cause diarrhoea in calves and, as rotavirus, cryptosporidia, and coronavirus cause cumulative damage in the small and large intestines is based studied by several groups. The interaction between on information presently available concerning the these two agents was studied despite the fact that predilection sites of these pathogens. There is, however, ETEC cause diarrhoea in calves that are less than evidence that different strains of rotavirus and coronavirus may have different predilection sites within the

*rotavirus diarrhoea is ten days. The conclusions reached
small intestine and this could affect the development of
from these studies were that combined infections were
cumulative damage.*

*more severe, although it was unclear whether this was
due to additive effects or synergism.*





Management of diarrhoea

tion containing glutamine, a simple amino acid and high levels of glucose has also recently been introduced with The first priority is to treat fluid depletion, i.e. restore the claim that this combination will have a beneficial extracellular fluid volume so as to counter shock and effect on mucosal architecture by reducing the severity acidosis.

of villous atrophy and promoting repair. Commercially prepared oral rehydration solutions are sophisticated formulations and should always be used.

As a rule of thumb, if the calf is weak and presents

Rehydration (see also p. 195)

in lateral recumbency, use intravenous therapy; if it Oral fluids are very satisfactory in the less severely can lift its head but has no suck reflex, use intravenous affected animals. They are usually isotonic with plasma therapy. If the calf can drink, offer electrolyte solution and contain sufficient K^+ and HCO^-

3

to replace faecal

by teat and bottle as reluctance to suck may be the first losses and Na^+ and glucose in equimolar amounts. Such indicator of septicaemia/bacterial meningoencephalitis. preparations are formulated to utilize the pathways of The calf should be fed every two to four hours and care-absorption of glucose, amino acids and citrate, which fully monitored; loss of an active suck reflex indicates carry water with them. These formulations have been deterioration of the calf's condition and veterinary improved by inclusion of 80 to 100 mmol/l bicarbonate attendance is necessary without further delay.

as a precursor, usually citrate, which is catabolized to The efficacy of oral rehydration varies with the type give bicarbonate ions which helps to counter metabolic of diarrhoea. In secretory diarrhoea, the sodium acidosis. Energy is supplied as glucose and citrate. More glucose absorption system is unaffected by bacterial recently, oral rehydration solutions have been intro-enterotoxins and glucose/electrolyte solutions are very duced which contain up to 375 mmol/l glucose in an effective in rehydrating the animal. Nevertheless, the

attempt to counteract the energy deficit when milk is diarrhoea continues and this should be stressed to the removed from the calf's diet. An oral rehydration solution (Fig. 14.22). The small intestine may be so

Fig. 14.22

Effect of orally administered glucose/electrolyte solution of fluid fluxes in secretory diarrhoea in the pig. Where glucose/electrolyte solution is not given, there is a net loss of water and dehydration develops. Where glucose/electrolyte solution is given, the diarrhoea continues but there is a net gain of fluid by intestinal absorption and rehydration occurs. Source: Argenzio (1984).

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severely damaged in very severe viral infections that

Microbial environment

oral electrolyte solutions are ineffective. In less severe The enteropathogens that have been discussed are all viral infections, for example many rotavirus infections excreted in faeces in very large numbers by clinically of calves, sufficient functional surface may remain for

sick calves. It is clear, therefore, that once an outbreak of oral electrolytes to be effective.

is established the major source of infection is faecal contamination of the environment by clinically affected

Nutrition

calves. Other animal sources or reservoirs of infection have been described; rotavirus and coronavirus are

The energy requirements of a newborn calf are approximately 2500 kcal/day, equivalent to 600 g of glucose excreted by calves and cows with subclinical infections

and similar situations could be expected for the other

The glucose content of 10 litres of an oral glucose/electrolyte solution containing 80 mmol glucose is 144 g, enteropathogens, which have been investigated less

well. Studies of dairy systems (Greene, 1983; McNulty

which does not supply sufficient energy but this can be & Logan, 1983) have shown the value of cleansing,

largely overcome by feeding an oral rehydration solution

disinfection and use of the 'all-in all-out' system in the

tion containing 375 mmol/l. A rational approach is to control of diagnosed and undiagnosed calf diarrhoea.

feed electrolyte solution alone for one to two days and Calving accommodation and calf pens should be subsequently alternate feeds of milk and electrolyte designed for ease of cleaning, steaming and disinfection solution. While mixing electrolyte solution and milk and with adequate drainage. Pens should be well ventilated, but draught free. Pens and utensils should be kept clean, and pens kept dry with adequate straw.

With suckled calves, separate the calf and the cow, feed The same principles apply in beef systems (Radostits electrolyte and then allow the calf to suck the cow for & Acres, 1980). Firstly, it is important to remove the approximately five minutes.

source of infection from the calves' environment, or minimize it. Measures that can be adopted include the following.

Drugs (pp. 189, 229)

- *Avoid confining the herd, especially at calving.*

In severe dehydration, rehydration must be the first pri-

Avoid using calving paddocks in which mud and ority. There are two situations where the use of anti-faeces soon predominate and where animal density biotics is indicated: infections with Salmonella spp. or is high. with pathogenic E. coli (ETEC, EHEC and VTEC).

- Regularly change the pastures that the herd grazes.*

Broad-spectrum antibiotics and antimicrobials have

- Do not calve cows and heifers on the pasture on been used widely as a primary form of treatment of which they have been held during winter; move calves with neonatal diarrhoea, without sound reason them to clean areas just before calving.*

in many cases. Most therapeutic trials for treatment of

- Do not calve on the same area year after year.*

diarrhoeal calves with antibiotics have been uncon-

Avoid calving in barns and sheds where infection trolled or used small numbers of animals and have not, builds up rapidly, and ventilation and sunlight are therefore, been convincing.

restricted, encouraging survival of infection.

The disadvantages of using antibiotics are that some

- Choose a calving ground that is sheltered and well drained. Avoid creating local areas within calving grounds where animals congregate and infection builds up, i.e. restricted feeding or watering areas.

Antibiotics add significantly to production costs, they may contribute to the problem of resistance factors in

- Calving areas should be cleaned up and left vacant during summer.

and antibiotic use may result in the carrier state in

- Isolate calves, as far as is possible, from the contaminated environment by removing calves from areas contaminated by diarrhoeic calves. Reduce crowding of calves by dividing the calving herd into

Salmonella infections.

Environment

small subgroups and dispersing newborn calves with their mothers soon after birth.

The impact of environment on the pathogenesis of

calf diarrhoea may be discussed under the follow-

Immunological environment

ing headings: microbial environment, immunological environment, nutritional environment and physical environment. Neonatal calves are more resistant to enteric infections when suckled than when fed artificial feeds,

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regardless of whether or not there is absorption of colostral immunoglobulins. This suggests that components of colostrum and milk that are not absorbed have been shown to protect calves from ETEC, probably by anti-adhesive activity. Absorbed colostral antibody nents of colostrum and milk that are not absorbed have may also influence enteric infections because IgA is an intestinal role in protecting the neonate. The other absorbed along with IgG1 and some is resecreted onto quite distinct protective function provided by the mucosal surfaces.

immune components of mammary secretions is their Studies of the concentration of immunoglobulins in absorption to provide passive circulating antibody,

the blood of diseased calves showed that calves with the which prevents invasion of micro-organisms.

lowest levels of immunoglobulin had highest mortality; colisepticaemia was the major killer, either rapidly or via abscesses in the umbilicus, liver and joints. Calves Specific protective systems in colostrum and milk with low levels of immunoglobulins may also show a (see p. 1002)

greater incidence of diarrhoea.

Immunoglobulins are concentrated from the cow's Colostral antibodies, if they are to be absorbed effec-
serum into colostrum from five weeks prepartum.

tively or if they are to have a protective function in the

The classes of immunoglobulins that are present in gut lumen, must resist rapid degradation. The mecha-
colostrum and milk are IgA, IgG1, IgG2 and IgM.

nisms that protect colostrum from degradation in the IgA and IgG2 each comprises 5 per cent of colostral gastrointestinal tract are the low activity of pancreatic immunoglobulins and IgM comprises approximately 7 protease in the neonatal calf, the presence of a trypsin

per cent. The majority of colostral immunoglobulins (80 inhibitor in colostrum, the high buffering capacity of to 90 per cent) are IgG1 and the concentration of IgG1 colostrum, the reduced secretion of acid in the abomasum in colostrum is three to twelve times that of maternal sum in the first three days of life and the resistance of serum, because of selective secretion by the acinar IgG1 to proteolysis by chymotrypsin. The enterocytes epithelial cells of the mammary gland.

of the newborn calf are specialized for non-selective Colostral proteins are absorbed very rapidly and efficiently by the small intestine of the newborn calf. The transport. This process results in rapid and effective small intestinal villi are covered by highly vacuolated uptake of antibodies (IgG1) because these are the major enterocytes, which are specialized in the uptake of macromolecules in colostrum.

macromolecules by pinocytosis. The ability to absorb There are, in addition to protective immunoglobulins, macromolecules may persist for up to 24 hours after

non-specific protective systems in colostrum and milk birth and its disappearance is known as 'closure of the gut' (Reiter, 1978; see also Chapter 58). Lactoferrin, an iron-binding protein present in colostrum and milk, inhibits bacterial growth, possibly by reducing availability of iron to bacteria. There are appreciable amounts in the first feed is colostrum, all is well, but if the first feed is milk, the gut will 'close' and colostrum fed subsequently will not be absorbed. Thus, IgG1 is present in colostrum, competes for iron, making it available for bacteria. Therefore, normal colostrum is not absorbed into the blood of the calf in large amounts during the first 24 hours of life. All macromolecules present are absorbed but IgG1 is predominant and there-fore it is absorbed in greatest amounts. IgA in milk intestine apparently favour the action of lactoferrin provides a passive intestinal humoral immunity in other

because citrate is rapidly absorbed from the calf small species. In ruminants the predominance of IgG1 in milk intestine and bicarbonate, which assists binding of iron suggests that it has a similar function. Determination and lactoferrin, is secreted into the small intestine. The of passive antibody transfer can be easily achieved by antibacterial action of lactoferrin is enhanced by spe- measuring total plasma protein concentration using cific antibodies in colostrum. The lactoperoxidase/thio- a refractometer where values greater than 65 g/l are cyanate/hydrogen peroxidase system is inhibitory or considered optimum and concentration below 50 g/l lethal to bacteria via the production of an oxidation inadequate.

product.

Lactoperoxidase is synthesized by the

The immune components of mammary secretions mammary gland, thiocyanate ions are present in milk have two functions.

Firstly, colostral antibodies

and are secreted into the calf stomach and cow's milk

absorbed into the circulation of the neonate provide contains glucose and glucose oxidase, which provide passive circulating antibody, which prevents invasion hydrogen peroxide; hydrogen peroxide is also produced of micro-organisms (e.g. septicaemic *E. coli*). Secondly, by lactobacilli, which are the predominant bacteria in colostral antibodies that are not absorbed due to gut the flora of the stomach and small intestines of newborn closure, and milk antibodies, act within the gut lumen, calves. Thus, all the components of this protective providing passive local immunity. Milk antibody has system are present in the calf in vivo.

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The absorption of colostral immunoglobulins is influ-

- Encourage outdoor calving in warm weather; it is

enced by several factors and 20 to 50 per cent of calves more likely to lead to higher levels of serum immunoglobulins. The time of the first

colostrum meal is an important determinant of serum immunoglobulin concentration; late feeding leads to

Methods of measuring the concentration of lower levels. Also, first-milk colostrum has the highest immunoglobulins in calf serum include the following: concentration of immunoglobulins, therefore preparturient leaking or milking leads to poorer quality post-

- Single radial immunodiffusion: convenient, accurate and capable of measuring IgG1, IgG2 and IgA. Colostrum is secreted prepartum and once removed is not replaced. The amount of parturient colostrum. Colostrum is secreted prepartum rate and capable of measuring IgG1, IgG2 and IgA.
- Zinc sulphate turbidity test: depends on the selective precipitation of immunoglobulin by zinc in the colostrum and this is more important than the sulphate; there is good correlation between zinc quantity of colostrum consumed.

sulphate turbidity and total immunoglobulin

Maternal nutrition may affect colostrum production. content of serum.

Poor nutrition does not, apparently, influence the

- Sodium sulphite precipitation method. concentration of immunoglobulin in colostrum but

- *Refractometry: measures total protein.*

decreases the amount of colostrum produced, therefore sucking calves achieve lower levels of serum

The immunoglobulin content of colostrum can be immunoglobulin; heifers produce less colostrum than assessed simply and accurately with a hydrometer cows. A second feed of colostrum increases the (Fleenor & Stott, 1980) and one designed specifically absorption of immunoglobulin from the first feed, for the purpose is available in the UK.

presumably because the second feed displaces colostrum from the upper gastrointestinal tract into the jejunum.

Nutritional environment

The presence of the dam improves the efficiency of Skim milk powders, which have been severely heat absorption of immunoglobulins by the calf by up to 80 treated, and some non-milk proteins (e.g. soya) do not per cent. Lower serum levels are obtained if the calf and coagulate well in the abomasum. This may result in dam are separated at birth and colostrum is fed by

gastric stasis and reduced secretion of gastric acid and bucket. The dam licks and cleans the newborn calf in enzymes, which causes increased escape of undigested the order: thorax, back, abdomen, head, neck and per-protein into the duodenum and reduced secretion of ineum. The placenta is then eaten. Suckling does not pancreatic enzymes. These events result in diarrhoea. occur unless the licking process occurs. An easy partu-Soya initiates immune-mediated intestinal damage in rition leads to early licking. Beef cows mother better some calves and heated soya may contain substances than dairy cows; they lick longer and stand better to be that are directly damaging to the intestinal mucosa. sucked. The speed at which calves stand is important Some feeding methods may upset the balance between because they then start to seek the teats. If cows have the calf and the infectious agents to which it is exposed, calved standing then sucking is likely to start earlier. thus precipitating diarrhoea. These include feeding cold Field-born calves have higher levels of serum milk, bucket feeding, high-fat diets and skim milk.

immunoglobulins than box-born calves. The cow is Alternatively, feeding methods that are said to alleviate likely to be the primary object of teat-seeking attention diarrhoea include use of acidified milk and fermented but in a box the walls may confuse the calf. Low colostrum, even though it is less well absorbed than pendulous udders delay successful sucking. fresh colostrum.

Farmers should be encouraged to ensure that all calves receive a minimum of 2 litres of colostrum soon after birth, certainly within 4 to 6 hours. Practical

Physical environment

recommendations to enhance colostrum uptake include the following:

Hypogammaglobinaemia is more common in single-suckled beef herds where the stocking density is high,

- Do not separate dam and calf until 24 hours presumably due to poor mothering and supervision. postpartum.*

Calf mortality is less if the person who looks after the

- Provide bedding to allow the calf to stand easily.*

calves is the farmer, or a member of his or her family.

- *Encourage early feeding, which is very beneficial.*

Single penning helps reduce the spread of infection,

The calf may need assistance to ensure this.

as do solid walls between pens. Aspects of the physical

- *Keep a supply of frozen colostrum for use after environment that contribute to the development of prepartum leakage.*

diarrhoea by stressing calves are inclement weather,

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particularly snow and rain, cold wet bedding and

minal ileum and colon. Abomasal lesions range from dis- overcrowding.

crete 1 to 3 cm diameter oval ulcers to large confluent areas

of ulceration and necrosis in the pyloric region. Haemor-

rhagic or necrotic lesions overlying gut-associated lym-

Necrotic enteritis (See p. 238)

phoid tissue (Peyer's patches) are common.

Necrotic or necrotizing enteritis is the term used to

describe a distinct clinicopathological syndrome char-

References

acterized by severe diarrhoea, pyrexia and neutropenia in two to three month-old beef calves (Penny et al., Argenzio, R.A. (1984) *Pathophysiology of neonatal diarrhoea.*

1994). The disease has been reported to occur in the *Agri-Practice*, **9**, 25–32.

same beef herd for four consecutive years, affecting

Brooks, H.W., White, D.G., Wagstaff, A.J. & Michell, A.R.

approximately 5 per cent of calves when seven to 12

(1997) *Evaluation of a glutamine-containing oral rehydra-*

weeks-old (Penny et al., 1994). Despite intensive sup-

tion solution for the treatment of calf diarrhoea using an

portive and antibiotic therapy approximately 25 per

Escherichia coli model. The Veterinary Journal, **153**, 163–70.

Dupe, R., Bywater, R.J. & Goddard, M. (1993) *A hypertonic*

cent of affected calves died after five to 10 days of

infusion in the treatment of experimental shock in calves

illness. The clinical presentation of necrotic enteritis is

and clinical shock in dogs and cats. Veterinary Record, **133**, not dissimilar to *coccidiosis but the morbidity is much*

585–90.

lower and the findings more severe in nature.

Fleenor, W.A. & Stott, G.H. (1980) *Hydrometer test for*

*Affected calves are dull and depressed and do not
estimation of immunoglobulin concentration in bovine
suck. These calves are moderately (5 to 7 per cent) to
colostrum. Journal of Dairy Science, 63, 973–7.*

*severely (8 to 10 per cent) dehydrated, and pyrexia
Greene, H.J. (1983) Minimise calf diarrhoea by good hus-
(39.5 to 42.0°C) with pale mucous and oral membranes.
bandry: treat sick calves by fluid therapy. Annales de
Sudden onset profuse dark green diarrhoea containing
Recherches Vétérinaires, 14, 548–55.*

*fresh blood is a consistent feature. Tenesmus is also a
Grove-White, D. (2000) Intravenous fluid therapy in the
common feature and becomes more prominent during
diarrhoeic calf. UK Vet, 5, 56–61.*

*Hall, G.A. (1989) Mechanisms of mucosal injury: animal study.
the latter stages of the disease and may cause tem-
In: Viruses and the Gut, Proceedings of the Ninth BSG:
porary rectal prolapse. There is rapid loss of body con-
SK&F International Workshop, 27–29.*

*dition and affected calves have a gaunt appearance with
Hall, G.A., Reynolds, D.J., Parsons, K.R., Bland, A.P. &*

a tucked-up abdomen. This syndrome has many clinical

Morgan, J.H. (1988) Pathology of calves with diarrhoea in

features of mucosal disease but oral erosions and

southern Britain. Research in Veterinary Science, 45, 240–50.

ulceration are observed in less than 50 per cent of

Kasari, T.R. & Naylor, J.M. (1986) Clinical evaluation of

calves, and there are no interdigital lesions.

sodium bicarbonate, sodium lactate and sodium acetate for

Muco-purulent ocular and nasal discharges, com-

the treatment of acidosis in diarrhoeic calves. Journal of the monly seen in mucosal disease, are not observed in

American Veterinary Medical Association, 187, 392–7.

necrotic enteritis. The age-specific occurrence of necrotic

Lewis, L.D. & Philips, R.W. (1978) Pathophysiologic changes enteritis differs from mucosal disease which more

due to coronavirus-induced diarrhoea in the calf. Journal of the American Veterinary Medical Association, 173, 636–42.

commonly affects yearling cattle although BVD/MD

McNulty, M.S. & Logan, E.F. (1983) Longitudinal survey of

persistently affected cattle may die of intercurrent

rotavirus infection in calves. Veterinary Record, 113, 333–5.

disease, especially bacterial pneumonia, from two

Mebus, C.A., Stair, E.L., Underdahl, N.R. & Twiehaus, M.J.

months old. BVD antigen has not been detected

(1971) Pathology of neonatal calf diarrhoea induced by a

in blood samples from affected calves nor from fresh

reo-like virus. *Veterinary Pathology*, **8**, 490–505.

tissue samples. No recognized enteropathogen has

Morgan, J.H. (1990) Epidemiology, diagnosis and control of

been isolated from faecal examinations. There is mild to

undifferentiated calf diarrhoea. *In Practice*, **12**, 17–20.

severe anaemia and profound leucopaenia caused pre-

Naylor, J.M. (1996) Neonatal ruminant diarrhoea. *In Large*

dominantly by a severe non-regenerative neutropaenia.

Animal Internal Medicine (ed. by B.P. Smith), 2nd edn.

The administration of oral and/or systemic antibiotics

Mosby, St Louis, p. 404.

has no effect on the outcome of necrotic enteritis.

Penny, C.D., Scott, P.R., Watt, N.J. & Greig, A. (1994) Necrotic enteritis of
unknown aetiology in young beef calves at

Supportive therapy comprising oral and intravenous

pasture. *Veterinary Record*, **134**, 296–9.

fluids, and flunixin meglumine, may effect a temporary

Radostits, O.M. & Acres, S.D. (1980) The prevention of epi-improvement but does not alter the prognosis.

demics of acute undifferentiated diarrhoea of beef calves in

Erosion or ulceration of the hard palate is common but

western Canada. *Canadian Veterinary Journal*, **21**, 243–9.

no oesophageal ulceration has been recorded. Consis-

Reiter, B. (1978) Review of the progress of dairy science:

tent findings are necrotizing lesions in the abomasum and

antimicrobial systems in milk. *Journal of Dairy Research*, **45**, small and large intestine, particularly involving the ter-131–47.

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Reynolds, D.J., Hall, G.A., Debney, T.G. & Parsons, K.R.

Grimshaw, W.T.R. (1987) Efficacy of sublactam-ampicillin in

(1985) Pathology of natural rotavirus infection in clinically

the treatment of neonatal calf diarrhoea. *Veterinary Record*, normal calves. *Research in Veterinary Science*, **38**, 264–9.

121, 162–6.

Reynolds, D.J., Morgan, J.H., Chanter, N., Jones, P.W., Bridget, Michell, A.R., Bywater, R.J., Clarke, K.W., Hall, L.W. &

J.C., Debney, T.G. & Bunch, K.J. (1986) *Microbiology of calf* Waterman, A.E. (eds) (1989) *Veterinary Fluid Therapy*.

diarrhoea in southern Britain. *Veterinary Record*, **119**, 34–9.

Blackwell Scientific Publications, Oxford.

Scott, P.R. & Penny, C.D. (1993) A field study of meningo-

Moerman, A., de Leeuw, P.W., van Zijderveld, F.G.,

encephalitis in calves with particular reference to cere-

Baanvinger, T. & Tiessink, J.W.A. (1982) Prevalence and

brospinal fluid analysis. *Veterinary Record*, **133**, 119–21.

significance of viral enteritis in Dutch dairy cattle. In

Snodgrass, D.R., Terzdo, H.R., Sherwood, D., Campbell, L,

Proceedings XIIth World Congress on Diseases of Cattle,

Menzies, J.D. & Synge, B. (1986) Aetiology of diarrhoea in Amsterdam. Vol. 1.
pp. 228–36.

young calves. *Veterinary Record*, **119**, 31–4.

Pearson, G.R. & Logan, E.F. (1986) Pathological and immunological aspects of
neonatal enteritis of calves. *Veterinary Annual*, **26**, 68–75.

Further reading

Roy, J.H.B. (1990) *The Calf*. In *The Management of Health*, Vol. 1, 5th edn.
Butterworths, London.

Acres, S.D. (1985) Enterotoxigenic. *Escherichia coli* infections Saif, L.J. &
Smith, K.L. (1985) Enteric viral infections of in newborn calves: a review.
Journal of Dairy Science, **68**, calves and passive immunity. *Journal of Dairy
Science*, **68**, 229–56.

206–8.

Angus, K. (1987) Update: Cryptosporidiosis in domestic

Snodgrass, D. (1986) Prevention of calf diarrhoea by vaccina-

animals and humans. *In Practice*, **9**, 47–9.

tion. *In Practice*, **8**, 239–40.

Chapter 15

Salmonellosis

P.W. Jones, P.R. Watson and T.S. Wallis

Introduction

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of three major syndromes: a peracute systemic infec-

Classification

216

tion, an acute enteritis or a chronic enteritis. In humans,

Resistance to antibiotics

217

this ranges from the generalized typhoid infection,

Epidemiology of salmonellosis in cattle

218

through the less severe paratyphoid infections to a

The carrier state

219

mild gastroenteritis. The majority of serotypes produce

Pathogenesis

220

a mild to severe gastroenteritis that only rarely be-

Initiation of infection

220

comes generalized and severe infections are most often

Interaction with the intestines

221

Systemic spread and persistence

224

encountered in very young, old or immunologically-

Signs of disease

225

compromised patients. It is generally accepted that

Adults

225

Salmonella gastroenteritis is a zoonotic disease, mainly Calves

226

contracted by consuming large numbers of salmonellas

Diagnosis of salmonellosis

226

in food of animal origin or foods contaminated with

Control measures and vaccination

227

animal products in which the salmonellas have pro-

Vaccination

228

liferated. There is, however, convincing evidence not

Antibiotic therapy

229

only that infection can be a sequel to the consumption

Summary

230

of small doses but that direct person-to-person contact

is involved in many outbreaks.

The disease in cattle differs from the disease in

Introduction

humans, where the majority of cases in the developed

world represent self-limiting intestinal infections, but

Salmonellosis is a collective description of a group of

which occasionally have a high mortality even when

diseases caused by bacteria of the genus Salmonella

treated.

with signs that vary from severe enteric fever to mild

Until recently salmonellosis in animals in the UK
food poisoning. One of the characteristics of *Salmonella*
was characterized by the large proportion of infec-
tions caused by the 'host-specific' and 'host-restricted'
that they can infect and cause disease. For example, *S.*
serotypes: *S. choleraesuis* in pigs, *S. abortusovis* in sheep, *typhimurium* and *S. enteritidis* have a very broad host range and infections with these serotypes have been
cattle. Recently, apart from the latter, these serotypes
associated with virtually all warm-blooded animals.
have virtually disappeared in the UK although they
In contrast, other serotypes have a more limited host
remain important in other parts of the world.
range and this has been interchangeably referred to as
Salmonellas may be carried by animals in the absence
host specificity, restriction or adaptation. The highly
of clinical signs and this is probably the normal situation
host-specific serotypes infect only phylogenetically
in pigs and poultry, although *S. enteritidis* (phagetype 4) closely-related host
species; for example *S. typhi* infects only human beings, *S. abortusovis* infects only sheep

The cycle of infection between man and farm animals

*and goats and *S. gallinarum* infects only poultry. Other is often called the ‘*Salmonella cycle*’, which is shown in serotypes are predominately associated with disease*

Fig. 15.1. Some of the links in the cycle are tentative and

in one species but may also infect a limited number of

the main source of infection for humans is animal prod-

*other host species. For example, *S. dublin* is usually ucts and the principal sources of infection for domestic*

associated with cattle, but natural infection by this

animals are other animals of the same species and

serotype may occur in other animals, including human

contaminated feed.

beings and sheep .

Whilst it is accepted that infections in the human

Salmonellosis as a disease of human beings, cattle,

population are usually associated with the consumption

sheep, pigs and poultry is manifested clinically by one

of animal products such as eggs, meats and milk, it is

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Wild animals

Imported

(mammals, birds,

human food

insects)

Animal

products

Imported

FARM

MAN

animals

ANIMALS

Animal

waste/slurry

Animal

Domestic

feed

pets

Infected

pasture

Contaminated

water

Imported

Sewage,

protein

sludge and

effluent

Fig. 15.1

The ‘ Salmonella cycle’.

not always realized that it is possible for the farm

strains are auxotrophic, e.g. S. typhi requires tryptophan animal population to become infected by direct contact

for growth, S. dublin requires nicotinic acid and S.

with man or the waste products of man such as sewage

choleraesuis requires cysteine.

and sewage-polluted waters. It is further complicated

The genus is subdivided into serotypes (or serovars),

by the possibility of the spread of disease by wild

which in some classifications are grouped into sub-

animals such as rodents, birds and insects and by the

genera on the basis of their biochemical reactions.

recycling of animal products and wastes from one

Subgenus I contains the ‘typical’ salmonellas isolated

animal species to another. Thus an understanding of salmonellosis mainly from warm-blooded animals, and these are the bacteria which normally cause disease in cattle. The disease problems may only arise from an interrelated serotypes (or serovars) within a subgenus cannot, with study of the disease in all groups of animals and of the rare exceptions, be distinguished biochemically and differentiation is on the basis of the possession of somatic the environment and pass from one animal to another. (O) antigens and diphasic flagellar (H) antigens in the Kauffmann–White scheme. The antigenic formula consists of three parts delimiting the O antigens and the

Classification

H antigens which may occur in two phases. O antigens have arabic numbers and H antigens have letters in Strains of the genus *Salmonella* obey the definition phase 1 and letters and numerals in phase 2, since of the family *Enterobacteriaceae*: they are straight the same antigens may occasionally occur as phase 1 or

rods, usually motile with peritrichous flagella (*S.*
phase 2 in different serotypes. Thus the common cattle
pullorum/gallinarum which cause disease in poultry are pathogen *S. dublin* has
the antigenic formula 1,9,12:gp:-; the non-motile exceptions), facultatively
anaerobic,
this denotes that it is distinguished by the presence
ferment glucose usually with the production of gas (*S.*
of O antigens 1, 9 and 12 and H antigens g and p. It
typhi and some strains of *S. dublin* are the exceptions) does not have a second
phase. The other common cattle
and reduce nitrate to nitrite. Most are prototrophic
pathogen *S. typhimurium*, which is diphasic, has the
(grow in artificial media with glucose as the sole source
formula 1,4,5,12:i:1,2. When initiated the scheme
of carbon and energy and ammonium ions as a
comprised 44 serotypes and varieties organized in five
nitrogen source). Most host-specific and host-restricted
O groups (A–E), but it has since expanded to include
Salmonellosis • 217

Table 15.1

The antigenic formulae (Kauffmann–White scheme)

and *S. enterica* subsp. *indica*). The names of serotypes of *Salmonella* serotypes
commonly isolated from cattle in the UK.

are not considered as species names and are not printed in italics. The species name is usually *S. enterica* and **Serotype**

Antigens

the serotypes are written, for example, as *S. enterica* (sometimes subsp. *enterica*) serovar typhimurium or

Somatic

Flagella

S. enterica serovar dublin.

Phase 1

Phase 2

S. saintpaul

1,4,[5],12

e,h

1,2

Resistance to antibiotics (see p. 1038)

S. derby

1,4,[5],12

f,g

—

S. agona

1,4,12

f,g,s

—

A feature of salmonellosis in cattle during the last four

S. typhimurium

1,4,[5],12

i

1,2

decades has been the development of strains resistant

S. agama

4,12

i

1,6

to one or more antibiotics. This has occurred most com-

S. bredeny

1,4,12,27

l,v

1,7

monly in a relatively small number of phagetypes of S.

S. heidelberg

1,4,[5],12

r

1,2

typhimurium and has been the subject of considerable

S. indiana

1,4,12

z

1,7

debate on the use of antibiotics prophylactically,

S. stanleyville

1,4,[5],12, 27

z4,z23

[1,2]

therapeutically and as growth promoters in calves. It is,

S. montevideo

6,7, 14

g,m,[p],s

[1,2,7]

however, generally accepted that resistance is develop-

S. virchow

6,7

r

1,2

ing under the selective pressure of the indiscriminate

S. infantis

6,7, 14

r

1,5

S. mbandaka

6,7, 14

z

use of antibiotics. This is thought to be particularly true

10

e,n,z15

S. newport

6,8

e,h

1,2

both of the use of mixtures of antimicrobials for therapy

S. enteritidis

1,9,12

g,m

[1,7]

without diagnosis and antimicrobials mixed into animal

S. dublin

1,9,12,[Vi]

g,p

—

feeds. The problem was first recognized in the 1960s

S. panama

1,9,12

l,v

1,5

when the number of antibiotic-resistant strains isolated

S. anatum

3,10

e,h

1,6

from cattle rose from less than 3% at the beginning

S. give

3,10

l,v

1,7

of the decade to more than 60% by 1965 and most of

S. havana

1,13,23

f,g,[s]

—

the isolates belonged to one phagetype (DT29). This phagetype disappeared at the end of the decade and the —, flagella antigens occur in first phase only.

proportion of antibiotic-resistant isolates declined, but [], not always present.

1, 14, 27, phage-determined, not always present.

by the end of the next decade the proportion had again risen to approximately 60%. This was due to the emergence of two closely related types (DT193 and DT204) and was associated with the development of chlo- over 2400 serotypes organized in 48 O groups. The anti-ramphenicol resistance. Although these two phagetypes genic formulae of serotypes commonly involved in also subsequently declined, they were replaced during disease in cattle are shown in Table 15.1.

the 1980s by the closely related phagetype DT204c.

The names given to the serotypes do not follow the

This phagetype became endemic, particularly amongst

usual rules of nomenclature. The first serotypes to be market-purchased calves and in the calf-rearing identified, such as *S. typhi*, *S. choleraesuis*, *S. abortus*-trade. It subsequently declined and was replaced by *S. typhimurium*, were given names that indicate phagetype DT104. This multiply-resistant phagetype indicated the disease with which they were associated or also became established in sheep and pigs. their common animal host, and these names, which have Strains of *S. dublin* isolated in the UK, unlike *S. typhimurium*, have remained comparatively sensitive to antibiotics. In contrast, strains isolated in Holland, named after the town or region in which they were first isolated, such as *S. dublin*, *S. liverpool*, *S. crossness* and to a number of antibiotics. The reason for the difference between British isolates and those from North America formulae. As an aid to epidemiological studies many of and Europe has not been explained satisfactorily but is the commonest serotypes have been further subdivided

usually attributed to the selection pressure of antibiotics in animal feed. Presumably the same pressure is exerted in the UK as elsewhere and is equally applied

In alternative classifications which are increasingly being used *Salmonella* is considered as one or two

on *S. dublin* as on *S. typhimurium*. Multiply-antibiotic-species (with subspecies *S. enterica* subsp. *enterica*, *S.*

resistant strains of *S. dublin* resistant to chloramphenicol-*enterica* subsp. *salamae*, *S. enterica* subsp. *arizonae*, *S.*

col began to be reported in the UK from 1979 and the

enterica subsp. *diarizonae*, *S. enterica* subsp. *houtenae* chloramphenicol resistance was transmissible, although

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located on R plasmids distinct from those carrying the

epidemic of *S. enteritidis* phage type 4, which replaced *S.*

same resistance genes in *S. typhimurium*.

typhimurium as the predominant serotype in poultry

and the human population in the 1980s and 1990s, will

follow the same course. It is always possible, therefore,

Epidemiology of salmonellosis

for the relative importance of *S. typhimurium* and *S.*

in cattle

dublin to change and the relative importance of these two serotypes in cattle should not be judged on the

In cattle the disease, which has been recognized for two basis of isolations over only a few years.

centuries, has a world-wide distribution and has been Salmonellosis occurs in calves in all months of

associated primarily with S. dublin and S. typhimurium.

the year but there is a peak of disease associated with

Other serotypes infect cattle more sporadically and

all serotypes between October and December, with a

these 'exotic' serotypes have become more common in

low incidence in June and July. This is associated with

the UK during the past 50 years, although during the

calving patterns rather than a true seasonal increase

last 20 years approximately 100 serotypes other than S.

and reflects the distribution of calvings in dairy herds

dublin and S. typhimurium have accounted for only and may change. Similarly, since the source of calves

about 10 per cent of incidents. The number of outbreaks

sold through markets to fattening units is bull calves

caused by the 'exotic' serotypes has declined in the last

from dairy herds and surplus cross-bred heifer calves
few years. There was a dramatic rise in the incidence of
from dairy and beef units, there is also a seasonal
salmonellosis in cattle between 1960 and 1969 due to an
incidence in fattening units since most outbreaks in
increase in isolations of *S. dublin*. This was followed by calves occur within a
few weeks of purchase from
an equally dramatic decline in both *S. dublin* and total markets.
incidents and a slight rise in *S. typhimurium*. Over the The importance of
salmonellosis as a disease of
last 20 years the incidence has remained about the same
calves was demonstrated by a survey carried out
and at present *S. dublin* and *S. typhimurium* are isolated by researchers at the
Institute for Animal Health,
at about the same frequency, with the former being
Compton, and is described in detail later in this chapter
more common in adults and the latter more common in
(p. 219). In this survey samples of faeces from diar-
calves.
rheic calves on 45 farms in the south of England were
Recent estimates would suggest that approximately
examined for the presence of a variety of putative

20 per cent of UK cattle herds may be infected with sal-
enteropathogens including salmonellas, rotavirus, calici-
monellas at any one time, although obvious disease is
like viruses, coronavirus, E. coli, cryptosporidia and a much rarer event.
Serological surveys have suggested
campylobacters. Salmonellas were isolated from 12 per
a history of infection in up to 75 per cent of UK dairy
cent of diarrhoeic calves in 24 per cent of diarrhoea out-
herds. In the UK approximately 400 to 500 incidents
breaks. In 37 of the outbreaks, samples were taken from
(outbreaks in a single herd) of salmonellosis in cattle
normal calves of similar age to the calves with diarrhoea
are reported annually. This is almost certainly an under-
and a comparison between isolation rates from healthy
estimate since disease outbreaks often are not reported
and diarrhoeic calves was prepared. Salmonellas were
and carriage in the absence of disease is common.
isolated from 12 per cent of diarrhoeic calves compared
The distribution of salmonellas in cattle in other parts
with only 3 per cent of normal calves, thus showing a
of the world is similar to the UK. S. dublin is endemic clear association of the
organism with disease. Salmo-in northern Europe and western North America

and *S.*

nellas were associated with an outbreak of severe

dublin and *S. typhimurium* tend to be present in most dysentery on six farms while single cases of typical *sal*-advanced dairying areas and intensive cattle rearing

monellosis occurred in three outbreaks, and *salmonel*-

regions. In the rest of the world occurrence is more

las were detected with other agents as part of a

sporadic and serotypes other than *S. dublin* and *S.*

diarrhoea problem in two outbreaks where dysentery

typhimurium are commonly involved.

and pyrexia were not recorded. This highlighted the

A feature of *Salmonella* infections in all species,

problem of arriving at a diagnosis in an outbreak of

however, is a continual fluctuation in the proportions of

enteritis when several organisms may be involved as

the serotypes involved. It is common for a serotype to

part of a disease syndrome. Since *salmonellas* may be

be introduced to the country and to establish in one or

carried by apparently healthy animals, isolation of the

more species, possibly as the predominant serotype, and

organism, unless it is present in large numbers ($>10^5$ per

then to decline without any apparent reason or without
gramme of faeces), is not proof of the cause of disease.

the intervention of public health or veterinary authori-

There was a correlation between the type of farm and

ties. The predominance of *S. hadar* in poultry and

the isolation of salmonellas from diarrhoeic calves.

the human population in the 1970s to 1980s is a good

Diarrhoea was more likely to be associated with sal-

example and it may possibly be predicted that the

monellas in units (calf-rearer, multiple-suckler) where

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large numbers of calves were purchased from markets,

ronmental contamination the organism was not

although a large number of isolations were also made

isolated from the calves on arrival. Subsequent batches

from calves on dairy farms.

of calves (20 or 30 per batch) were examined on arrival

The source of most outbreaks is probably animal-to-

and three further serotypes, *S. typhimurium*, *S. agama* animal contact, although
there are distinct differences

and

S.

binza, were isolated .

Salmonellas were

in the epidemiology of the disease in adults and calves, subsequently isolated from 20 per cent of calves that and between serotypes. The main sources of infection developed diarrhoea on the farm.

for cattle herds are probably bought-in cattle, contam-

Both of these surveys amply demonstrate that infec-

inated feed, contaminated animal wastes spread on

tion in a limited number of calves can spread rapidly in

pasture, human sewage sludge spread on pasture, con-

young, susceptible animals subjected to the stress of the

taminated water courses, birds, particularly gulls,

marketing and rearing systems and once established

rodents, insects and human contact. Infected cattle may

it may be difficult to remove the organism from an

excrete up to 108 salmonella/g of faeces and con-

infected environment. It may also be possible that

tamination of the environment in the proximity of other

salmonellas, which cannot be recovered on normal

cattle by excreting animals will obviously be a potent bacteriological media may, although non-recoverable, source of infection. In an outbreak of *S. saintpaul* infection remain viable and infective for animals.

tion in two large dairy herds, infection rapidly spread

The manner in which animals in previously uninfected dairy herds become infected is more problematic, and the source of infection may vary from serotype to serotype. Most outbreaks are probably associated with the introduction of infected stock or contaminated feed but polluted water supplies, the spreading of calves or the contaminated environment. The collection of contaminated animal manures and human sewage on pasture, and contamination of pasture or feed to markets and dealers' premises, produces an ideal

by scavenging birds may also be involved. Outbreaks environment for dissemination. When salmonellosis have also been described where the probable source of gains a foothold in calves subjected to this treatment it infection was direct human contact. Insects may also be spreads so rapidly that entire herds in rearing premises involved in mechanical transmission since it is difficult may become infected.

to contain salmonellosis outbreaks when animals are There have been several reports that demonstrate the kept in units that are not insect-proofed and similar importance of salmonellas in market-purchased calves difficulties have been described in rearing Salmonella- and in rearing units. One of these was a survey of almost free pigs unless fattening units were rendered fly-proof. 600 market-purchased calves supplied to 11 rearing The epidemiology of S. dublin in dairy herds differs units. Frequent swabbing of the calves indicated that from that of other serotypes. Salmonella dublin has less than 1 per cent were infected when they arrived at a precise geographical distribution. The organism has

the rearing units but within the next six weeks over half remained established in adult cattle in Wales and in the calves became infected. The calf units examined southwest and northwest England. It is thought that in this survey were cleaned and disinfected between this distribution may, in part, be due to an association between *S. dublin* and *Fasciola hepatica*, although this isolated from the environment after cleaning in over may be fortuitous and merely indicate that both half of the units. In a similar study of a calf unit in are influenced by similar climates and survive better in Berkshire, UK, which bought calves from local markets, wet conditions. *Salmonella dublin* probably persists on 250 calves were examined over a two-year period and farms in these areas because recovered animals remain 51 were shown to be infected with one of four different infected and excrete the bacteria in their faeces, either phagetypes of *S. typhimurium*. Phagetype DT204c, continuously or intermittently. Elsewhere in the UK the which was multiply-antibiotic-resistant, was isolated disease is predominantly one of calves in calf-rearing

at the beginning of the survey. This was eventually units rather than adults and it is introduced to the units removed by destocking and a rigid hygiene programme. with the introduction of infected animals.

The calf units were steam-cleaned, after which *S. typhimurium* DT204c was still isolated from environmental samples. Following further steam-cleaning and

The carrier state

washing with disinfectant the organism was no longer detectable in the environment but was detectable

Animals which excrete salmonellas continuously from the next batch of calves introduced to the units.

(‘active carriers’), usually in concentrations of greater

Although it is not certain that this was the result of environmental contamination, salmonellas, which are present in more than 10⁵/g of faeces, can be detected by bacteriological

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examination which will also detect ‘passive carriers’, particularly from field studies, would favour the latent animals that ingest salmonellas in their feed and carrier hypothesis.

pass them in their faeces without actual infection of

In contrast to S. dublin, infection with other serotypes the intestine or the mesenteric lymph nodes. When

does not appear to result in active or latent carriers.

removed from an infected environment these latter

Adult cattle usually excrete for a maximum of a few

animals stop excreting. Active excretion is usually a

weeks following infection and excretion that is detected

sequel to clinical enteritis or septicaemia and infected

for longer periods probably reflects recontamination

animals may excrete for many years and perhaps for

from the environment, although the establishment of

life. It may also develop in animals that have not shown

some 'exotic' serotypes such as S. saintpaul for up to a clinical signs, although this probably occurs only in

year has been reported. Similarly, S. saintpaul has been cases of concurrent fascioliasis. In such cattle, S. dublin shown to be retained in the tissues of calves for up to

is characteristically present in the gall-bladder and

eight weeks after excretion had apparently ceased.

alimentary contents without necessarily colonizing the

rest of the animal. However, although active (persist-

Pathogenesis

ent) excretors are an obvious source of infection for

other cattle, and although the source of calf infection is

An overview of the main stages involved in *Salmonella*

assumed to be adult cattle, field studies have shown that

pathogenesis is presented in Fig. 15.2.

the majority of calves infected with *S. dublin* are home-bred on farms where there is no clinical evidence of

Initiation of infection

adult salmonellosis. This is characteristic of the disease

and has led to a search for other sources of infection.

Cattle are probably infected with salmonellas by the

Many of the infections occur in closed herds; other

oral route, although respiratory and conjunctival infec-

sources of infection such as feed are not appropriate

tion may also occur. The dose required to initiate infec-

to a 'host-restricted' serotype such as *S. dublin* and tion is thought to be high and will be dependent on the

although contaminated streams may be implicated, this

age, immunity and dietary status of the animal and vir-

gap in the epidemiology is usually explained by the exis-

ulence of the infecting strain. However, it is probable

tence of 'latent carriers'. These are animals that have

that animals are infected naturally by much smaller

salmonellas somewhere in their tissues or alimentary doses since factors such as concurrent parasitism (par-tract but only rarely excrete the organisms in their ticularly fascioliosis), ketosis, metritis, mastitis, cystitis, faeces. It has been suggested that infection with S.

pneumonia, viral infection, dietary changes, pregnancy,

dublin is mainly latent, but is activated by stress,

food and water deprivation and other stress factors

particularly at parturition, and the birth of congenitally

such as freezing, wet weather or worming may lead to

infected calves to latent carriers or to cows that excrete

increased susceptibility. In calves, colostrum, whether

S. dublin would only intermittently explain the occur-normal or immune, is particularly important and

rence of disease in calves on farms where searches for

animals that have either not received colostrum or have

active carriers are unsuccessful. It is possible to detect

received insufficient amounts are particularly suscepti-

greater numbers of excretors by bacteriological exami-

ble. Natural infections have been described where

nation of faeces taken at calving and surveys have

animals may have been infected with very small doses,

revealed animals which, although found to be infected particularly from infected feed. In one such outbreak with *S. dublin* at necropsy, were not detected as carrier-dairy cattle were infected from a component of feeders by previous faecal sampling. It has not proved possible to create such animals experimentally, however, to create such animals experimentally infection spread to their calves. The experimental infection and serological tests for their detection have been tedious oral dose for calves has been variously estimated singularly unsuccessful.

as between 10⁵ and 10¹¹ organisms, although doses in Serum agglutination tests (SAT) may detect a excess of 10⁸ are normally required. The dose for adult proportion of actively infected animals but they do not cattle is approximately 10¹¹ orally and greater than 10⁸ detect latent carriers and so far other immunological intravenously.

tests are equally unsuccessful, although they may be Gastric acidity is responsible for eliminating a large useful in detecting active carriers and infected herds.

proportion of ingested organisms in both monogastric
Delayed hypersensitivity skin tests can distinguish sys-
animals and ruminants. In ruminants, survival of sal-
temically infected animals but they may, unfortunately,
monellas is further reduced by the high concentration
also detect convalescent and recovered animals. It
of short-chain fatty acids in rumen fluid and conse-
is possible that many unexplained outbreaks may be the
quently salmonellas disappear rapidly from the rumen
result of human contact or extended survival of the
of regularly fed cattle. Factors which alter the acidity and
organism in the environment, although most evidence,
composition of rumen fluid, such as starvation and

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Bacterial virulence

Host defence

properties

Ingestion

Survival in

Acid adaptation

Gastric acid, normal flora

stomach/rumen

Fimbriae

Colonisation of intestinal

Mucus, intestinal motility

lipopolysaccharide

epithelium

Fluid secretion,

TTSS-1 secreted

Invasion of intestinal

inflammation phagocytosis,

effector proteins

epithelium

complement activation

TTSS-2 secreted

Evasion of host

Macrophages, neutrophils

effector proteins

defences

TTSS-1

secreted

Induction of

Systemic

Iron-binding proteins

effector

enteritis

spread

proteins

Genes not

identified

TTSS-2, virulence plasmid,

Systemic

iron-binding proteins, SodC,

Pyrexia response

Fig. 15.2

A tentative overview of the

salmonellosis

endotoxin

pathogenesis of salmonellosis. TTSS =

For description see pp. 220–25

type three protein secretory system.

refeeding, achlorhydria or administration of antacid,

the intestines may also be inhibitory to infection and

increase susceptibility to infection. *Salmonellas* that the absence of a fully developed intestinal flora may survive passage through the stomach(s) will probably in part account for the comparative lack of resistance be phenotypically altered. Experiments in vitro have of young animals to salmonellosis. Despite these host shown that exposure to low pH and/or short-chain fatty defences, *salmonellas* are able to associate rapidly with acids results in the induction of expression of up to 50 the intestinal epithelial monolayer. Bacterial motility proteins. This correlates to increased bacterial resistance and chemotaxis may aid this process by allowing to further exposure to low pH. Several bacterial genetic the bacteria to move more freely within the intestinal loci have been shown to regulate this acid-lumen, to sense the presence of the epithelial mono-adaptation response. Three of these genetic loci, *rpoS*, layer and to penetrate the overlying mucus layer.

fur and *phoP/phoQ*, have previously been implicated in The interaction between *salmonellas* and the intes-regulating virulence in the mouse model of systemic tinal mucosa is highly dynamic. *Salmonellas* are rapidly salmonellosis and mutation of several of the other genes

able to mediate their own uptake into epithelial cells has also suggested a link between acid adaptation, and this is associated with a dramatic, but temporary, survival within murine macrophages and virulence in 'ruffling' of the apical membrane of the epithelial cell mice. To date, there have been no studies on the effect of at the site of bacterial entry (Figs 15.3 and 15.4). The acid adaptation on pathogenesis in bovine salmonellosis. main initial site of entry is probably the distal ileum. This may be because the intestinal contents are held within the distal ileum for some time before entry into

Interaction with the intestines

the caecum and it may also be partly due to the relatively high number of M cells (specialized epithelial salmonellas must be able to resist several host innate cells overlying the Peyer's patches) in the distal ileum. defences, including lysozyme and lactoferrin, and to In mice M cells are interspersed between enterocytes prevent their removal from the intestines in the normal and salmonellas can clearly be seen preferentially

movement of food by peristalsis. The bacterial flora of interacting with the M cells. In calves, M cells form a





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Fig. 15.3

Scanning electron micrograph of uninfected bovine ileum showing a group of typical M cells (M) on a domed villus. The cells have characteristic microfolds with a central area of short microvilli.

Fig. 15.5

Scanning electron micrograph of uninfected bovine ileum showing the normal morphology of villi. homogeneous monolayer over the dome villi (villi associated with the Peyer's patches) and so the relative

interaction of salmonellas with M cells and enterocytes is more difficult to assess. It appears, however, that salmonellas will initially interact with the M cells (within 10 minutes of direct inoculation into the distal ileum) and are not taken up by the enterocytes until slightly later (approximately 20 minutes after inoculation).

The mechanism of Salmonella invasion has been extensively studied. The bacterial genes involved in invasion are clustered together at one site on the chromosome, which has been named Salmonella pathogenicity island 1 (SPI-1). SPI-1 encodes a type 3 protein secretion system (TTSS-1: so called after comparison to two previously described secretion systems), regulatory

Fig. 15.4

Scanning electron micrograph of bovine ileum after

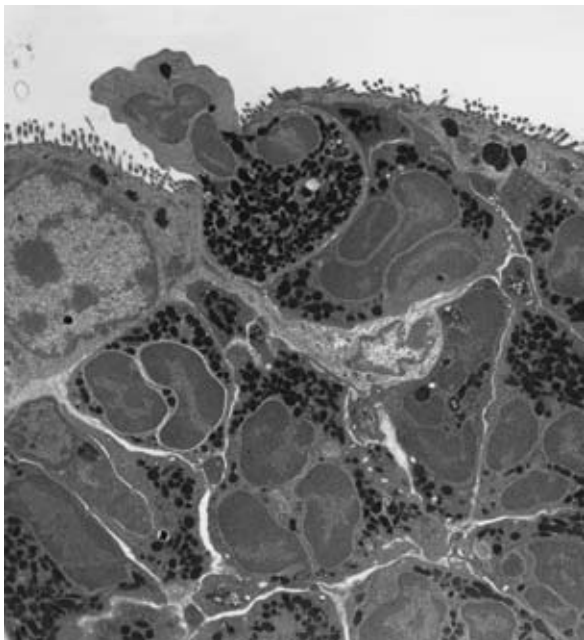
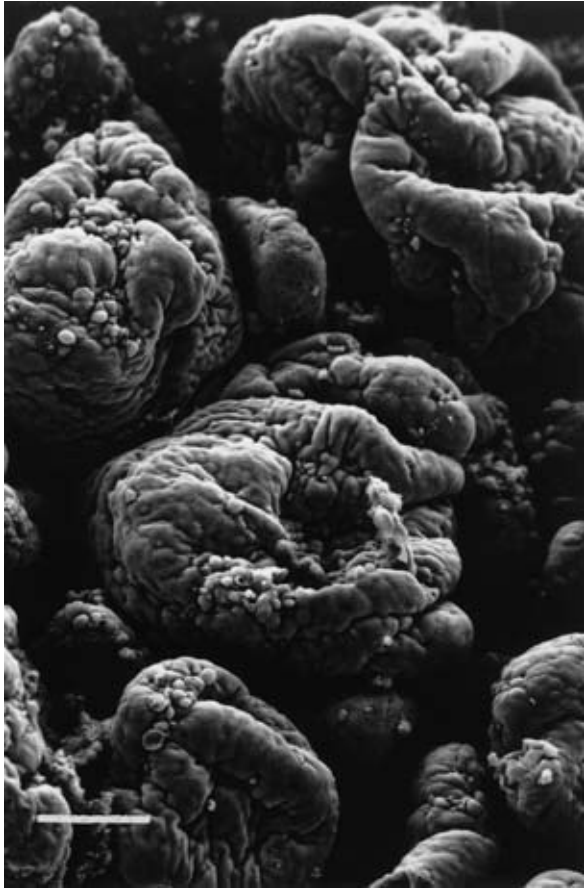
genes and several effector proteins which are secreted

infection with S. typhimurium. The peripheral microfolds of by TTSS-1. TTSS-1 can be viewed as a protein-the M cells are ruffled and bacteria (arrowheads) are being

aceous microsyringe that spans the bacterial inner and

engulfed by the membrane ruffles.

outer membranes. The main function of TTSS-1 is the



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Fig. 15.7

Transmission electron micrograph of dome villus

after infection with S. typhimurium. There are a large number of PMN leucocytes, one of which appears to be crossing the epithelial monolayer.

to several strains can be quantified in a single animal,

Fig. 15.6

Scanning electron micrograph of bovine ileum after

thus dramatically reducing the number of animals

infection with S. typhimurium. The villi are shortened and there required for such experiments. In infected ileal loops

is an abnormal extrusion of enterocytes.

there is an increase in the rate of epithelial cell

shedding, which could be viewed either as a bacterial

cytotoxic activity, allowing further invasion, or as a host

defence mechanism to eliminate infected cells rapidly.

co-ordinated secretion of specific bacterial proteins, not

An inflammatory response is initiated in the

only out of the bacterial cell, but also directly into the

intestines by the production of cytokines and other

target epithelial cell. The translocated proteins interfere

pro-inflammatory mediators by infected host cells. It is

with specific components of the host cell signalling

unclear whether this is a generalized host response to pathways and cytoskeleton, resulting in bacterial invading bacteria, which will release pro-inflammatory uptake and potentially influencing the subsequent molecules such as endotoxin, or if it is the result of induction of enteropathogenic responses.

the direct action of specific bacterial proteins acting on Infection of the intestines by *Salmonella* may result epithelial cells without necessarily requiring bacterial in several pathological changes (Figs 15.5–15.7). These invasion. It results in a large influx of inflammatory can be studied using the calf ligated ileal loop model, cells, particularly polymorphonuclear (PMN) leucocytes, which has been developed by researchers at the Institute for Animal Health, Compton. Results from this salmonellas are relatively resistant to the bactericidal model correlate well with those from orally-inoculated activity of PMN leucocytes and their influx may cause animals. Short sections of the ileum are sealed in situ considerable damage to the mucosa by disrupting

by surgical silk to form individual loops and each loop cellular junctions and releasing the contents of their is directly inoculated with up to 10⁹ salmonellas. This is granules extracellularly. The most striking pathological an appropriate inoculum size, since faeces from infected change is the increase in net fluid secretion by infected calves may contain up to 10⁸ salmonellas/g. The main mucosa. By 12 hours after inoculation with either *S.* advantage of this model is that pathological responses typhimurium or *S. dublin*, as much as 30 to 40 ml of fluid

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may have accumulated in a 6 cm ligated ileal loop. This in orally inoculated calves. The mechanisms of bacter-fluid secretion may initiate the physiological response ial spread from the epithelial monolayer and the way in of the infected host to eliminate infecting bacteria, but which intracellular persistence contributes to enteritis it will also be advantageous to the bacteria, because it are not known. Several of the host responses to inva-mediates their dispersal into the environment. sive bacteria may contribute to enteritis. For example,

This characteristic secretory response has led many investigators to look for enterotoxin activity associated with salmonellas. Despite numerous reports of enterotoxin activity in various extracts from salmonellas and movements, and the PMN leucocyte influx and associated vasodilation and mucosal damage may alter evidence that Salmonella produce a classical enterotoxin. A Salmonella gene has been cloned on the basis of its homology to the cholera toxin gene from Vibrio cholerae. However, mutation of this gene in either S. typhimurium or S. dublin did not reduce fluid secretion known into infected ligated ileal loops in calves. Despite the lack of evidence that Salmonellae produce a classical

Systemic spread and persistence

enterotoxin, there is growing evidence that they do produce proteins with enterotoxin activity, but which Salmonellas are able to spread rapidly to systemic sites, lack a receptor-binding domain and therefore require particularly the liver, lungs and spleen. In experimentally infected calves this may occur within 6 hours after oral inoculation. The best characterized of these proteins is called SopB, and it is dependent on TTSS-1 for translocation into mesenteric lymph nodes, the efferent lymph draining epithelial cells. SopB affects inositol phosphate eukaryotic cell signalling, which may lead to interference with circulation via the thoracic duct. Cannulation of chloride channel function and hence affect movement of electrolytes and water. Mutation of SopB in S. dublin lymph nodes has demonstrated that S. dublin significantly reduces fluid secretion and the influx of

nodes in efferent lymph. In general, the growth of *S.*

PMN leucocytes in calf ligated ileal loops. *SopB* is

typhimurium and *S. dublin* in the bovine liver and located within a small *Salmonella* pathogenicity island spleen appears to be relatively well controlled and in

called *SPI-5* and mutation of the other genes within

calves infected experimentally it is unusual to recover

SPI-5 also reduces enteropathogenic responses in

more than 10⁵ bacteria/g of tissue, even at the peak of

bovine ligated ileal loops, although the biochemical

disease. However, the bovine infection model most

basis of this is not known.

commonly used (four-week-old Friesian calves) usually

Elucidation of bacterial factors involved in the inter-

results in a predominantly enteric disease and may not

action of *Salmonella* with the intestines is advancing be an appropriate model for the systemic disease occur-rapidly, but there is still a lack of knowledge as to how

ring in some very young calves or adult cattle. The

Salmonella induces enteritis. In addition to the intimate pathogenesis of systemic disease has been mainly

interaction with epithelial cells, bacteria also spread

studied in mice and the results extrapolated to other

rapidly to the lamina propria and mesenteric lymph animal species. Natural resistance of mice to infection nodes, as well as being returned to the gut lumen by with *Salmonella* is controlled by a genetic locus called extrusion of infected epithelial cells. A significant pro-Nramp 1, which affects the ability of macrophages to portion of luminal bacteria appear not to invade during control intracellular bacterial growth in the reticuloen- the initial stages of infection. The presence of high dothelial organs during the early stages of infection. numbers of luminal bacteria may extend the symptoms Resistance to salmonellosis in cattle may also be under of enteritis by providing a continual source of bacteria genetic control and several researchers have com- to interact with epithelial cells. In addition, many of mented on the variations in susceptibility to salmonel- them will be expelled in the copious diarrhoea associ- losis amongst breeds of cattle. For example, Friesian ated with disease, and are therefore vital to bacterial calves are thought to be less susceptible than Channel spread between animals. *Salmonellas* that have invaded Island calves, although the genetic basis of this is not

the lamina propria and beyond may also contribute

known. A homologue of Nramp 1 has been identified in

to enteritis. For example, S. dublin carrying mutations cattle, but its contribution to susceptibility to salmonel-in TTSS-2, a second TTSS which is not required for lysis is not yet clear.

invasion of bovine intestines but is required for sub-

*Several bacterial factors involved in systemic spread
sequent intracellular persistence, are highly attenuated
and/or persistence have been identified. A variety of
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Salmonella mutants that are unable to persist within

Systemic spread and persistence may have several

*murine macrophages in vitro have reduced virulence in pathological
consequences. Release of endotoxin in sys-mice and several bacterial genetic
loci influencing*

temic organs may result in haemorrhages, leucopenia,

persistence, including those encoding TTSS-2 and the

leucocytosis, hypotension, hypoglycaemia and shock.

two component transcription regulators PhoP/PhoQ

Mutation of endotoxin to make it less inflamma-

*have been identified. Mutation of one such gene, slyA, tory reduces the mortality
in mice infected with S.*

is highly attenuating in mice but only slightly attenuat-

typhimurium without affecting the net rate of bacterial ing in calves, which highlights the differences in patho-growth in the liver and spleen. This suggests that fatal

genesis between these infection models. In contrast,

systemic infection, at least in mice, is due to the inflam-

the *spv* (*Salmonella* plasmid virulence) genetic locus is matory response of the host to endotoxin. Bacterial

required for systemic virulence in both mice and cattle.

spread to systemic sites may prolong enteritis by allow-

This locus is located on a high molecular weight

ing continual reinfection of the intestines via the biliary

plasmid, called the virulence plasmid, which is present

system and may also lead to systemic complications

in many *Salmonella* serotypes. The existence of large

such as pneumonia or abortion. Abortion may occur

molecular weight plasmids in many of the more impor-

before, during, after or even in the absence of enteric

tant serotypes of *Salmonella*, including *S. typhimurium*, symptoms. The placenta becomes infected and the

S. dublin, *S. choleraesuis* and *S. enteritidis* (but interest-subsequent rapid bacterial multiplication results in a

ingly not in *S. typhi* which causes the severe disease pyrexia response, severe damage to the placenta and

typhoid fever in humans), has been described for

possible infection and death of the fetus in utero or the more than 20 years. The plasmid harboured is charac-birth of infected calves. Systemic spread may also lead

teristic of the serotype and they were originally

to the development of the carrier state, characterized

called 'serotype-specific plasmids'. Naturally-occurring

by the continual faecal excretion of salmonellas.

plasmid-free strains and strains from which the plasmid

has been removed are avirulent in the mouse model

Signs of disease

of salmonellosis. In calves infected experimentally,

plasmid-containing strains cause enteritis and a

Adults

generalized systemic infection while plasmid-free

strains induce enteritis in the absence of the general-

The disease in adult cattle is usually sporadic, although

ized infection. It thus appears that plasmid genes are

S. dublin has become endemic in some areas of the

involved in the ability of salmonellas to proliferate

country and acute and subacute forms are recognized.

within the host and that in cattle both plasmid and

In the characteristically severe form of the disease

chromosomal genes are essential for the full expression produced by *S. dublin* in adult cattle the onset is of the disease. The virulence plasmid may not be usually sudden. Fever, dullness, anorexia and abruptly carried by all isolates of a given serotype, particularly depressed milk yield at first associated with firm faeces, *S. typhimurium*, and this may explain, in part, the which may contain blood, are rapidly followed by variation in the degree of severity of salmonellosis severe diarrhoea. The faeces often become watery and observed in the field where some outbreaks involve contain large numbers of salmonellas (up to 108/g). The enteritis only whilst in others this develops into fever usually persists for several days, then animals systemic infection.

rapidly become cold and recumbent and death, which The mechanism by which the *spv* genes contribute to may be preceded by abortion, occurs in approximately systemic virulence is not known, although one study 75 per cent of untreated animals between four and suggests the locus promotes bacterial replication in

seven days from the onset of clinical signs. In some vivo. The virulence plasmid does not contribute to animals the disease is more protracted and cattle may persistence within macrophages in vitro and although become emaciated and dehydrated and show signs of carriage of the virulence plasmid increases the lysis abdominal pain. A milder disease, characterized by of macrophages during Salmonella infection in vitro, diarrhoea and abortion, or abortion in the absence of this is not attributable to the spv locus. The lysis other clinical signs, from which animals usually recover, of macrophages during Salmonella infections in vivo also occurs. A similar disease is produced by other has received little study and the relative contributions serotypes including *S. typhimurium*, although in these of macrophage persistence and lysis to pathogenesis cases abortion is a less frequent event. Survivors of *S.* need to be assessed. The TTSS-2-dependent secreted dublin infections often remain as carriers, possibly for effector proteins are thought to promote bacterial net life, while infection with other serotypes seldom results growth in vivo by two distinct mechanisms: firstly by in the carrier state although excretion of types such as

promoting bacterial replication rates; and secondly by

S. saintpaul for up to two years after infection has been preventing killing of bacteria by macrophages.

reported.

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Calves

usually excrete large numbers of organisms in their faeces and determination of viable counts, rather than
In calves, disease usually occurs between two and six
enrichment cultures, should be used (see below). For
weeks after birth. Characteristically, calves become dull
this reason faecal samples rather than swabs should be
and anorexic and develop a fever. Diarrhoea follows,
taken and these should obviously be obtained before
which in young calves involves the excretion of faeces
administration of antibiotics. It may also be possible to
with the colour and consistency of putty. This may be
isolate the organism from oral secretions and by blood
bloodstained and may contain fibrin and mucus. Even-
culture, although these are less reliable than faeces cul-
tually, the faeces become dark brown and watery with

tures and must be taken with care to avoid contamination. More rarely the faeces are heavily bloodstained, become stringy due to the presence of undigested milk and pseudomembrane formation and out their tissues and samples of spleen, liver, hepatic, may contain shreds of necrotic intestinal mucosae. In mediastinal and bronchial lymph nodes may yield older calves the faeces are usually watery, dark brown counts in excess of 10^6 organisms/g. Similar concentrations and offensive. The calves become very weak and dehydrations may also be present in the wall and contents of ileum, caecum, colon and associated lymph nodes. of illness in untreated individuals.

Samples should be taken from the gut and internal organs in order to distinguish animals that have died of those two to three days old, bacteraemia and septicaemia. Samples of fetal fluids,

caemia occur and the animals collapse and die without vaginal mucus and cotyledons should be taken from diarrhoea. In other animals the disease may be so mild animals that have aborted. Counts will again give a as to pass unnoticed or may be associated with diarrhoea in the absence of systemic disease. Diarrhoea that have aborted due to *S. dublin* infection will usually may be prolonged in some calves, which may eventu-contain between 10⁸ and 10¹⁰ organisms/g. ally die as a result of dehydration, electrolyte loss and Identification of carriers by bacteriological examination acid–base imbalance. In both calves and adults systemic tion is more difficult and an attempt must be made to infection may lead to complications such as pneumonia, distinguish ‘active carriers’ (persistent excretors) from meningitis and osteitis; polyarthritis and gangrene may ‘passive’ carriers and ‘latent carriers’ (intermittent occur when the disease has been prolonged. Mortality excretors). Faeces samples are more reliable than from acute salmonellosis may be as high as 70 per swabs and should distinguish persistent excretors whose

cent and all calves in a herd may become infected.

faeces usually contain in excess of 10⁵ salmonellas/g.

Recovered calves do not normally appear to become

Animals should not be assumed to be excretors on the

carriers of either *S. dublin*, *S. typhimurium* or the exotic basis of one isolation, nor should they be considered to

serotypes and consequently infected calves do not grow

be free of infection unless at least three negative faecal

into infected adults. However, the disease is often slow

samples have been obtained. Even in this latter case

to resolve and salmonellas have been isolated from

‘latent carriers’ and animals that will eventually clear

calves up to six months after their initial infection. The

the infection, and yet are still harbouring salmonellas,

disease produced by different serotypes in calves is

will be missed. There may be an advantage in taking

usually similar, although the peak incidence of disease

samples from adults at calving although care must still

and mortality with *S. dublin* is at four weeks of age

be observed to distinguish ‘passive carriers’. Tradition-

compared with three weeks for *S. typhimurium*. Calves

ally, samples of gall-bladder or bile have been taken from dams infected with *S. dublin* may be infected from to identify carriers at slaughter. Unfortunately, these birth and these are particularly likely to succumb to may not always contain salmonellas unless animals are septicaemia rather than enteritis.

infested concurrently with *F. hepatica* and a variety of tissues including alimentary contents and superficial

lymph nodes need to be examined. It may be possible

Diagnosis of salmonellosis

to isolate salmonellas from the walls of the omasum, abomasum and rumen or from lymph nodes such as the It will be apparent from the description above that diagnosis of salmonellosis presents several difficulties. The node when all other tissues are negative. Similarly, sal-clinical signs and findings at post-mortem examination monellas have only very rarely been isolated from the are not unique to salmonellosis and although a tonsil although this has been the preferred site for some tentative diagnosis may be made this should be confirmed in investigators. Identification of infected herds may be

diseased animals or at necropsy by isolation of the organism. Diseased animals showing signs of enteritis achieved by faecal samples but this may present similar difficulties to identifying individual infected animals, *Salmonellosis* • 227

particularly in the case of *S. dublin*. A simple, less expensive and probably more reliable method is to take restriction fragment length polymorphism) and AFLP samples of slurry where this is available.

(amplified fragment length polymorphism where DNA fragments are amplified by PCR). Although used for media are available for the cultivation of salmonellas. research purposes, a phagotyping scheme for *S. dublin*

They rely on promoting the selective growth of salmonellas, whilst inhibiting the growth of contaminants, and 26 groups of *S. dublin* by phagotyping but more than 50 per cent belonged to one group) and this serotype

biochemical reactions. The choice of media depends is subdivided into biotypes. Unfortunately, although upon the environment from which the organism must seven distinct, stable biotypes have been identified, the be isolated and often depends upon the subjective majority (approximately 75 per cent) of strains isolated choice of bacteriologists who specialize in *Salmonella* in the UK belong to the same biotype and the scheme isolation. In general, a range of liquid enrichment is of little use in epidemiological investigations.

media and the use of at least two types of solid isolation media are necessary to guarantee the isolation of an attempt to identify *Salmonella*- infected animals. A number of serological techniques are also used in the majority of serotypes. Enrichment media depend Unfortunately, no single test has proved useful in distinguishing all infected animals, some of which do not upon allowing salmonellas to grow, albeit sometimes in an inhibited manner, whilst suppressing other bacteria mount an antibody response, and all suffer from an through the action of chemicals, dyes, antibiotics and

inability to distinguish infected animals from others enhanced incubation temperature. In common use are which, although previously infected, no longer harbour media containing sodium selenite or tetrathionate and the organism. Various techniques such as ELISA dyes such as brilliant green or malachite green to which have been developed but they are usually no more successful than more traditional methods and are not

After enrichment in liquid culture, colonies of

in routine use.

Salmonella are isolated on solid selective agars such as MacConkey, Brilliant Green, Deoxycholate citrate,

EF-18, Rambach, Hektoen, Bismuth sulphite and

Control measures and vaccination

Salmonella–Shigella. These rely on the resistance of salmonellas to a variety of antibiotics, bile salts and

Attempts to control salmonellosis in cattle have

dyes, which inhibit other bacteria, and lack of ferment-

involved the use of strict hygiene measures, antibiotics

tation of sugars such as lactose and sucrose to differen-

and vaccination, either singly or in combination. Most
tiate colonies of *Salmonella* from other bacteria which of the control measures
are obvious from the discussion
grow on the media despite the presence of inhibitors.
presented above and the role of antibiotics and vacci-
More recent techniques include the use of immuno-
nation is described below. To prevent the introduction
magnetic separation (IMS) and PCR (polymerase chain
of salmonellosis to herds it is necessary to provide
reaction) or fluorescence PCR, but the use of these is
animals with uncontaminated feed and water, to control
usually confined to research laboratories. Once iso-
ingress of rodents and birds and limit human contact.
lated, the identification of salmonellas to genus level is
When adult stock have to be 'bought in' these should
carried out on the basis of biochemical reactions for
be quarantined and examined bacteriologically. Exac-
which a variety of rapid kits are available. Further
erbing factors such as ketosis and liver fluke infesta-
identification is carried out by slide and tube aggluti-
tion should be controlled and particular care should be

nation tests, which define the serotypes according to taken in maintaining the health and hygiene of animals the Kauffmann–White scheme. Since a large number of at calving. This should include the use of individual differential sera are required this is usually the province calving boxes and thorough cleaning between animals. of dedicated laboratories.

Calves should be encouraged to absorb sufficient Important serotypes such as S. typhimurium and S. colostrum. To prevent acquisition of infection in calf-enteritidis may be further subdivided by phagetyping rearing units only good quality calves from herds of (there are more than 200 phagetypes of S. typhimurium known health history should be purchased and strict but one or two phagetypes predominate in the UK at hygiene should be observed during feeding and clean-any one time, although 20 to 30 are usually present), ing. Units should be run on an all-in, all-out basis and plasmid analysis or a variety of molecular techniques calf houses should be thoroughly cleaned (preferably including ribotyping (rRNA gene restriction patterns),

by steam-cleaning, disinfection or fumigation) between PFGE (pulse-field gel electrophoresis), IS200 typing (a batches. In the event of a disease outbreak, affected method of comparing restriction patterns of DNA after animals should be quarantined and restrictions should hybridization to a probe to a unique (IS200) Salmonella be placed on the movement of susceptible stock.

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Human contact with infected animals should be allowed to graze pasture dressed with slurry for at restricted and strict hygiene should be observed when least six months after spreading.

moving between groups. During the course of an exten-

(4)

*The principal danger to cattle occurs when fresh sive outbreak of salmonellosis due to *S. saintpaul* in two unstored slurry, particularly pig slurry or poultry*

large dairy herds many methods to control infections manure, must be applied to pasture. This danger and reduce mortality were attempted. These included can be reduced by using low application rates and

the use of an emergency vaccine, which had no effect by leaving pasture as long as possible before on mortality but reduced the duration of faecal excre-grazing.

tion, and treatment with oxytetracycline and ampicillin,

(5)

Consideration should be given to mechanical which reduced mortality and the number of persistent separation of slurry. This produces a solid frac-excretors. Other measures included restriction of move-tion that readily composts and a liquid fraction ment of stock and personnel, the use of impermeable in which salmonellas die off rapidly.

clothing and disinfectant sprays, removal of predispos-

(6)

Slurry should not be spread by equipment which ing factors such as dietary imbalance, reduction of involves the production of spray.

stocking densities, segregation of susceptible animals,

These recommendations were designed to reduce the prompt antibiotic treatment of sick animals and

risk of infections from animal wastes but they may be removal of 'persistent excretors'. These measures were applied, with minor modifications, to the disposal of designed to reduce the level of environmental contamination. This is primarily due to contamination with the water authorities disposing of sewage sludge produce faeces of diarrhoeic animals and 'persistent excretors' their own codes of practice that should be followed. from which calves may be infected either at birth, particularly when communal calving facilities are used, or when housed in the same buildings as adults.

Vaccination (see p. 1006)

Once infected, calves may excrete such large numbers of organisms that less than 0.1 g of faeces may contain Prevention of salmonellosis by vaccination has been a lethal dose for other calves. Thus any measure attempted since the late nineteenth century. There has been considerable scientific debate on the relative to break the cycle of infection. Methods that appeared

importance of humoral and cell-mediated immunity, successful in this particular outbreak were prompt but it is now generally agreed that solid immunity probably depends on both humoral and cellular responses. carriers, reduction in stocking densities and segregation Although the humoral response is not totally protective of susceptible animals.

it plays an important role in the suppression of infection Salmonellosis may be spread from farm to farm and in its early stages. However, a cellular response is amongst cattle on infected premises by the disposal or probably required for complete elimination of the use of animal wastes on pastures as a fertilizer. The organism. In consequence, both live and killed vaccines number of organisms contained in such materials is have been used for the prevention of salmonellosis in usually low and the risk can be reduced to an acceptable level if sensible restrictions are observed. The actively and passively by transfer of maternal antibody

*following code of practice designed to reduce the
in colostrum. In the UK only an inactivated vaccine is
number of salmonellas should be followed:
available. The inactivated vaccine, which is licensed for
use in both adults and calves, is a formalin-killed prepa-*

(1)

*When possible, slurry should be spread on arable
ration of S. dublin and S. typhimurium. This vaccine has land or land used for
conservation. Salmonellas do
been the subject of recent field and experimental tests
not survive for long enough on grass to be a
and has been shown to be effective when used to induce
danger in hay, nor do they survive efficient silage
antibodies in adults, which could be transferred to
making.
calves in colostrum.*

(2)

*Composted waste may safely be spread on pasture
The majority of recent attempts at developing new
to be grazed by animals but slurry should be
vaccines have utilized live, attenuated Salmonella*

stored for at least one month prior to spreading.

strains. These have either been naturally occurring

(3)

Pasture treated with stored slurry should not be

avirulent strains, laboratory-derived strains, sponta-

grazed for at least one month after spreading. If

neous undefined mutants or, more recently, geneti-

the slurry cannot be stored, this interval between

cally-manipulated strains. Amongst the first of these,

spreading and grazing should be extended. In

originally produced in the USA, were strains in which

addition, since young animals may be more

mutations were introduced into genes encoding

susceptible to salmonellosis they should not be

enzymes in aromatic amino acid biosynthesis pathways

Salmonellosis • 229

known as Aro deletants or Aro strains. Such strains

are responsible for the delivery of effector proteins into

are unable to synthesize chorismate from which p-

host cells, which influence Salmonella intestinal inva-aminobenzoic acid, a precursor of folate, and dihydrox-

sion, induction of inflammation, fluid secretion, bacterial
ybenzoate, a precursor of the iron-binding protein
replication and avoidance of host defence mechanisms
enterochelin, are produced. In the absence of p-
(vide infra). Targeted mutagenesis in *Salmonella* that aminobenzoic acid and
dihydroxybenzoate, which are
results in the disruption of genes influencing *Salmonella*-
not found in mammalian tissues, they will not grow. Aro
induced inflammatory responses and net bacterial
strains of *S. dublin* and *S. typhimurium* have been pro-growth, but not invasion,
can optimise the phenotype of
duced and tested experimentally in calves. They induce
vaccine strains such that they remain invasive, and thus
good protection when administered orally or parenter-
immunogenic, but not virulent.
ally, are claimed to cross-protect against challenge with
Since calves may become infected within the first
heterologous serotypes and are claimed to be safe. They
few days after birth and the peak of mortality occurs
do not establish in the host, do not cause side-effects and
between three and four weeks of age, passive protec-
are genetically stable. Their only disadvantages are that

tion by vaccination of adult animals would appear to be not all constructs are equally protective and that they the ideal way of protecting calves. Unfortunately, may not be able to protect calves less than three weeks attempts at passive protection have met with conflict-of age. More recently, constructs deficient in more than ing results. However, an experimental vaccination one enzyme or constructs with auxotrophic and other regimen based on dam vaccination and prolonged mutations have also been produced. These strains, which colostrum feeding has been described. Cows were because they have two or more independent mutations vaccinated with formalin-inactivated *S. typhimurium* are thought to be safer than strains with a single muta- approximately seven weeks and two weeks preparturi- tion, have also been shown to be effective vaccines for tion. Calves were given the opportunity to suck from calves and may even be able to protect animals within their dam for 48 hours following parturition and were the first month of life. As yet none of these strains are then fed cold, stored colostrum from their own dam for

available commercially for use in the UK.

a further eight days. These calves were resistant to a

It is also probable that such strains are not able to

normally lethal challenge of S. typhimurium or S. dublin protect against infection with a variety of serotypes. No

given five days after birth and excreted salmonellas in

study to date has shown good cross-protection between

their faeces for only a short period after infection.

different serotypes for any significant period of time

Mortality was also reduced in calves that sucked from

after vaccination. In one recent study wild-type S.

a vaccinated dam and were then fed on normal colo-

typhimurium and S. dublin strains were used to infect strum and in calves born to unvaccinated cows and

ligated loops of calves previously immunized with

later fed on 'immune' colostrum. In cases of outbreaks

either serotype. Although protection against bacterial

with exotic serotypes the use of emergency vaccines

invasion and induction of enteropathogenic responses

for control has been successful.

was demonstrated it was clearly serotype-specific,

Vaccines may have an important role to play in the

despite the two serotypes being closely related and prophylaxis of bovine salmonellosis, but until improved sharing common virulence mechanisms. It is difficult vaccines become available they will not be a substitute to imagine how effective cross-protection between for good husbandry and hygiene.

serotypes may be obtained in future. It may be possible to include more than one serotype in a live vaccine.

Recent evidence suggests, however, that distinct vaccine

Antibiotic therapy

strains can interfere with each other when used in combination. Such interference could compromise efficacy.

It is not intended to discuss treatment of animals

Another problem with live vaccines that depend for infected with salmonellosis in detail here since this is attenuation on metabolic defects is that they are still based on the use of antibiotics and fluid replacement equipped with a full battery of virulence mechanisms

therapy as described earlier (p. 195) . Although antibi-and may in some circumstances still be able to cause

otics are not used in the treatment of gastroenteritis in

disease such as diarrhoea either in vaccinated animals, humans their use is justified for bovine salmonellosis, animals in contact or, in the case of food animals, in the which can become a systemic disease with a high mor-human population who later consume their meat. A tality in the absence of treatment.

common problem with live vaccines has been mild diar-

The concern with the use of antibiotics is that they rhoea and excretion of the vaccine strain in the faeces of have the potential of predisposing to colonization with vaccinated animals. Recent research on the mechanisms salmonellas, increasing levels and duration of excretion by which salmonellas induce enteritis may alleviate this by carriers and of selecting for antibiotic-resistant problem. *Salmonella* type III protein secretion systems strains (see p. 210). The effect of antibiotics on an

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animal and its resident micro-organisms will depend

Summary

upon the dose level, duration of administration and antimicrobial spectrum of the antibiotic. The effects of

- *Salmonellosis is a disease which ranges in man antibiotics to which salmonellas are sensitive at therapeutic doses must be distinguished from the effects of severe enteritis (with complications) to mild food poisoning.*

and the effects at either concentration of antibiotics to

- *The disease has a worldwide distribution.*

which salmonellas are insensitive. Similarly, since the

- *Salmonellosis is caused by bacteria of the genus antibiotic may have an effect upon the salmonellas or Salmonella, of which over 2400 types (serotypes) the normal flora or both, the age of the animal host is have been identified.*

important since age is often the principal factor in

- *The disease is characterized by host specificity. determining the composition of the normal flora. An Some types cause disease only, or primarily in one antibiotic to which salmonellas are sensitive, used at the animal species, whilst others are ubiquitous and*

correct therapeutic dose, may be predicted to reduce cause disease in many species.

excretion. The same antibiotic at a subtherapeutic dose

- The disease in animals in the UK is most severe in may lead to increased multiplication of salmonellas due cattle and particularly calves, and can result in high to a suppressive effect on the activity of growth-sup-morbidity and mortality. The principal serotypes

pressing normal flora. Similarly, the use of an antibiotic

involved are *S. dublin* and *S. typhimurium*. The to which salmonellas are resistant, but the normal flora

former is endemic in dairy herds, particularly in the

sensitive, may be expected to result in increased multi-

west of the UK, and may be maintained in herds

plication and excretion.

by 'carriers'. The disease is also frequently mild or

Antibiotic therapy in uncomplicated *Salmonella*

characterized by carriage of salmonellas in the

gastroenteritis in humans has not been recommended

absence of disease.

for more than 40 years. Therapy apparently does not

- *The disease is controlled in cattle by vaccination shorten or otherwise alter the clinical course of infection and the application of strict hygiene.*

tion and has frequently been shown to prolong

- *Some serotypes, and particularly S. typhimurium, postconvalescent excretion. However, antibiotics have have developed resistance to a number of antibiotics been used extensively to treat salmonellosis in cattle and multiple antibiotic resistance may be transferred without undue complications. It should be clear from*

ferred between strains.

the remarks above that the antibiotic sensitivity of the organism should be determined and the information used to choose the preferred antibiotic. The use of antibiotics for prophylaxis is more controversial and most authorities have reported that medication with

Further reading

antibiotics has no part to play in the prevention of salmonellosis. The use of antibiotics in the treatment of an

Wallis, T.S. & Galyov, E.E. (2000) Molecular basis of outbreak of S. saintpaul infection has been described Salmonella-induced

enteritis. Molecular Microbiology,

above. In the same outbreak antibiotics were success-

36, 997–1005.

fully used prophylactically and have also been used to

Wray, C. & Wray, A. (2000) Salmonella in Domestic Animals.

effect a bacteriological cure in adult carriers and calves.

CAB International, Wallingford, Oxon.

Chapter 16

Digestive Disorders of Calves

R.W. Blowey

Introduction

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within minutes of ingestion. The clot contracts, express-

Abomasal milk clot failure and milk scour

231

ing the whey proteins (albumin and globulin), minerals

Problems with milk substitutes

232

and lactose in liquid whey, which begins its passage into

Oesophageal groove dysfunction

233

the duodenum 5–10 minutes later. After two to three

Ruminal bloat

234

days the number of parietal cells in the abomasal

Signs

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epithelium increases and they begin to secrete

Treatment

235

hydrochloric acid. Abomasal pH falls. This converts

Rumen impaction and ‘pot-bellied’ calves

236

Abomasal ulceration

236

pepsinogen secreted by chief cells into the active form

Signs

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of pepsin. Both pepsin and renin are capable of clotting

Treatment

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milk and both can digest the milk protein casein, but

Abomasal dilatation

237

pepsin is most effective at pH 5.2 and, additionally, can

Chronic peri-weaning diarrhoea

237

digest a wider range of proteins. The pepsin digestion

Colic

238

system is not fully developed until approximately seven

Necrotic enteritis

238

to ten days old and until that stage calves should,

ideally, receive either whole milk or a substitute con-

Introduction

sisting of whole milk. Similarly, the very young calf has

an immature intestinal digestive system. Pancreatic

The young calf is particularly susceptible to digestive

proteases can cope with whey proteins, pancreatic

disorders, with the major presenting clinical sign of

lipases with fat and intestinal lactase degrades the milk

scouring (diarrhoea) being a significant cause of eco-

sugar lactose into glucose and galactose. The ability to
nomic loss due to ill-thrift and death. Immediately after
digest starch does not develop until the calf is at least
birth the calf is exposed to a wide range of infectious
seven days old and full activity of maltase, sucrase and
agents, to which it must develop an immunity. Many
amylase systems, allowing the calf to digest non-milk
calves are hand-reared and consequently within a few
carbohydrate, is not complete until three weeks old.
days their diet may change from whole milk to milk
Figure 16.1 is a flow diagram of digestion in the young
substitute. Later they must learn to eat solid food, to be
calf. Complete digestion of the abomasal milk clot (or
weaned at six to eight weeks old. Such rapid changes of
'curd') by lipases and proteases may take 6–8 hours and
feed and feeding systems, in combination with a chang-
hence calves fed twice daily are without nutrients for
ing immune status, render the calf susceptible to a wide
only a short period. Calves left with their dams may
variety of infectious diseases and nutritional disorders.
suckle seven to ten times each day and any remaining

Infectious conditions are described elsewhere in this book. curd forms a nucleus for the next milk clot.

book (see Chapters 14, 15). This chapter discusses the If abomasal milk clot formation is poor, then whole common nutritional disorders.

milk spills over into the duodenum, where casein cannot be digested. In addition to altering the osmotic balance, this provides an excellent medium for bacter-

Abomasal milk clot failure and

ial fermentation in the lower intestine and scouring
milk scour

results. Similarly, if excessive quantities of milk are provided (greater than 1.5 l for the young calf), or if a Abomasal volume in the newborn calf is 1.0–1.5 litres.

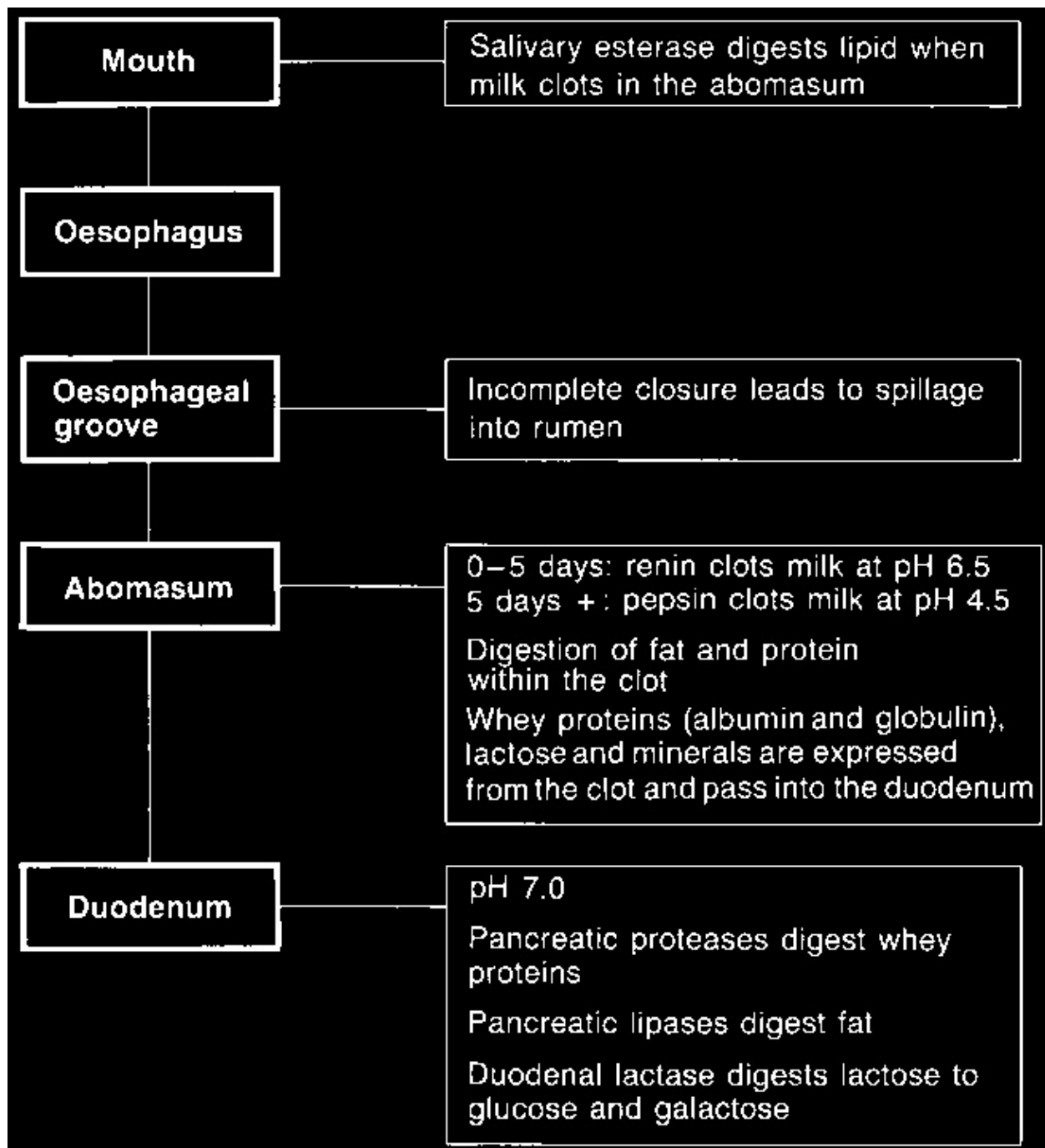
hungry calf is allowed to gorge itself, then abomasal

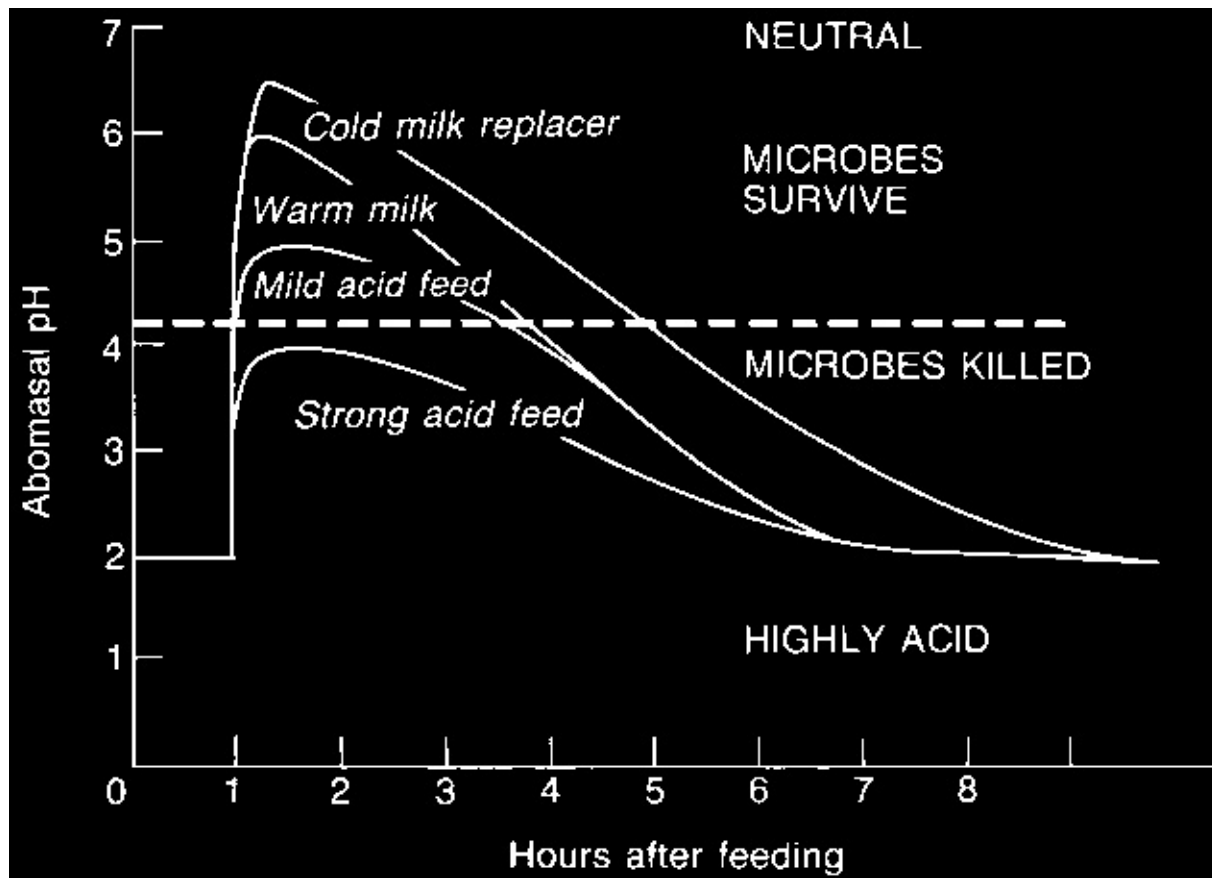
It has a neutral pH, thus allowing the first feed of digestive processes become overloaded and whole milk colostrum to pass through unclotted,

so that

spills into the duodenum. This can also be a problem immunoglobulin molecules can be absorbed whole

with suckler calves, especially if the dam is a 'milky' through the intestinal epithelium. Initially, renin coagulates milk (optimally at pH 6.5), with clot formation calf may suckle every few hours, overloading and





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Fig. 16.2

The effect of type of milk and milk substitute on abomasal pH (Webster, 1984).

for the first few days of life has an abomasal pH of 6.5, is particularly susceptible to infection therefore.

However, this is counteracted by surface-active antibodies contained in whole milk and colostrum. In the older calf, the acidity of the empty abomasum, i.e. after

Fig. 16.1

Flow diagram of digestion in the young calf.

complete digestion of the curd 6–8 hours after the previous feed, may fall to as low as pH 2.0. Following a feed of milk, pH rapidly rises towards neutrality (e.g. pH 6.5), the extent and persistence of this rise depending on the volume and nature of the liquid milk ingested.

On bucket-rearing systems a variety of management

With warm whole milk, abomasal pH falls to below factors can lead to poor abomasal milk clot formation.

4.2 within three hours of a meal and bacterial killing

These include:

once again becomes effective. Figure 16.2 indicates the periods required for other feeds.

- *Nervous or stressed calves, for example feeding*

Clearly, calves that develop a poor abomasal milk immediately after arrival from market, dehorning clot, or that have other abomasal disorders (e.g. abomasal dilation, p. 237), resulting in increased pH values,

- *Irregular feeding times.*

will be more susceptible to infection. In addition,

- *Milk substitute fed at the wrong temperature or improperly digested fats passed in the faeces produce incorrect strength (see p. 233).*

steatorrhoea, with subsequent hair loss over the legs

- *Inflammation of the abomasum (see p. 237).*

and perineal region (Plate 16.1). Affected calves will be

Many enzyme systems are induced, that is, enzyme

unthrifty and in poor condition, due to poor absorption

activity develops following exposure to substrate.

of nutrients.

Calves should therefore be reared on a single type of

Treatment with oral electrolytes for two to three days

milk substitute and, preferably, from a single batch pur-

and dietary correction is normally effective.

chased to cover the whole rearing period. A sudden

change in the ingredients of the milk substitute could

lead to incomplete digestion of protein, fat or carbohy-

Problems with milk substitutes

drates, which later undergo putrefactive fermentation

in the lower intestine, resulting in malabsorption, a fetid diarrhoea and weight loss. Such calves are more susceptible to colonization by pathogenic *Escherichia coli* and salmonellae.

Because of the immaturity of the pepsin-HCl system, irregular feeding and its influence on abomasal pH calves should receive colostrum and whole milk for the first three to four days of life. Most conventional milk substitutes are based on skim milk powder (SMP), to an abomasal pH below 4.5. The very young calf, which is added lipids and fats from a variety of sources,

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all highly emulsified to ensure even suspension fore, be fed 'half strength' for scouring calves, as this throughout the milk when it is reconstituted. These may retard clot formation. Although some proprietary

SMP substitutes should clot in the abomasum. Addition of electrolyte preparations state that they can be used with whole milk and improve clotting time, there is some evidence that the pH to 5.7 and thus improves keeping quality. Fully acidified milk powders, with a pH of approximately 4.2, therefore, it is best to avoid diluting milk substitutes have an even better keeping quality. As casein would and if electrolytes are to be given, they should be fed separately. Similarly, calves should not be allowed to drink large volumes of water immediately after milk, as powders primarily consist of whey proteins (albumin and globulin) with added fat. Without casein they do this will have the effect of diluting the milk.

not clot in the abomasum, but this does not seem to Probably the biggest problem with milk substitutes produce any increase in digestive problems. Such 'zero' comes from careless mixing. Stirring with the hand is replacers (so-called because they contain no skim milk

simply not adequate: a whisk, whether manual or powder) are commonly used in ad libitum calf feeding mechanical, is essential. Carelessly mixed powders systems.

leave lumps and a sediment of protein in the bottom of

If skim milk powder is overheated during manufac-

the bucket and poorly dispersed fat rises to the top.

ture, casein is denatured, abomasal milk clot formation

Trials have shown that up to 60 per cent of the oils in

is poor, undigested casein passes into the small intestine

a replacer may be wasted in this way, producing

and scouring may result from putrefactive bacterial

poor growth, stunted calves and possibly scouring due

fermentation. Most proprietary milk substitutes are

to inadequate abomasal clot formation.

carefully monitored and the majority of problems

associated with their feeding are managerial in

origin. The first golden rule must be to read the manu-

Oesophageal groove dysfunction

facturer's instructions. To achieve even dispersal of the product, and especially of the fat, many manufacturers

From as early as two weeks of age the young calf begins to pick at hay, grass or other solid foods. Bacteria (45–50°C) and then cooled to just above blood heat (42°C) before feeding. A thermometer is needed to do this. The temperature cannot be judged manually. On a hot day milk will feel cool and consequently may be fed to adult ruminal flora eliminates the early organisms. The milk substitute consequently fed too cold. Milk substitute mixed below the optimum temperature produces an inferior product. Added protein in the rumen fermentation alone would be unable to provide adequate nutrients for the rapidly growing calf and are wasted. In addition, poorly dispersed fats remain as

milk needs to continue 'monogastric' digestion in the
 a layer on the surface of the milk, forming a ring around
 abomasum. Milk entering the rumen is both wasteful
 the calf's muzzle after feeding, which produces second-
 and dangerous and hence it should bypass the rumen
 ary alopecia (Plate 16.2). If a long row of calves is fed
 and reticulum to enter the abomasum direct. This is
 from a single container, the milk for the last calf may
 achieved via the oesophageal groove. In the anterior
 be appreciably cooler. Again fat will rise to the surface
 wall of the dorsal sac of the rumen there is a muscular
 and alopecia may develop on the muzzle. A fall of only
 channel that runs from the distal end of the oesopha-
 6°C in the temperature of the milk fed will double the gus into the rumino-
 omasal orifice, as shown in Fig.

time taken to form the abomasal clot. Undigested milk

16.3. A reflex action from suckling results in muscular
 may then leak from the abomasum, thus reducing its
 closure of this groove, to form an enclosed pipe, and
 nutritive value and possibly inducing diarrhoea. Con-
 milk is then transferred directly into the abomasum. As

versely, however, if milk is fed too hot calves simply will not drink it, but no adverse effects have been observed. sight, sounds and stimuli associated with the arrival of It is interesting to compare this with our own liking for its milk will be sufficient to evoke closure. It is most hot drinks.

important that closure occurs prior to feeding and Milk fed at the wrong strength can cause problems. hence the establishment of a standardized feeding Powder is normally added at 125 g/l for twice-daily regimen is vital. The calf needs to know that it is about feeding and 150 g/l on once-daily systems. Abomasal to be fed, that a pleasurable event is forthcoming. milk clot will be optimal at these concentrations. If too Ideally the calves should be able to both see and hear dilute, clot formation is poor. Milk should not, there- the feed being prepared, as this will induce a state of

should be provided at the correct and consistent way below pen level. Teats for milk substitute should temperature, in the same amount at each feed and be positioned such that the calf has sufficient space to of a similar taste and consistency. Calves with wagging stand back to suckle and at a height that allows its tails and calves which butt the bucket (or udder, or teat) nose to be tilted upwards and the oesophagus at least are enjoying their food and the oesophageal groove is horizontal.

well closed.

Milk spilt into the rumen undergoes rapid fermenta-
Calves on a teat need to work reasonably hard to get
tion. This may produce an acute and sometimes fatal
their milk – a pile of saliva beside the teat is a good sign.
bloat and colic, within 15–30 minutes of feeding. In
Conversely, if milk flow is too rapid, for example a badly
more chronic cases, ruminal development is retarded
damaged teat with an open orifice, the calf will almost
and abnormal products of digestion pass into the
choke with the rate of flow and milk could spill into the

intestine, producing scour. Chronic pain restricts food rumen. A sawn-off milking machine liner fitted onto a intake and therefore growth and development. The bottle and used by some farmers as a teat produces a Charolais calf in Plate 16.3 has a typically distended much too rapid flow rate. Stressed calves, for example left flank caused by ruminal bloat. The superficial those that have just arrived from market, may not haemorrhage resulted from trocarization with a needle. achieve groove closure and should therefore be given A pasty scour is evident on the tail.

electrolyte solutions for their first feed. Similarly, calves Treatment of oesophageal groove closure is, in effect, should not be moved, handled, dehorned, etc. immedi-treatment of chronic bloat and is discussed in the next ately before feeding. Slow drinkers present a problem. section.

While the milk is warm, oesophageal groove closure may be adequate. However, if the bucket is left in front of the calf it may drink later, with possible milk spillage

Ruminal bloat (see also p. 832)

into the rumen. Some calves fail to learn to drink adequately and must be finger or teat suckled until

The development of normal rumen function is a weaning, although this is preferable to reluctant drinking complex process and any interference can produce bloat. Bucket height and teat position are important. The bottom of the bucket must be at least 30 cm above the floor of the pen, which means that it must be raised as straw bedding accumulates. It is the ingestion and subsequent fermentation of solid feed that stimulates the

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rapid expansion in size of the rumen, although full

Signs

development is not complete until 12 weeks old, when Typically, bloat is seen 15–30 minutes after a feed of the rumen comprises approximately 80 per cent of the milk or concentrate. Mild cases may deflate spontaneously volume of the four stomachs. During this period,

neously over 3–4 hours, as ruminal contractions redevelop of the rumen papillae is affected by the type of food offered. Inadequate long fibre and fermentation of a high-concentrate diet leads to low ruminal pH and acidosis. This can restrict development of papillae to such an extent that inflammation and colic, often leading to kicking at their abdomen. Many ulceration of the ruminal wall permits bacterial develop a chronic scour (see colour plate 16.3), leading ‘leakage’ and subsequent hepatic abscessation. The addition of 10–15 per cent chopped straw to concentrates, especially high-starch products, will improve rumen fermentation, thereby increasing the overall digestibility of the ration and promoting increased dry

trates, especially high-starch products, will improve rumen fermentation, thereby increasing the overall digestibility of the ration and promoting increased dry

Treatment

matter intake, since high acidity depresses the activity of cellulolytic bacteria. Calves that do not have hay on offer should therefore be bedded freshly each day, with postweaned calves and treatment is similar, the method used depending on the severity of the condition. Acute bloat must be deflated, either using a large bore needle both neutralizes and dilutes ruminal acidity. Long fibre through the rumen wall or, preferably, by stomach tube. essentially acts as a 'scratch factor' and stimulates rumen motility. Oral antibiotic will depress gas production. Penicillin may be the drug of choice, since lactobacilli proliferate it seems critical that the fibre length is 5.0 cm or above. in both concentrate overload acidosis and milk fermentation. Withdrawal of food and feeding electrolyte solutions for two to three days helps to remove the fer-

tation of the diet, as shown in Table 16.1, and with menting substrate. Calves should then be returned to a dietary constituents. High-forage diets produce greater liquid milk diet for two to three weeks and eventually ruminal activity than high concentrate. It has been suggested that concentrate intakes in heifer calves being cases recur and in many instances this protracted treatment is, for a variety of reasons, impractical. In such this will encourage greater consumption of forage and cases the construction of a permanent ruminal fistula in the development of a larger rumen, capable of sustaining greater intakes later in life.

rib and the lumbar transverse processes is indicated.

Irregularities at any stage of rumen development can

Under local anaesthesia, the rumen wall is sutured to

lead to bloat, but the two most common causes are

the skin and a fistula is created. Although rumen con-

oesophageal groove failure, with subsequent fermenta-

tents may spill onto the flank, this does not appear to
tion of milk in the rumen, and acidosis caused by high-
upset the calf and in many animals the improvement
level feeding of improperly formulated concentrates. In
in growth and general condition following surgery is
both cases the bloat is caused by ruminal atony.

dramatic. A proportion of fistulae seal spontaneously
after six to twelve months. Others require surgical
correction, although this is not normally performed
for beef animals.

On farms where bloat is a common problem, feeding

Table 16.1

*Effect of the physical form of diet on ruminating
behaviour of calves 6–9 weeks old (from Webster 1984, adapted
and management systems should be examined, espe-
from Hodgson, 1971).*

*cially in relation to the encouragement of early ruminal
development. Highly nutritious concentrates should be*

Grass Long

or

offered from two to three weeks old onwards, prefer-

pellets

chopped grass

ably in small amounts each day, to ensure freshness and palatability. Water should be freely available, especially

Eating (min/24 h)

132

276

on once-daily milk feeding. Inadequate access to water

Ruminating (min/24 h)

138

459

restricts concentrate intake and occasionally a long

Eating (min/kg DM)

85

320

drink by a thirsty calf with a dry rumen leads to rapid

Ruminating (min/kg DM)

70

535

fermentation of concentrates and subsequent bloat. In

Dry matter intake (g/24 h)

1553

862

this respect the placing of buckets is important. Water

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*should always be freely available, but the calf must be
as the left flank, but overall body condition, in terms of
‘programmed’ to regard it as a drink, namely without
fat and muscle cover, will be poor. Ruminal contrac-
stimulating the oesophageal groove closure reflex.*

tions may be slow and the rumen feels ‘doughy’ on pal-

However, milk is a food and as soon as the calf has fin-

pation. In an extreme case, where poorly grown calves

ished, replace the milk bucket with another containing

left without food for many hours were allowed unlim-

concentrate. (Many farmers leave the milk bucket in

ited access to palatable straw, the author has seen

*situ – to avoid the transfer of infection – and simply add severe illness and even
death due to ruminal impaction.*

the concentrate bucket.) The calf immediately puts its

However, this is rare. Rumen impaction is almost the

head in and starts to eat, thus encouraging concentrate

reverse of bloat, in that it is associated with overconsumption and, at the same time, discouraging excessive consumption of dry, fibrous and relatively indigestible water drinking. Even so, occasional calves will still eat foods, sometimes compounded by inadequate access to water. It may be a group problem of poor dietary management when they do so. In such cases, water is best withheld until 2–3 hours after the feed of milk, accepting the risk the smaller members of the group get pushed out and of bloat following rapid fermentation of dry ingested have to exist on forage alone.

concentrates.

Abomasal ulceration

Rumen impaction and

‘pot-bellied’ calves

Abomasal ulceration is common in artificially reared calves, the majority of cases being asymptomatic and Postweaning feeding is a critical stage of the calf’s seen only at post mortem (for example in veal calves).

development. The rumen is not fully mature, being
Surprisingly, perhaps, the incidence is higher in those
more acid (i.e. lower pH) than the adult and unable
veal calves that have had access to forage. Many clini-
to synthesize sufficient microbial protein, relative to
cal cases are seen at two to three weeks old, when they
energy, to meet the calf's high requirements for growth.
first start eating solid food, or in animals that have
Prewaning, milk has passed directly into the aboma-
developed pica, often as a result of chronic illness.
sum and this source of undegradable protein must be
These points suggest that ulcers may result from large,
replaced by high-quality undegradable foods, e.g.
inadequately digested particles of hay or straw, passing
fishmeal or linseed meal, after weaning. The higher
from the rumen to the abomasum. There is undoubt-
the growth rate required, the higher will be the require-
edly an increase in ulceration in calves with trichobe-
ment for additional undegradable protein. As feed con-
zoars (hair balls), but whether the hair balls lead to
version is much more efficient at a younger age (Table

ulceration, or whether chronic ulceration stimulates the
16.2), it is still cost-effective to feed expensive, nutri-
calf to lick and eat hair (in the same way as chronic
tious diets to freshly weaned calves.

ruminitis) is uncertain. Ulceration occurs in association

If the diet largely consists of poor-quality forages,
with abomasal dilation (see p. 237) and may also result
the calf's growth will be stunted, and the rumen will
from ruminal acidosis and starch overload, allowing
be distended with slowly fermenting foods. If con-
undigested starch to spill over into the abomasum.

concentrate intakes are inadequate, hungry calves may

Fungal hyphae are visible microscopically in a propor-
consume large amounts of forage. Fermentation rates
tion of ulcers and may be involved in the pathogenesis.
will decrease and rumen distension is exacerbated.

Affected calves have a 'pot-bellied' appearance, i.e. the

Signs

lower abdomen is distended, often on the right as well

The majority of ulcers in calves are subclinical although
some may haemorrhage, producing melaena and

anaemia, which is occasionally fatal. Others produce a

Table 16.2

*The effect of age on feed conversion (Blowey,
localized peritonitis of the abomasal serosa, whilst per-
1999).*

*forating ulcers lead to acute peritonitis and death. The
three-week-old Friesian calf in Plate 16.4 was recum-*

Body weight (kg)

Feed conversion ratio

*bent, in considerable pain, with a subnormal tempera-
ture (37.5°C) and sunken eyes. Regurgitated rumen*

50

2 : 1

contents are visible at the mouth. Death followed

100

3 : 1

within two hours, despite abomasal drainage, lavage

300

5.5 : 1

and parenteral metoclopramide. On post mortem

500

8.5 : 1

(Plate 16.5) two large ulcers, one perforated with a

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white diphtheritic lining, were present, typically on the
after feeding. Affected animals are seen in severe dis-
greater curvature of the abomasum.

tress, kicking at the distended abdomen or rolling on
the floor. Some cases are fatal, others respond to relax-
ant therapy, for example pethidine or mepramizole. On

Treatment

post mortem the milk has often failed to clot, but no

The treatment of abomasal ulcers is largely sympto-
other lesions are seen and the cause remains unknown.

matic. Valuable calves with extensive haemorrhage may

It has been suggested that it is more common with

be given a blood transfusion, but bleeding ulcers are
infrequent and irregular feeding times.

less common in calves than in adult cattle. Kaolin has
been used, but probably antacids such as magnesium
oxide or magnesium silicate give a better response.

Chronic peri-weaning diarrhoea

Metoclopramide at 0.5–1.0 mg/kg body weight has been used to alleviate abomasal atony (Biggs et al. , 1989).

A well-recognized syndrome of a chronic persistent

Drainage and lavage may be indicated in concurrent

grey/brown scour occurs in calves 4–10 weeks old, i.e.

abomasal dilation and ulceration. Antibiotics are

before or after weaning (Blowey, 1988, 1994, 2000;

indicated when peritonitis is suspected.

Bidewell et al., 1999) and when consuming concen-

trates. Morbidity is usually high but mortality is low and

the few detailed post mortems reported have not shown

Abomasal dilatation

consistent findings. There may be fibrous impaction of

the rumen and a distended colon. Many cases of mild

Also known as abomasal bloat, this condition of

‘looseness’ often pass unreported, but if the condition

unknown aetiology produces a shock syndrome in

progresses, appetite falls and marked weight loss

calves, typically at two to three weeks old, when they

occurs. The diarrhoea may be unusually persistent, with

first start eating solid food. Many cases are fatal.

many cases continuing for 4–6 weeks, leading to marked Affected calves are very dull, with sunken eyes and sub-weight loss and stunting. The condition can be a major normal temperature, and are often reluctant or unable problem for feed companies because it often becomes to stand. Enteritis may occur, but it is not a consistent apparent following (but not necessarily associated with) feature. The enlarged abomasum, distended by excess a delivery of food.

fluid and occasionally with gas to produce tympany, can A range of treatments have been used, but none is particularly effective and most calves seem to eventually fluid has a much higher pH (range 5–7) than a normal self-cure. Faecal examinations of affected calves reveal calf with an empty abomasum (pH 2–3), thus allowing

a range of organisms including Giardia, cryptosporidia, the bacterial count to increase to 10⁵–10⁹ ml, with coli-coccidiosis, Campylobacter species and rotavirus. Motile forms predominating. A normal calf has an abomasal protozoa are commonly seen in fresh faecal smears and pH range 2–4 and a bacterial count of 50 000/ml, with although calves can excrete giardial cysts asymptomati-

mainly staphylococci and *Bacillus* species and very rarely, *Giardia duodenalis* has been reported to cause low numbers of *E. coli*. This has led to the proposal that chronic diarrhoea in calves in the USA (O'Handley et al., 1999). Although faecal coccidial oocyst levels are 'watery mouth' in young lambs (Price, 1989). often low, the author has examined an outbreak where Abomasal drainage may be achieved by deep intestinal histopathology was typical of coccidiosis and insertion of a stomach tube. With the calf in lateral recumbency there was an apparent improvement in the remaining recumbency on a straw bale and its head hanging down, the calves following the use of in-feed decoquinate at 1.67 g/kg excess fluid is discharged under gravity. The abomasum can then be flushed two to three times with electrolyte solution 10 g/50 kg body weight of calf. It has been suggested that solutions and, if the calves are treated symptomatically the syndrome is similar to colitis in pigs, where a primary treatment for shock and acidosis and fed electrolytes for 24–48

*dietary imbalance leads to proliferation of intestinal
hours, recovery rates are acceptable. Metoclopramide
parasites. The rate at which the syndrome passes
at 0.5–1.0 mg/kg body weight has been reported useful
through several groups of calves would suggest that an
to overcome abomasal atony (Biggs et al. , 1989), but the infectious cause is also
involved (Plate 16.6).*

current author has met with very limited success.

*Management factors have been implicated, particu-
In those cases that reach post mortem, a degree of
larly those relating to digestive upsets. The syndrome is
abomasal ulceration, sometimes with fungal hyphae, is
seen more commonly (but not exclusively) with calf
often present.*

*pellets rather than coarse mix, presumably because
Acute abomasal bloat may also occur in milk
calves eat coarse mix more slowly, chew it more thor-
substitute-fed calves within 20 to 30 minutes or less
oughly and saliva production is therefore increased.*

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Feeding rancid food, digestive upsets associated with

days. There may be additional respiratory signs, with poor abomasal groove closure and use of 'milking a muco-purulent occulo-nasal discharge originating cow' concentrates and maize gluten, both of which are from ulceration of the mouth and pharynx and in some unsuitable for calves with an improperly developed cases crusting lesions of the nostrils are seen. On post rumen, have all been suggested as causes. Hygiene mortem the characteristic findings are erosive ulcers seems to be important and a thorough cleaning of the throughout the gastro-intestinal tract, with some being affected area helps. The syndrome is more common in sufficiently deep to produce areas of localized peritonitis group housed/fed calves rather than in animals penned. The ileum, caecum and colon are the organs most commonly affected. Kidneys may be pale and swollen, to two weeks postweaning will help (Blowey, 2000). with infarcts, and there may be lesions of inhalation Treatment is largely symptomatic and includes pneumonia as a consequence of the pharyngeal

antimicrobials, anticoccidials and vitamins. Macrolides, ulceration (Penny, 1999).

for example tilimicosin, have been used and sul-

Typical haematological findings reflect the pathology phonamides and other anticoccidial agents. Badly af-

and include anaemia, leucopenia, neutropenia and

affected calves should be returned to a whole milk diet,

raised urea values. Treatment is symptomatic and

without access to solid food, and possibly fed three to

includes antibiotics, NSAIDs and fluid therapy. The

four times daily. One severe outbreak dealt with by the

aetiology is unknown. Although the pathology is

author appeared to improve through the use of in-feed

strongly suggestive of BVD (p. 853), the virus is rarely

decoquinate. Lasalocid could also be used. There is an

isolated from affected calves.

urgent need for more epidemiological information on

predisposing factors.

References

Colic (see also p. 845)

Bidewell, C.A., David, G.P., Gonning, R.F., Harwood, D.G.,

Higgiov, R.J., Jones, J.R. & Laren, R.D. (1999) Persistent Colic is commonly seen in young calves. It occurs in asso-periweaning calf diarrhoea. *UK Vet*, **4**, 35–8.

ciation with many of the syndromes described earlier in

Biggs, A.M., Dainton, J.T. & Tucker, M.E. (1989) *Metoclo-*

this chapter, namely oesophageal groove dysfunction,

pramide for preventing watery mouth. Veterinary Record, acidosis, ruminal bloat, ruminal impaction, abomasal

124, 312.

ulceration and abomasal dilatation. True intestinal spas-

Blowey, R.W. (1988) *A Veterinary Book for Dairy Farmers*, modic colic occurs and may precede enteritis and subse-2nd edn. Farming Press, Ipswich, p. 54.

quent diarrhoea. Colic may also be a sign of a more

Blowey, R.W. (1994) *Calf feeding practices in relation to*

serious abdominal disorder, for example torsion of the

health. Cattle Practice, **2**, 375–82.

Blowey, R.W. (1999) *A Veterinary Book for Dairy Farmers*, root of the mesentery, intussusception, acute peritonitis,

3rd edn. Old Pond Publishing, Ipswich, pp. 21–66.

cystitis with urethral obstruction or atresia ani/coli. The

Blowey, R.W. (2000) *Chronic peri-wearing diarrhoea – the*

latter condition is seen within 24–48 hours of birth.

clinical syndrome. Cattle Practice, **8**, 89–90.

The treatment of colic therefore depends on the

Blowey, R.W. & Weaver, A.D. (1991). A Colour Atlas of

diagnosis and is discussed in the relevant section.

Diseases of Cattle. Wolfe Publications, London.

O'Handley, R.M., Cockwill, C., McAllister, T.A., Jelinski, M., Morck, D.W. & Olsen, M.E. (1999) Duration of naturally

Necrotic enteritis (see also p. 213)

acquired giardiasis and cryptosporidiosis in dairy calves and

their association with diarrhoea. Journal of the American

This syndrome, first reported in the UK in 1991, affects

Veterinary Medical Association, 214, 391–6.

spring-born suckler calves at 2–4 months old, when they

Orskov, R. (1987) The Feeding of Ruminants. Chalcombe Publications, Marlow, Bucks, pp. 1–90.

are at pasture. Morbidity is low but mortality is high

Penny, C.D. (1999) Necrotising enteritis in beef suckler calves.

(Penny, 1999). Clinical signs are a sudden onset of

UK Vet, 4, 24–8.

profuse haemorrhagic diarrhoea, which later becomes

Price, T.P. (1989) A treatment of calf enterotoxaemia. In

scant and mucoid with tenesmus. Coccidiosis is there-

Proceedings of British Cattle Veterinary Association

fore an important differential. Pyrexia is common and
1988–1989, pp. 185–93.

the animal becomes progressively more depressed,
Webster, J. (1984) Calf Husbandry Health and Welfare.
anaemic and dehydrated, with death following in 7–10
Granada Technical Books, pp. 1–202.

Chapter 17

Calf Respiratory Disease

A.H. Andrews

Introduction

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The second problem is in a management system

Enzootic and cuffing pneumonias

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involving weaned suckled calves, usually six months to

Epidemiology

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two years old and mainly reared outside. This disease

Aetiology

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occurs following transport and housing and results in a

The environment

240

condition best described as transit fever or shipping

Management

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fever (see Chapter 20). All the syndromes are best

The calf

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defined by the circumstances in which they occur

because their aetiology is complex and mainly multi-

factorial in nature. This involves the susceptibility of the

Introduction

animal, the environment in which it is kept, the man-

agement of the animal as well as the various disease

Bovine respiratory disease (BRD) in calves in the

agents to which it is subjected. In many BRD problems

developed world occurs under two main management

the causal agents will be similar in each of the syn-

systems. The first involves young, housed calves, usually dromes and it is only the management conditions that

dairy bred and either reared for beef or as dairy

will be different.

replacements. These are weaned from their dams within a few days of birth and then fed milk substitute or milk until weaned, usually between five and eight weeks of age. Under such conditions these calves can

Enzootic and cuffing pneumonias

succumb to one of two different respiratory syndromes. Often one will lead into the other. The first is a problem The aetiology of pneumonia in young calves is of slow, insidious onset known as chronic or cuffing extremely complex in both the acute and the chronic pneumonia, whereas the second is more sudden and form. It is fair to suggest that the disease is multifactorial. It is usually seen in calves reared indoors, particularly when they are reared for beef production and so or enzootic pneumonia.

have moved farms at an early age. Most cases occur Fewer problems occur with calves suckling their between two and five months and usually following

mothers in the beef herd until weaning. However, on weaning from a milk-substitute diet. The causes of the occasion similar types of acute and chronic pneumonia problem are partly infectious agents, the environment, occur while the animals are housed, Unusually, out-management and the animals themselves.

breaks occur in suckler calves whilst with their dams at grass. These outbreaks are often precipitated by a stress such as a management change or marked alteration

Epidemiology

in the weather. However some of these are less obvious

Chronic

in their precipitating factors. Investigation of these problems often shows *M. haemolytica* to be present.

It is very difficult to determine the incidence of chronic

However, *Haemophilus somnus*, infectious bovine or capping pneumonia as most affected calves are not rhinotracheitis (IBR), bovine respiratory syncytial virus treated and they do not die. In addition, by the time (RSV) and parainfluenza III virus (PI3) have all been animals reach slaughter weight or are culled the resid-

detected in various outbreaks. It must also be remembered that calves can pick up lungworm infection and bred animals reared on a farm it was found that 11 per cent had significant pneumonic lesions and it was estimated that this resulted in a 7.2 per cent reduction in liveweight gain (Thomas et al., 1978).

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Acute

involved. Thus the three main mycoplasmal agents are M. bovis, M. dispar and Ureaplasma spp., although M.

Very few studies have been undertaken into the inci-

canis is increasing. In the case of viruses, those most common of this condition. However, Thomas (1978)

commonly implicated are bovine respiratory syncytial

obtained health records for 12 beef farms in 11 British virus (BRSV), parainfluenza III (PI

counties. The records covered 11 050 animals between

3) virus, infectious

bovine rhinotracheitis (IBR) and bovine viral diarrhoea

1970 and 1977. Mortality of bought-in calves is variable (BVDV) virus.

according to the rearing system used. However, a figure

The bacterial pathogens involved form a very long

of 5.5 per cent is usual and it can be suggested that

list and one or more tends to be isolated from most cases

about half these deaths are due to enzootic pneumonia.

of disease. It is often postulated that these may be sec-

This was seen in the Thomas study, which showed a

ondary invaders after primary damage by mycoplasma

death rate of 2.7 per cent from pneumonia out of a total

or viruses. The most commonly isolated organisms are

mortality of 5.9 per cent. The overall number of animals

Mannheimia haemolytica, *Pasteurella multocida* and treated for pneumonia was 32.6 per cent although the

Haemophilus spp., especially *Haemophilus somnus*. In levels on individual farms varied from 3.1 to 52 per cent.

addition, cases that involve toxæmia often include

Recent routine studies of individual outbreaks pro-

Arcanobacterium (formerly *Actinomyces*, *Corynebacterium*) *pyogenes* and *Fusobacterium necrophorum*. *M.* (range 41.7–90.5 per cent) (Andrews, 2000). It should be remembered that some calves require more than one treatment for pneumonia. Again, there are common. The organism has also been involved in pleuropneumonia problems. The involvement of bacteria in this complex condition of enzootic pneumonia is also common. The recorded level was 8.9 per cent being treated enforced by the fact that in most naturally occurring outbreaks of disease there is some clinical response following the use of antibiotics. (Thomas, 1978) in a group of 2040 animals of which 22 per cent became ill initially. Another problem is that besides mortality, calves may become chronically

affected and fail to thrive. Culling probably doubles the

The environment

mortality due to disease and a level of 3.6 per cent was

Various factors in the environment tend to be consid-

found in the study by Thomas. The reduction in

ered to be important. There is a popular belief that the

liveweight gain in animals treated for pneumonia but

disease is often associated with low environmental tem-

which were not culled or died, compared with those

peratures and a high humidity (see p. 974). Often a res-

untreated, was found to be 2.6 per cent (Thomas et al., piratory problem is associated with a sudden drop in

1978). A study of eight outbreaks on dairy herds in

temperature 24 to 72 hours previously. It is thought that

1997–99 showed an average cost per ill animal of £43

the cold may allow infection to flare up partly by affect-

and a cost per animal within the group of £30. Veteri-

ing the respiratory defence mechanisms and it also

nary and medicine costs amounted to only 40 per cent

allows disease spread by encouraging calves to huddle

of the total costs of the outbreak (Andrews, 2000).

together. It appears that the alveolar macrophages, ciliated and mucus-secreting cells are susceptible to the

Aetiology

environment and cold stress inhibits clearance from the

A large number of different infectious agents have been

lungs. In the cold, animals reduce heat loss by slowing

isolated and suggested to be involved in the aetiology

down their respiratory rate and by a partial reduction

of both chronic and acute calf pneumonia. Often the

in the pulmonary ventilation rate (Webster, 1981b).

enzootic form is called 'viral pneumonia'. However, it

This causes a reduced pulmonary oxygen tension and it

is generally considered this term is a misnomer as it pre-

has been shown that hypoxia reduces the clearance rate

supposes the aetiology and in most cases, even when

of some organisms in mice (Green & Kass, 1965), and

viral agents are present, they only form part of the

there is a reduction in mucociliary rate and alveolar

disease complex. It is usually considered that the most

phagocytic activity (Thomson & Gilka, 1974). The prob-

important agents involved in the chronic or cuffing

able role of hypoxia in respiratory infection is indicated
form are mycoplasmal. They include *Mycoplasma*
by the fact that most lung consolidation occurs in the
dispar and *Ureaplasma spp.* When it comes to the acute ventral parts of the
apical and cardiac lobes and these
form there is a very long list of pathogens. Many have
tend to be the areas of lowest oxygen tension and the
been seen in the disease but may not necessarily have
slowest rate of clearance of pathogens (Veit et al., 1978).
produced the problem experimentally (see Table 17.1).
At times, outbreaks of disease occur with a high tem-
However, certain agents do seem to be more commonly
perature and a low humidity. Thus rearing at a temper-

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Table 17.1

Infectious causes of enzootic pneumonia. The cause is often multifactorial, probably often mycoplasmal infection followed by viral and bacterial causes.

Viruses

Mycoplasmas

Bacteria

Respiratory syncytial virus (BRSV)^a

Mycoplasma bovirhinis

Mannheimia (Pasteurella) haemolytica a

Parainfluenza virus II

M. dispar a

P. multocida a

Parainfluenza virus III (PI3)a

M. mycoides subsp. mycoides

Arcanobacterium (Actinomyces, Corynebacterium)

Reovirus types 1,2,3 (Reo)

(little importance in Europe)

pyogenes a

Bovine viral diarrhoea virus (BVDV)a

M. alkalescens

Streptococcus pneumoniae

Adenovirus types 1,2,3,4

M. arginini

Staphylococcus aureus

Enterovirus

M. bovis a

Strep. bovis

Rhinovirus type 1 (RV)

M. canis

Staph. epidermidis

Infectious bovine rhinotracheitis

M. bovigentialium

Strep. mitis

(IBR)^a

Acholeplasma laidlawii

Strep. faecalis

Respiratory bovine coronavirus

A. modicum

Aerococcus viridans

A. axanthum

Acinetobacter spp.

Ureaplasma spp.^a

Micrococcus luteus

Ureaplasma diversum

Staphylococcus spp.

Leach's group 7 mycoplasmas

Neisseria spp.

Chlamydiales spp.

Actinobacillus lignieresii

Klebsiella spp.

Corynebacterium bovis

C. xerosis

Streptococcus spp.

Aerococcus spp.

Haemophilus spp.

Haemophilus somnus a

Aeromonas spp.

Bacillus spp.

Alcaligenes faecalis

Micrococcus roseus

Micrococcus spp.

Escherichia coli

Fusobacterium necrophorum a

a Thought to be the most important causes.

ature of 21°C (70°F) and 47 per cent relative humidity

October and December and the second from February

(RH) predisposed Friesian and Jersey calves to respi-

to May (Thomas, 1978). A study of a veal-calf unit over

ratory disease compared with those kept at a tempera-

a 14-month period showed extensive outbreaks of

ture of 14°C (60°F) and 36 per cent RH (Roy et al.,

disease in October, April and early June (Miller et al., 1971). It does seem that a high relative humidity may

1980).

be beneficial at a high environmental temperature as it

The relationship between season and respiratory

increases the sedimentation rate of airborne particles

disease is probably partly due to management influ-

and thereby reduces the bacterial count of the envi-

ences, particularly the housing of animals in close prox-

imity. These factors have formed the basis for the

establishment of the sweat box system for pigs

infection. However, the correlation between weather

(Gordon, 1963) and this is why water drips are often

and disease is harder to prove, and in a disease outbreak used in buildings for veal production.

involving veal calves no relationship was found (Miller

Although it is generally recognized that respiratory

et al., 1980). A sudden drop in environmental tem-

disease commonly occurs at certain times of the year,

perature is often followed one to three days later by an

and under certain weather conditions, it is only recently outbreak of respiratory disease in housed, dairy-bred that attempts have been made to determine why. In calves or housed single-suckler calves (Wiseman, 1978). housed dairy-bred calves on one farm two peak levels Pneumonia in Irish calves housed in naturally ventilated buildings also tended to be precipitated by change

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in weather, such as frost, rain and wind or damp, humid authors (Wiseman, 1978) that it is the collection of a conditions (Bryson et al., 1978).

large number of young calves of a similar age and Body temperature regulation in cattle is well developed, so they can tolerate both heat and cold (Webster, 1981a). However, draughts in buildings can result in reared calf systems. It is not always accepted that the chilling, which increases the metabolic rate and perhaps number of calves in a single air space is more important

reduces resistance to infection (Webster, 1981b), but than the air space or floor area per calf. However, over-high relative humidity on its own does not chill cattle crowding is a major factor in allowing disease development and spread. Webster (1979). Webster (1981b) therefore postulated that the main effect of weather was compounded by the disease levels are the inadequate cleaning and disinfecting building itself. Thus a badly constructed building with poor ventilation would reduce ventilation rate, and the allowing the accommodation sufficient rest between water-carrying capacity of the air would be detrimental calf intakes. In some cases younger animals are mixed with an older group, thereby allowing transmission of drainage. Such conditions could lead to the relative micro-organisms from the old, apparently clinically humidity remaining at more than 90 per cent for several normal animals. Economic considerations may also days, and this in turn would increase the survival and

influence the picture if there is a reluctance to employ spread of pathogenic organisms. Some support for this preventive measures such as vaccination, etc. Several view has been obtained by work showing an increased management procedures such as castration, disbudding survival of small airborne bacteria at high relative and weaning all appear to have an effect on the level of humidities (Jones & Webster, 1981). There is thus an disease. The importance of microbial agents is indirectly increase in the number of infective particles that are not indicated by the importance of adequate uptake of removed in the nasal passages and these are deposited colostrum (Phillips, 1975; Thomas, 1979).

further down the respiratory tract, possibly causing disease.

The calf

There appear to be some breed differences in suscepti-

Management

bility to respiratory disease and Friesian and Jersey

The time of weaning appears to be important in that calves are more likely to be infected than Ayrshire or

more lung lesions are found in calves weaned at five

Hereford × Friesians (Roy, 1980). It has been postulated

weeks than those on an ad libitum milk substitute diet that this may be due to an increased skin thickness in

until 14 weeks old (Roy et al., 1971). The reason for this some breeds, which provides better insulation.

is not clear but it might be due to the lower levels of

energy intake, or lower availability of a micronutrient

Signs

(Roy et al., 1971); or perhaps inhaled dust or fungal

spores, which are more prevalent when dry feed is

Chronic: The condition is one of gradual onset. There

eaten, exacerbate the problem (Lacey, 1968); or due to

is generally no illness and so the calf is bright, eats well,

weaning being less stressful or the calf more resistant

but it may have a slight mucoid or mucopurulent oculo-

to respiratory disease at an older age.

nasal discharge. The temperature is normal or slightly

There is a relationship between colostral antibody

raised at 38.5–39.5°C (101–103°F); the respiratory rate

levels and respiratory disease (Thomas & Swann, 1973).

may be at any level from normal to 100 per minute with

This apparent resistance to disease might be due to the a normal pulse. There is a dry, explosive cough that is ingestion of increased levels of specific antibody or due usually produced singly. On chest auscultation there are to an indirect effect resulting from a reduced amount noises of whistling, wheezing or squeaking and these of enteric disease, or possibly the gradual reduction in are more commonly heard at expiration, although often passive immunity allowing a more orderly exposure of they occur at both inspiration and expiration. The the calf to respiratory pathogens. When colostral anti-sounds are most common in the anterior and ventral bodies are low there is a relatively fast loss of antibody, parts of the chest.

which may then allow the invasion of many pathogens (Williams et al., 1975).

Acute: Although one calf may be seen to be ill to begin The purchase of a large number of calves from dif-with, several animals will usually become sick within the

ferent sources for dairy and beef production and next 24–48 hours. There is normally a reduction in feed keeping them in the same housing allows the easy

intake of the group and widespread coughing will be spread of potential respiratory pathogens to other apparent. The affected animals appear dull and the animals in close proximity. It is considered by many head tends to be carried lower than normal. There is

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inappetance, pyrexia (40–40°C, 104–107°F) and a dull, to bronchiolar necrosis; there are basophilic and sweaty coat. Other signs including a mucoid or mucop-eosinophilic intranuclear inclusion bodies in epithelial urulent oculo-nasal discharge, tachypnoea (respirations and other cells. Intranuclear inclusion bodies, particu-are usually over 40 per minute), dyspnoea and hyperp-larly in the epithelial cells of the trachea and bronchi, noea are normally present. In all but very severely ill are seen in IBR.

calves there tends to be an increase in the amount of Type 2. There is often marked consolidation of the coughing; the cough itself may be of a harsh, dry, cranial lobes with red or red/grey hepatization with hacking type, but in others it will be moist. Pinching the

widespread tissue necrosis and in many cases sup-
upper trachea often elicits a cough. On auscultation of
puration and this may involve up to 70–80 per cent of
the thorax there are usually loud, harsh sounds or
the lung. This type of lesion is often characteristic of
whistling, wheezing or squeaking. These sounds may be
bacterial infection.

Extensive consolidation and

present at inspiration or expiration, but more com-

*suppuration are particularly seen in A. pyogenes and monly they are heard at the
latter and in some cases*

F. necrophorum infections.

there are fluid sounds such as bubbling or gurgling,

which will be audible in the cranio-ventral parts of the

Type 3. This is characteristic of calves that suddenly

lungs. In some bacterial infections, where there is

develop respiratory distress. The syndrome is often

marked lung consolidation, few sounds are present.

called atypical interstitial pneumonia. At post-mortem

examination there is interstitial emphysema, pulmonary

Necropsy

oedema and congestion, with alveolar epithelial hyperplasia and hyaline membrane formation.

Chronic: Lesions tend to be confined to the ventral
Besides the lungs, there is usually gross enlargement
parts of the lung lobes and involve, in decreasing sever-
and congestion of the mediastinal and bronchial lymph
ity, the apical, cardiac and cranial parts of the caudal
nodes. In some cases there is fibrinous pleurisy and the
lobes. The area involved may be 5–40 per cent of the
heart may be enlarged with epicardial and endocardial
lung tissue and it tends to be red or purple in colour
haemorrhages. Sero-sanguinous fluid may be present in
and to be indurated. Histologically, there are accu-
the thorax and pericardial sac (Thomas, 1979).

mulations of lymphocytes in the peribronchiolar tissue
Lesions of pleuropneumonia result in a thickened
and it is this that produces a cuff; macroscopically it is
pleura with the lung showing thickened interlobular
seen as a mottling of the lesion's cut surface. When
septa with variable amounts of lung consolidation
accumulation is great, then the lymphocytes can cause

showing dark red to grey hepatization.

a narrowing of the bronchiolar lumen and cause the surrounding alveoli to be compressed. Resolution of the lesions occurs over several months provided that they

Diagnosis

are uncomplicated. The bronchial and mediastinal

Chronic: Several animals are affected and are

lymph nodes are usually enlarged and there is often a usually indoors. The problem is gradual in onset and fibrinous pneumonia.

although the animals show respiratory signs they are bright and eat well. The respiratory signs include single,

Acute: Within the clinical entity of acute pneumonia dry coughing.

there are three types of pathological entity that can be recognized involving the lungs (Pirie, 1979).

Acute: Again this affects a group of calves. Respiratory Type 1. There is localized consolidation particularly

signs are present and the animals are usually obviously of the cranial lobes and the tissue is dark red, friable ill. Nasopharyngeal swabs or broncho-alveolar lavage

and there is no gross evidence of necrosis. Interstitial can be undertaken to examine for bacterial, viral or emphysema may be present. Histologically, there is an mycoplasmal presence. The last two groups require absence of peribronchiolar cuffing, but there is necrosis placement in a transport medium. Fluorescent antibody of the bronchiolar epithelium. The changes are often tests are available for most of the more important viral suggestive of a viral infection, but in many cases the causes. Even when a potential pathogen is recovered it presence of a virus cannot be proven because of the does not always mean that it is the same agent as is absence of inclusion bodies or positive immunofluores-causing trouble in the lungs. Paired serum samples can cence. PI3 infection can be suspected when there is be taken two weeks apart. This may indicate the cause alveolar and bronchiolar epithelium proliferation and but is only of value if control measures can then be eosinophilic intracytoplasmic inclusion bodies are implemented for the future. Post-mortem material can present. RSV infection can result in alveolar epithelium

be checked for the presence of organisms and for hyperplasia and large multinucleate syncytial cells. histology to indicate the type of pathogen which might Bronchial collapse occurs with adenovirus infection due be involved.

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Differential diagnosis

tion, so that it is often a good policy to persuade the farmer to treat all the cattle in the group, ideally using Chronic: This needs to be differentiated form acute a different antibiotic. As with most disease, the earlier pneumonia but in the latter case the animal is ill. In-treatment is started, the less the mortality and the fewer halation pneumonia is likely only to involve a single the animals that have to be culled because of chronic animal, which again is ill, and there is usually a related disease. The drugs commonly used are given in Table history. Tuberculosis may cause problems but usually 17.2. The therapeutic agent administered should have a there is a herd history and the dam may be showing broad spectrum of activity and ideally it should be bac-

signs. Muscular dystrophy (see p. 258) can produce respiratory signs. The choice will most probably have to be based on previous successful usage on that farm or elsewhere and raised serum creatine kinase, and aspartate aminotransferase levels. Congenital heart defects because if there is no response to treatment it will make (see pp. 173–4) will usually only involve one animal and the initiation of subsequent therapy very difficult. If an animal dies then culture of swabs from the lung, *Salmonellosis* can result in signs but the calf is usually not really ill with diarrhoea (see p. 226). Subsequent antibiotic sensitivity of the isolates may be of use. The ideal is to culture swabs from material in carcasses. Acute. Chronic pneumonia is a major differential diagnosis but the animals are not really ill in such cases. Ryngeal swabs from live infected calves is of less value

Uncomplicated IBR infection (see p. 289) could cause as it cannot be certain that the organism cultured will difficulties but there are mainly upper respiratory signs be the same as the one causing disease in the lungs. and a noticeable conjunctivitis. Salmonellosis (Chapter However, it does indicate possible pathogens.

15) will usually present with enteric signs. Although Therapy should be continued for three to five days, mucosal disease (see p. 853) can give rise to respiratory depending on the drug used and the response to treatment signs, there is usually also diarrhoea and, in some ment. In acute cases the choice of antimicrobial agent cases, mouth ulceration. Inhalation pneumonia mainly is normally dictated by those that can be given intravenously. Intratracheal administration of antibiotics has history. Tuberculosis will be detected by a history of been advocated by some practitioners and in such cases disease in the herd. Congenital heart defects (see pp. those in aqueous solution are preferable to those in 173–4) involve a single animal and there is usually a

organic solvent. No antibiotic is at present licensed in heart murmur. Calf diphtheria (see p. 251) results in Britain for this route of administration. Those commensal and is again a single animal problem. Malignant pneumonia most commonly used have included erythrocatarrhal fever (see p. 935) affects single animals and mycin, trimethoprim and sulphadoxine.

there is lymph node enlargement, corneal opacity and often nervous signs present. Acidosis (p. 829) can mimic (2)

Corticosteroids. Some of the compounds available for pneumonia.

are shown in Table 17.3. The drugs commonly in use today include betamethasone, dexamethasone,

Treatment

prednisolone, flumethasone and triamcinolone. These

Chronic: Therapy is usually not necessary unless the calf compounds provide symptomatic relief only and do not

is showing severe coughing. Several antibiotics are of cure the condition. Many people consider them to be use, including tylosin at 4–10 mg/kg (2–5 mg/lb) body

overused (Pirie, 1979), but often an animal will show weight, oxytetracycline at 10 mg/kg (5 mg/lb) body little or no improvement on antibiotic therapy alone, weight, spiramycin at a dose of 20 mg/kg (10 mg/lb) only to recover rapidly once corticosteroids are added body weight of spectinomycin at 20–30 mg/kg (10– to the treatment regimen. Their action is to suppress all 15 mg/lb) body weight. The macrolide antibiotics stages of inflammation regardless of whether the cause (tilosin, erythromycin, spiramycin) are all concentrated is physical, chemical or immunological in origin. Thus in the lungs and have good efficacy against mycoplasmal in the acute stage, corticosteroids reduce vasodilation, infections. The fluorquinolones have activity against oedema formation and leucocyte infiltration (Eyre, mycoplasma but probably cannot be recommended for 1978). The drugs also have a ‘euphoric’ effect on the this purpose due to human health implications. dull animal and this may allow the calf to eat and otherwise speed its recovery (Pirie, 1979).

Acute:

The main problem with corticosteroids is the unselective suppression of inflammation that therefore affects animals at one time and need treatment it includes those parts of the inflammatory and immune response, such as macrophage infiltration, which are

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Table 17.2

Some of the antimicrobial compounds used in calf pneumonia therapy.

Antimicrobial

Bactericidal (C) or

Route of

Dosage

compounds

bacteriostatic (S)

administration

(mg/kg)

(mg/lb)

Amoxycillin

C

i.v., s.c., i.m., oral

7 (15 LA)

3.5 (7 LA)

Amoxycillin and

C

i.m., s.c.

8.75 (am)

4.38 (am)

clavulanic acid

1.75 (cl)

0.88 (cl)

Ampicillin

C

i.v., s.c., i.m., oral

2–7

1–3.75 (7.5 LA)

Baquiloprim and

C

oral

4–8 (baquil)

2–4 (baquil)

sulphamidine

36–72 (sulpha)

18–36 (sulpha)

Ceftiofur

C

i.m.

1

0.5

Chloramphenicol (some countries)

S

i.v., s.c., i.m., oral

4–10

2–5

Danofloxacin

C

i.m.

1.25

0.6

Enrofloxacin

C

s.c.

2.5 or 7.5

1.25 or 3.75

Erythromycin

S

i.m.

2.5–5

1.25–2.5

Florfenicol

S

s.c., i.m.

40 (*s.c.*), 20 (*i.m.*)

20 (*s.c.*), 10 (*i.m.*)

Marbofloxacin

C

i.v., s.c., i.m.

2

1

Oxytetracycline

S

i.v., s.c., i.m., oral

10 (20 LA)

5 (10 LA)

Penicillin plus

10–15 (pen)

5–7.5 (pen)

streptomycin

C

s.c., i.m.

10–20 (strep)

5–10 (strep)

Spectinomycin

C

i.m.

12.5–30

6.25–15

Spiramycin

S

i.m., oral

20

10

Sulphadimidine

S

i.v., s.c., oral

initial

200

100

maintenance

100

50

Sulphamethoxypyridazine

S

s.c., i.m.

22

11

Sulphapyrazole

S

i.v., s.c., i.m.

30–100

15–50

Tilmicosin

S

s.c.

10

5

Trimethoprim and sulphadiazine

C

i.m., oral

15–22.5 (active)

7.5–12 (active)

Trimethoprim and

sulphadoxine

C

i.m.

15 (active)

7.5 (active)

Tylosin

S

i.m., oral

4–10

2–5

LA, long-acting; am, amoxycillin; cl, clavulanic acid; baquil, baquiloprim; sulpha, sulphadimidine; pen, penicillin; strep, streptomycin; active, active ingredients.

Table 17.3

Corticosteroids used in respiratory disease in

immune response can also increase the ability of the calves.

same organisms to reinfect the animal following recovery. It is therefore essential that antibiotic therapy

Drug

Dose per animal (mg)

is administered concurrently with corticosteroid therapy and in most circumstances it is probable that a bacteri-

Betamethasone

2–10

cidal drug is indicated rather than a bacteriostatic drug.

Cortisone

up to 500

Adequate therapeutic doses of the antibiotic should be

Dexamethasone

2–5

given and maintained for sufficient time. Another

Flumethasone

0.5

problem with the prolonged use of corticosteroids is

Hydrocortisone

up to 300

that there is a risk of inducing permanent adrenal insuf-

Prednisolone

up to 20

Triamcinolone

up to 5

iciency, but this has produced little worry in cattle.

(3)

Non-steroidal anti-inflammatory drugs

(NSAIDs) (see Chapter 62). These drugs are salicylate-

concerned with the control and removal of infectious

like compounds and although many are not registered

organisms. In such an environment an organism can

for use in cattle, they do help reduce inflammatory

multiply and spread within the animal. This may be the

reactions by blocking the synthesis of prostaglandins

agent that caused the pneumonia or an opportunist

and inhibiting kinin formation (see p. 1050). They

organism that enters the body. The reduction in the

also antagonize the actions of some of the chemical

Table 17.4

Some non-steroidal anti-inflammatory drugs.

Route of administration

Dose

Acetyl salicylic acid

Oral

1.0–4.0 g/animal

Carprofen

Injection (i.v., s.c.)

1.4 mg/kg (0.7 mg/lb)

Flunixin meglumine

Injection (i.v.)

2.2 mg/kg (1.0 mg/lb)

Ketoprofen

Injection (i.v., i.m.)

3 mg/kg (1.5 mg/lb)

Meclofenamic acid

Oral

2.2 mg/kg (1.1 mg/lb)

Meloxicam

Injection (i.v., s.c.)

500 mg/kg (250 mg/lb)

Naproxen

Oral

10 mg/kg (5 mg/lb)

Phenylbutazone

Oral/injection

4.4 mg/kg (2.2 mg/lb)

Sodium meclophenamate

a Doses are only guidelines. Note that many of the drugs are not registered for use in cattle in some countries.

mediators in the lungs such as 5-hydroxytryptamine

others are available and are given. Their actions are re-

(5-HT) and histamine, which are mainly released

latively similar to those of adrenaline. There is stimula-

tion of the central nervous system where caffeine is the

reactions (Pirie, 1979). Besides an anti-inflammatory

most powerful, myocardial stimulation where amino-

action, NSAIDs have two properties that corticos-

phylline and diprophylline are strongest, bronchodila-

teroids do not possess, namely they are anti-pyretic

tion and diuresis. Their main uses in respiratory disease and analgesic. Until the availability of flunixin these are bronchodilation and, to a secondary extent, fluid compounds were little used in Britain although they removal. The mild action of these drugs compared with are frequently prescribed in Holland where corticosteroids are not allowed to be used for respiratory relatively safe.

diseases in calves. Some of the main drugs available are (7)

Expectorants. One drug used at present as a shown in Table 17.4. The drugs most commonly used in spasmolytic is bromhexine hydrochloride, which can be Britain are carprofen, flunixin meglumine, ketoprofen given orally or by intramuscular injection at a dose of and meloxicam.

about 0.5 mg/kg body weight for five to seven days. Its (4)

Antihistamines (see Chapter 62). These drugs action is mainly to reduce the viscosity of mucus and

have been used in the past but most clinicians have found thereby help in its expulsion, resulting in improved res- them to be of little use in calf pneumonia. This is proba- piratory function. Other expectorants have often been bly because the main chemical mediator of cattle is not used in chronic cases of coughing. These include a histamine but 5-HT. The histamine that is released mixture of strychnine hydroxide, arsenic trioxide and occurs very quickly following the antibody–antigen ferric ammonium citrate given at a dose of about 5 ml reaction so that antihistamines can only be of use in the orally twice daily, or diphenhydramine hydrochloride, early stages of the inflammatory response (Pirie, 1979). ammonium chloride, sodium citrate and menthol at 5–10 ml orally two or three times daily. There is limited (5)

Sympathomimetics.

These drugs are all benefit from the antihistaminic action of diphenhy- adrenaline-like and their actions to a varying degree are dramine hydrochloride in cases of calf pneumonia.

similar to it.

(8)

Antisera. Several antisera are available in some

Although both adrenaline and isoprenaline are of

countries that have been produced either in cattle or

some use in relieving respiratory signs, they are little

*horses against *P. multocida*, the septicaemic and pneu-used because of their stimulatory effects on the heart.*

*monic strains of *M. haemolytica* and, in some cases,*

This is because adrenaline acts on many types of recep-

diplococci. Little experimental work is available to

tors that are designated a and b, those of the heart and

demonstrate their efficacy or lack of it and so their use

lung being b1 and b2 receptors, respectively. Recent

is speculative (Thomas, 1979). As has already been indi-

work has produced compounds that will act on only one

cated there are numerous organisms involved in the

type of receptor and not on the others. For various

aetiology of calf pneumonia and it would be only right

reasons including some of their other potential proper-

to expect benefit in some cases where the organisms in

ties their use has been curtailed in many countries.

the anti-sera are a major factor in the disease process.

(6)

Xanthine derivatives. Those commonly in use

(9)

Supportive therapy. During the disease phase

are etamiphylline camsylate and diprophylline, but

many animals become partially or completely anorexic

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and it has been suggested that multivitamin injections,

year, the time when disease starts should be noted and

particularly of the vitamin B group, may be of use in

checked to see if it can be associated with any changes

overcoming any temporary deficiencies that might

in management, etc. Calves bought as batches should

occur as the result of the low storage of vitamins

be kept together and not mixed with other batches. An

(Thomas, 1979). As vitamin A is of use in epithelial

all-in all-out policy for rearing calves is best advocated.

repair it may be advantageous to inject this compound

Ideally, calves should be reared before and after

to ensure speedy respiratory mucosal regeneration.

weaning in the same building for at least a month and preferably longer.

(10)

Nursing. As with many other diseases,

There are various vaccines available and, as might be although nursing is important, it is often neglected.

expected with a disease of such complex aetiology, the

Affected animals should be removed from the in-

results experienced by different farmers and veterinary

contact group partly to reduce spread of infection and

surgeons are extremely variable (see p. 1007). In some

also to allow access to feed and water away from com-

cases this may be due to the type of vaccine used. It is

petition. Feed supplied should be highly palatable and

thought that the age incidence of enzootic pneumonia

non-dusty to encourage uptake. The environment of the

may well coincide with the decline in colostral immu-

convalescent calf should include plenty of bedding, and

nity. Thus peak onset of pneumonia in housed calves is

draughts must be avoided. The provision of oxygen by

often at two to four months old when concentrations of means of a mask and reducing valve has been used in serum IgG

animals at indoor agricultural and fatstock shows.

1, IgG2 and IgA are at their lowest (Corbeil et al., 1984). It must also be remembered that several of (11)

Vaccination. It is possible to obtain rapid con-

the pathogens involved in the disease complex are

firmation as to the aetiology of outbreaks of calf pneu-

immunosuppressive. These organisms include *M.*

monia. If the cause is viral it is possible to use live

dispar, ureaplasmas and *M. bovis* as well as BRSV. In vaccines and to vaccinate in the face of an outbreak.

consequence, vaccination of calves where the organisms

This can be very effective provided it is undertaken in

are already present in the animals is likely to reduce the

the early stages of disease among the group. It appears

immune response.

to produce non-specific interferon followed by good

Dead vaccines are used to provide immunity against

specific immunity to the virus vaccinated against. To be

P. multocida and septicaemic and pneumonic strains of effective a live vaccine needs to be used and it must be

M. haemolytica. Following successful experiments administered intranasally.

(Gilmour et al., 1979) several *M. haemolytica* vaccines for serotypes 1 and 6 are now available in Britain.

Killed polyvalent vaccines are used in some countries,

Prevention (see pp. 1007–11)

and contain antigens such as RSV, PI3, IBR and BVDV.

As the disease is multifactorial, preventive measures

They are usually administered parenterally and usually

include attention to management as well as possible

require two injections to produce immunity. Recent

vaccination. Thus ensuring there are no more than 30

developments have included combined live and dead

calves in any one air space as well as making sure that

viral components of the four main antigens which are

different age groups are not mixed is helpful. If disease

injected intramuscularly. When live vaccines are used,

tends to follow particular patterns it is best to alter the

the integrity of the vaccine must ensure absence of

time of undertaking stressful procedures such as

other potential contaminants, both viral and other

weaning, castration and disbudding. Often ensuring cas-
pathogens. Subsequently, modified live intranasal vac-
tration and disbudding are undertaken more than two
cines have been available. A study of the live IBR
weeks before weaning can be helpful. Increasing the
vaccine in the absence of disease showed virus could be
duration of feeding milk substitute can also be useful,
isolated from most animals (10/11) and there was sero-
as well as ensuring weaning is not undertaken at a time
conversion in 7/11 (Lucas et al., 1982). Although good when this is likely to
result in pneumonia. Gradual
immunity was conferred to animals by modified live
weaning is often helpful, particularly when cattle are
vaccination, it was shown that some cattle became car-
fed in groups on a milk dispensing machine that will
riers after exposure to field strains of IBR (Nettleton &
provide feed throughout the 24 hours.
Sharp, 1980).

When calves are home-reared, it should be ensured
A dead vaccine has been produced against *M. bovis*,
that they receive adequate amounts of colostrum. All

but it has had only limited success experimentally. calves should be given adequate good quality feed. If There are several live vaccines used in Europe including ones against respiratory syncytial virus and parainfluenza III virus. A vaccine for PI3 has been licensed and rejected. Calves should not receive dusty or overmilled feed. As disease occurs on the same farms year after year in Britain and elsewhere.

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From experience, the vaccines themselves do appear to have variable results on individual farms. This is probably due to the type of vaccine used, i.e. live or dead, Lacey, J.C. (1968) The microflora of fodders associated with bovine respiratory disease. Journal of General Microbiology, 51, 173–7.

other underlying pathogens, the level of infective dose Lucas, M.H., Roberts, D.H., Sands, J.J. & Westcott, D.V.E. and whether or not the particular pathogens present in

(1982) *The use of infectious bovine rhinotracheitis vaccine in a commercial veal unit: antibody response and spread of the vaccine are responsible for disease on that farm.* virus. *British Veterinary Journal*, **138**, 23–8.

Before embarking on a vaccination programme it is
Miller, W.M., Harkness, J.W., Richards, M.S. & Pritchard, D.G.
important to ensure that the pathogens on the farm are

(1980) *Epidemiological studies of calf respiratory disease in*
correctly identified so that the programme can be intro-
a large commercial veal unit. Research in Veterinary Science, duced correctly.
Such regimens will usually work well
28, 267–74.

on farms housing just home produced cattle. However,
Nettleton, P.F. & Sharp, J.M. (1980) *Infectious bovine rhino-there is a particular*
problem for farmers who continu-
tracheitis virus excretion after vaccination. Veterinary
ally buy in batches of calves as the pathogens intro-
Record, **107**, 379.

duced with each group are likely to be different. Thus
Phillips, J.I.H. (1975) *Bovine respiratory disease. In Veterinary it is very difficult*
to create a suitable vaccination policy
Annual, 15th issue (ed. by C.S.G. Grunsell & F.W.G. Hill), pp. 13–15. Wright,
Bristol.

to prevent pneumonia, unless several vaccines are used

Pirie, H.M. (1979) Respiratory Diseases of Animals, pp. 68–70.

to cover most of the major pathogens. Such a policy is

Notes for a Postgraduate Course, Glasgow Veterinary

obviously costly and at times very wasteful as it will

School.

result in immunization for diseases that are not present.

Roy, J.H.B. (1980) The Calf, 4th edn. Butterworth, London.

This has led to the suggestion that in such units it is best

*Roy, J.H.B., Stobo, J.F., Gaston, H.J.G., Anderton, P., Shotton, to make a
diagnosis as to the likely cause of enzootic*

*J.M. & Ostler, D.C. (1971) The effect of environmental tem-pneumonia in the
first animal(s) to be affected. If the*

perature on the performance and health of the preruminant

agent is primarily viral then the calves can be vacci-

and ruminant calf. British Journal of Nutrition, 26, 363–81.

nated in the face of an outbreak.

Thomas, L.H. (1978) Disease incidence and epidemics – the

situation in the UK. In Respiratory Diseases of Cattle.

Current Topics in Veterinary Medicine, Volume 3 (ed. by W.B.

Martin), pp. 57–65. Martinus Nijhoff, The Hague.

References

Thomas, L.H. (1979) *Respiratory Disease in Housed Calves*, pp. 1–22. Booklet 2181. MAFF Publications, Middlesex.

Andrews, A.H. (2000) *Calf pneumonia costs! Cattle Practice*, Thomas, L.H. & Swann, R.C. (1973) *Influence of colostrum on* **8**, 109–14.

the incidence of calf pneumonia. Veterinary Record, **92**, Bryson, D.G., McFerran, J.B., Ball, H.J. & Neill, S.D. (1978) 454–5.

Observations on outbreaks of respiratory disease in housed

Thomas, L.H., Wood, R.D.P. & Longland, J.M. (1978) *The*

calves. I: Epidemiology, clinical and microbiological find-

influence of disease on the performance of beef cattle.

ings. Veterinary Record, **103**, 485–9.

British Veterinary Journal, **134**, 152–61.

Corbeil, L.B., Watt, B., Corbeil, R.R., Betzen, T.G., Brownson, Thomson, R.G. & Gilka, F. (1974) *A brief review of pulmonary R.K. & Morrill, J.L. (1984) Immunoglobulin concentrations*

clearance of bacterial aerosols emphasizing aspects of

in serum and nasal secretions of calves at the onset of

particular relevance to veterinary medicine. Canadian

pneumonia. American Journal of Veterinary Research, **45**, *Veterinary Journal*, **15**, 99–107.

773–8.

Veit, H.P., Farrell, R.T. & Troutt, H.F. (1978) *Pulmonary clear-Eyre, P. (1978) Pharmacological considerations of current*

ance of Serratia marcescens in calves . American Journal of methods of therapy. In Respiratory Diseases in Cattle.

Veterinary Research, **39**, 1646–50.

Current Topics in Veterinary Medicine Volume 3 (ed. by W.B.

Webster, A.J.F. (1979) *Housing and husbandry of the veal calf.*

Martin), pp. 409–16. Martinus Nijhoff, The Hague.

In Veterinary Annual, 19th issue (ed. by C.S.G. Grunsell & Gilmour, N.J.L., Martin, W.B., Sharp, J.M., Thompson, D.A. & F.W.G. Hill), pp. 49–53. Scientechnica, Bristol.

Wells, P.W. (1979) *The development of vaccines against*

Webster, A.J.F. (1981a) *Optimal housing criteria for rumi-*

pneumonic pasteurellosis in sheep. Veterinary Record, **104**, nants. In: *Environmental Aspects of Housing for Animal*

15.

Production. Proceedings of 31st Easter School, University of Gordon, W.A.M. (1963) Environmental studies in pig housing.

Nottingham School of Agriculture (ed. by J.A. Clark), pp.

IV. The bacterial content of air in piggeries and its influence 217–32. Butterworths, Sevenoaks.

on disease incidence. British Veterinary Journal, **119**, 263–

Webster, A.J.F. (1981b) *Weather and infectious disease in*

73.

cattle. Veterinary Record, **108**, 83–7.

Green, G.M. & Kass, E.H. (1965) *The influence of bacterial Williams, M.R., Spooner, R.P.L. & Thomas, L.H. (1975) Quan-species on pulmonary resistance to infection in mice sub-*

titative studies on bovine immunoglobulins. *Veterinary Record*, **96**, 81–4.

Record, **96**, 81–4.

British Journal of Experimental Pathology, **46**, 360–6.

Wiseman, A. (1978) Influence of environment on respiratory

Jones, C.R. & Webster, A.J.F. (1981) Weather-induced changes disease. In *Respiratory Diseases of Cattle. Current Topics in in airborne bacteria within a calf house. Veterinary Record, Veterinary Medicine, Volume 3* (ed. by W.B. Martin), pp.

109, 493–4.

149–57. Martinus Nijhoff, The Hague.

Chapter 18

Other Calf Problems

A.H. Andrews

Stillbirth/perinatal weak calf syndrome

249

have enlarged thyroids (greater than 14 g is suspect),

Joint ill or navel ill

249

parturition is slow and often there are retained placentae

Oral and laryngeal necrobacillosis

250

tae. Poor fertility may be seen with suboestrus and

Oral form

250

delayed ovulation, lowered milk yield, lowered libido in

Laryngeal form

251

bulls, ill thrift in cattle and lowered herd immunity.

Meningitis

251

Otitis

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Bovine papular stomatitis (BPS)

252

Joint ill or navel ill

Calcium, phosphorus and vitamin D deficiency

253

(see p. 455)

Copper deficiency

254

Hypomagnesaemic tetany of calves

255

Joint or navel ill is a common problem in the calf. At

Vitamin A deficiency

256

birth there is a sudden change from the fetal circulation

Iodine deficiency

257

to that of the newborn calf. The blood vessels in the

Iron deficiency

258

umbilical cord rapidly lose most of the blood within

Selenium/vitamin E deficiency

258

them but still remain patent, thereby allowing the in-

Zinc deficiency

260

roduction of infection. Infection can be caused by a

Furazolidone poisoning

260

single organism or a mixture. A wide variety can be

Iodism

261

involved including Streptococcus spp., Escherichia coli, Cerebrocortical necrosis (CCN, polioencephalomalacia)

261

Erysipelothrix insidiosa, Pasteurella multocida, Arcano-Urolithiasis

263

bacterium (Actinomyces, Corynebacterium) pyogenes

and Fusobacterium necrophorum. The problem nor-

mally arises from calving taking place in conditions with

Stillbirth/perinatal weak

poor hygiene. Often the calf will not have had sufficient

calf syndrome

intake of colostrum and usually the navel will not have

been treated. Many infections also enter via the tonsils.

This is an important cause of losses in the young calf

Infection entering the umbilicus may result in a local

and has considerable economic consequences to the

reaction at the point of entry into the body, between the

farmer. Very few studies have been undertaken to

muscle layers or in the peritoneum. In other cases entry

determine the factors involved. However work done in

is via the urachus and can lead to local infection. Other-

Northern Ireland (McCoy et al., 1997) showed that wise the bacteria may pass via the umbilical vein to nearly half (46 per cent) of the animals examined had the liver and then in the blood to the body. When infection is present in the blood it may cause a septicaemia another 22.7 per cent had severe trauma (mainly rib or or eventually result in chronic illness due to localization spinal fractures) from calving. Otherwise the largest in organs such as the heart, brain, eye and most often number of weak calves (36.7 per cent) had thyroid the joints, leading to joint ill.

abnormalities and within the study 20 per cent had low thyroid iodine levels. Other problems were of an infectious nature including leptospirosis (13.4 per cent), pneumonia (9.1 per cent) and bacterial isolation (9 per cent); virus isolation was low.

The signs vary and can be restricted to local inflammation of the navel or the abdominal wall muscles. In such Iodine deficiency is more common than recognized in

cases the navel is swollen, soft and usually painful. The suckler herds and dairy and suckler heifers who receive umbilical blood vessels are swollen at their base. Localized peritonitis may be difficult to detect. Where there arises from increased calf mortality as abortion, stillbirths, weak calves and neonatal mortality. The calves is septicaemia, the calf rapidly becomes ill with depression, pyrexia (40.5°C, 105°F) and accelerated respiration,

249

250 • Chapter 18

tory and pulse rates. The mucous membranes become the animals are dehydrated, parenteral or oral electrolyte solutions will be required. In the more localized There may be a varying degree of dehydration, followed forms involving the navel or urachus, the infected material by acidosis, recumbency and death.

terial should be removed. There are problems in the In cases of bacteraemia that localize, the signs are treatment of localized chronic infection such as joint

often missed for several weeks or even months. In some ill. In some cases the use of potentiated sulphonamides animals there is inappetance, dullness and an intermittent slightly raised temperature (39–40°C, 103–104°F). Surgical opening of the joints with removal of pus and Other signs depend on the organs affected. When there affected tissue and joint flushing can be useful. Slow is local infection of the urachus the animal will become release gentamicin polymethylmethacrylate (PMMA) unthrifty and slightly slow to move. There may be a beads have been used in cases of septic arthritis with slightly raised tail with micturition. In animals with evidence of concurrent osteomyelitis. The beads are localization in the heart valves, endocarditis results inserted after debridement of osteomyelitic bone via an with a heart murmur. If the eye is involved there is arthrotomy incision or with arthroscopic examination. panophthalmitis with hypopyon. In the case of meningitis there is likely to be nystagmus, hyperaesthesia

1994).

and tonic–clonic convulsions. The most common form
Control is dependent on whether the calves are on
is joint ill and one or more joints may be involved. In
their farm of origin or have been bought-in. If the
many cases there is bilateral involvement with pain and
former, then all navels should be dipped immediately
swelling, commonly of the carpal joints. Aspiration of
after birth in an appropriate disinfectant. Tincture of
the affected joints usually reveals thick pus. The animal
iodine or iodine teat washes are useful for this purpose.
tends to become lame and to have an altered stance.
Generally they allow the navel to be sterilized and help
to cause desiccation. Antibiotic aerosols are used but it
is often difficult to ensure the whole navel is completely

Necropsy

covered. In purchased animals the navels should be
Post-mortem examination may reveal the presence of
examined, and calves with enlarged navels rejected. The
infection in the umbilical vessels, which are swollen and
joints of the animals should also be inspected. The

contain blood. There may be localized peritonitis. In the navels should be dipped in an appropriate disinfectant septicaemic form petechial and ecchymotic haemorrhages are evident on the subserosa and submucosa of various organs. In the more chronic form various organs

Oral and laryngeal necrobacillosis

will show inflammation and abscessation.

This is also known as calf diphtheria and there are two

Diagnosis

forms: oral which is most common, and laryngeal. The

Diagnosis is aided by the presence of a swollen navel

condition is caused by *Fusobacterium necrophorum*.

as well as by the signs. There may be a neutrophilia and

blood culture may be helpful. The main difficulty is in

Oral form (see also p. 822)

differentiating the condition from other forms of enteri-

This is quite common and is usually sporadic in occur-

tis, septicaemia and locomotor problems such as mus-

rence although there may be outbreaks where hygiene

cular dystrophy (see p. 258). If surgery is contemplated

*is poor. In such cases it is probably spread by dirty milk
it is best to X-ray the joint lesions first.*

*pails, machine teats or feeding containers. Individual
cases sometimes occur where fibrous and coarse food is*

Treatment and control

*offered. Although mainly seen in housed calves, it can
also occur at pasture. Affected calves are usually up to*

Treatment of the septicaemia will usually involve

three months old and often have intercurrent disease,

the use of antimicrobials, which in the main should be

nutritional deficiency or their teeth are erupting. The

given intravenously. Appropriate antibiotics include

incubation period is about four days.

amoxycillin, ampicillin, oxytetracycline, sulphonamides

or potentiated sulphonamides. Chloramphenicol can be

Signs

of use in some countries and florfenicol has been used

successfully although not licensed for the purpose. In

The major sign is a swelling of the cheek, particularly

less severe cases other treatments such as penicillin and

in the region of the first cheek teeth. The calf is often

streptomycin may be given by the parenteral routes.

bright and active with a normal temperature. Opening

The duration of therapy should be at least five days. If

the mouth reveals a necrotic swelling in the cheek, in

Other Calf Problems • 251

which may be impacted food material, and there may

In the oral form, parenteral or oral therapy is usually

be a foul smell. The animal may salivate a little. In a few

successful. Suitable antibacterial agents include oxy-

cases there is also involvement of the tongue, which

tetracycline, potentiated sulphonamides, streptomycin,

may become swollen and protrude from the mouth. Ne-

sulphonamides orally or parenterally or penicillin par-

glected cases may extend to the nasal cavity, pharynx,

enterally. Therapy for three to five days is usually suffi-

lungs, abomasum and coronets of the legs.

cient in the oral form. When the animal is inappetant

it should be encouraged to eat. In the laryngeal form,

Necropsy

therapy needs to be continued for longer (e.g. 2–3

weeks) and should be parenteral. If breathing is very

On post-mortem oral lesions are usually well circumscribed then it may be necessary to undertake a tracheotomy and insert a tracheal tube. In some cases If the necrotic area is lost, an ulcer is seen.

success is only achieved by surgical removal of the necrotic area and then using an intratracheal tube until

Diagnosis

the laryngeal oedema has reduced.

The main differential diagnoses are foreign bodies in the mouth, papular stomatitis, mouth and jaw injuries

Control

and BVD/mucosal disease. All are quite easy to eliminate by thorough oral examination.

improved. The calves should be fed with their own buckets and quality feed should be used. The milk and

Laryngeal form

water buckets should be cleaned and disinfected after

This form is less common and is sporadic in occurrence. each feed and the feed bucket disinfected at least twice

*It has been seen in animals up to and over a year old.
a week. Occasionally, it is necessary to give oral antibiotics as a prophylactic measure. Suitable agents include chlortetracycline and oxytetracycline.*

Signs

These cattle tend to be dull with inappetance or anorexia. Often there is pyrexia (40.5°C, 105°F) and

Meningitis (see p. 901)

there may be stertor. Usually respirations are dyspnoeic to a varying degree. There may be a cough that is moist

This is an inflammation of the meninges. The condition and painful. Palpation of the larynx is resented and can

*is uncommon and most often occurs following a pre-
elicit the cough. The mouth may be foul smelling. Many*

existing disease such as septicaemia. The organisms

of these animals do not respond well to treatment and

that can be involved are usually bacteria and include

the diphtheritic area may become detached resulting in

Listeria monocytogenes, Escherichia coli, Pasteurella sudden asphyxiation or lung infection.

multocida and Haemophilus somnus. A secondary meningitis can follow infection with Mycobacterium

Necropsy

bovis. Although most cases are haematogenous in origin, a few result from spread of local infection or can follow disbudding, skull injuries, otitis media or frontal sinusitis. The lesion in the larynx is normally well embedded into the laryngeal cartilage. When lung lesions occur there are necrotic areas present surrounded by a catarrhal central infection causing local swelling and inflammation around the nerve trunks. Spinal meningitis will often lead to hyperaesthesia of the body, muscular pneumonia.

Diagnosis

tremors in the limbs and neck and an arched back.

The main differential diagnoses are laryngeal oedema, laryngitis, necrotic enteritis and vocal cord paralysis.

Signs

These may be hard to eliminate unless an endoscopic examination is undertaken or an exploratory laryngotomy is performed.

The condition usually starts with a sudden onset of

pyrexia and in many cases toxaemia. There is hyperaesthesia to any cutaneous sensation and muscular tremors of the neck and head with opisthotonus and pad-

Treatment

dling movements of the limbs. There may be ocular

In either case the animal should be isolated from the lesions with hypopyon and ophthalmitis. The animal may others. It should have its own feed and water buckets. appear blind and the pupillary reflex may be sluggish.

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Ophthalmoscopic examination may show the retinal

Signs

irises to be engorged with oedema of the optic disc.

In otitis externa the animal is well and has a normal temperature but the ear tends to be droopy with a foul-

Necropsy

smelling purulent discharge. In middle ear infection, unless bilateral, the head is rotated and there is a degree

On post-mortem examination the meninges are of incoordination. The animal is often dull and there is thickened and opaque, especially ventrally, and there

inappetance. Radiography may show rarefaction of the is engorgement of the meningeal vessels with haemorrhagic tympanic bulla.

rhaging. The cerebrospinal fluid (CSF) is often cloudy.

Diagnosis

Diagnosis

This is difficult in the live animal but the nervous

This condition is differentiated from brain or spinal

signs are an indication and there is a leucocytosis.

abscesses or injury by the rotation of the head.

Confirmation can only be obtained by examination of the CSF, showing it to be cloudy with a high white cell

Treatment and control

count and possibly bacteria present. The differential diagnosis includes coccidiosis (see p. 282), septicaemia

Treatment of the external form is relatively simple and

(see p. 204), vitamin A deficiency (see p. 256), hypo-

involves the local application of antibiotics, in practice

magnesaemia (see p. 255) and poisoning with various

often from intramammary tubes. Suitable antibiotics

substances (see Chapter 54)

include cloxacillin, chlortetracycline and penicillin and streptomycin. Otitis media is much less satisfactory to treat and involves puncturing the tympanic mucosa,

Treatment and control

irrigation with antiseptic solutions and local and par-

Treatment involves the use of a broad-spectrum anti-enteral antibiotics. Prevention is difficult as the cause is microbial that will penetrate the blood–brain barrier.

often unclear but it should involve adequate treatment

The potentiated sulphonamides or chloramphenicol of all cases of calf pneumonia.

in countries where available are probably most useful.

Florfenicol has also been used although it is not

licensed for this use. These preparations are able to

Bovine papular stomatitis (BPS)

diffuse into the CSF. Results of therapy are generally disappointing. Prevention is difficult but should involve

This is caused by a virus of the genus parapoxvirus.

the rapid treatment of all septicaemias, otitis or local

The condition is very common throughout the world.

injuries. Disbudding should always be undertaken with

Lesions can occur in any age of animal and both sexes.

care.

However, most lesions are seen in young cattle. The virus causes pseudocowpox in cows (see p. 364). The organism has a high morbidity and is found in the saliva

Otitis *(see also p. 903)*

and nasal secretions. Spread is by contact. The disease is usually of little importance except in the differential Otitis media and externa both occur. The condition has diagnosis of other oral lesions, although occasionally it recently been more frequently diagnosed in calf-rearing can be of financial significance. The condition found is units in America and Britain. The cause is unknown but variable but can be seen in calves of seven days old or most outbreaks have been preceded by enzootic pneumonia. It can be transferred to others, particularly following a bite, cut or scratch. Most lesions are local but occurs along the Eustachian tube. In some outbreaks may take many weeks to heal. Occasionally, systemic up to 30 per cent of calves have been involved. In in-

signs occur (see p. 822).

dividual cases of otitis media there may be extension from an otitis externa or from navel infection via

Signs

the haematogenous route. The organisms involved vary but vitamin A deficiencies have been reported in some

The lesions vary in severity. The mild form is the most outbreaks. The external ear form is such that there are common with the animal remaining healthy, eating well

often streptococci and staphylococci present. When with no evidence of pyrexia, diarrhoea or respiratory

there is middle ear infection these two species may distress. The lesions tend to be ring-like and pathogno-

*be present, or *M. haemolytica*, *Haemophilus* spp. or monic. The periphery of the lesion is a thin red zone,*

**Neisseria catarrhalis*.*

within which is a white, slightly raised area of hyper-

Other Calf Problems • 253

plasia, with a yellow or brown centre due to tissue

to primary and secondary deficiency, but conditions necrosis. Lesions heal from the middle outwards. Most

relating to their lack are very rare in calves. The diseases of the skeleton that do occur are usually associated with erupting teeth. Occasionally, a second type of lesion is a brownish-purple colour and usually the size problems.

of the ring lesions. It heals from the middle outwards. Calcium is well absorbed in the calf's small intestine, producing a horseshoe shape lasting from four days to about two weeks. However, areas of lesions may last is secondary deficiency, which could possibly occur if several months overall.

very high levels of cereals were fed without additives or The severe form is less common and often associated high phosphorus levels were used. The daily calcium with other intercurrent disease, e.g. parasitism. Lesions requirement of calves is 10–30 g (1/3–1 oz) depending on are slightly raised, diffuse and roughened, and are size and growth rate.

yellow or grey in colour. They are seen in any part of Phosphorus is very efficiently absorbed from milk, the mouth and can involve marked sloughing of the but less so from dry feeds. Primary deficiencies may mucosae. As the more diffuse lesions heal, an underlying resemble rickets. Secondary problems resulting from ing circular form often remains. In some cases saliva is low vitamin D levels, high calcium or high vitamin A are held in the mouth making the lips wet.

rare. Phosphorus deficiency is widespread due to the types of soil present in an area. Leaching by rain or constant removal by cropping can lead to phosphorus-

Necropsy

deficient soil. Excessive calcium, iron or aluminium Death from BPS is rare. Most lesions are in the mouth can also result in the problem. The optimum calcium : or rhinarium but may occasionally be seen in the phosphorus ratio is 2 : 1.

oesophagus, rumen, reticulum and abomasum. Typical Vitamin D is usually provided by good quality hay cally, there are no vesicles and on histology there is a

and exposure to sunlight. Present feeding systems include ballooning degeneration of the stratum spinosum cells, include supplementation of milk substitutes and so do which may contain eosinophilic intracytoplasmic inclusion bodies. not normally predispose to the problem, although

lush green feeds contain much carotene and other substances that have anti-vitamin D properties.

The optimal daily intake is 7–12 iu/kg body weight

Diagnosis

(3.5–6 iu/lb).

Diagnosis is dependent on examining the lesions, the animal remaining healthy, viral isolation, histopathol-

Signs

ogy and use of an electron microscope. Immunity can

The signs are usually only seen in the best-growing be measured by the serum neutralization test but levels calves. There is some degree of lameness, particularly of are usually low. The main conditions to be differentiated the forelimbs, which are bent forwards or laterally. The ated are foot-and-mouth disease, mucosal disease,

limb joints and costochondral junctions are swollen. In malignant catarrhal fever, rinderpest, vesicular stomatitis, some calves the back will be arched and in severe cases BIV, BLAD and mycotic stomatitis.

the tail is elevated. There may be a marked tendency to lie down.

Treatment

Treatment is not justified but in severe cases concurrent

Necropsy

infections can be treated. Antibiotic therapy is recom-

On death the animal is in poor condition. The ribs are mended to resolve secondary infections. Prevention is easily cut and the limb bones are soft with thin, compact not practical at present but the condition is less severe bone and they are easily fractured. The joints tend to in healthy herds so good nutrition and freedom from be enlarged with thickened epiphyseal cartilage.

parasites are important.

Diagnosis

Calcium, phosphorus and vitamin D

Diagnosis is usually on clinical signs. The alkaline phos-

deficiency (see p. 462, 791)

phatase level will be raised. The levels of serum calcium and phosphorus will depend on the cause of the condition. These conditions are all closely related and it is best to consider them together. All three compounds can lead to vitamin D or phosphorus deficiency. Radiographic examination shows the bones to lack density and the ends have a diffuse appearance. The epiphyses tend to the form of protein. High levels of iron, zinc, lead, cadmium and calcium carbonate also reduce copper absorption.

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ash : organic matter ratio will be reduced from the normal 3 : 2. The main problems to be differentiated are chrome oxidase, which regulates oxidation processes copper deficiency (but the plasma and/or liver copper

Copper is concerned with the formation of cytochrome oxidase, which regulates oxidation processes copper deficiency (but the plasma and/or liver copper

and electron transfer in tissues. It is also part of the concentrations will be low), arthritis and epiphysitis. enzyme lysyl oxidase, which is used for elastin or collagen synthesis and deficiency results in skeletal defects

Treatment and prevention

and blood vessel fragility. Copper is also present in caeruloplasmin, which releases iron from stores into Treatment varies according to the cause. In minor Ca : plasma for erythropoiesis and deficiency results in P imbalances sufficient vitamin D is all that is necessary. anaemia.

Where the skeletal deformities are pronounced, treatment will have only limited effect. Otherwise in calcium deficiency check there is no excess phosphorus present.

Signs

Calcium should be provided but not to excess as this

The signs of primary deficiency are that the calves have may cause other deficiencies. If phosphorus is deficient,

a reduced growth rate; sometimes there is a scour but dicalcium phosphorus or disodium phosphate should not usually as pronounced as in secondary deficiency.

be used. The ratio of calcium : phosphorus of 2 : 1 should

There may be a stilted gait with some ataxia develop-

be provided by the diet. A vitamin D injection of

ing after exercise, but recovery occurs after rest. Ribs

3000–5000 iu/kg body weight will provide adequate

and limb bones may develop spontaneous fractures, the

levels for one to three months. Response to treatment

shaft thickness may be reduced and there is osteoporo-

is usually slow. Prevention requires ensuring adequate

sis. Thickened epiphyses, particularly in the fetlock

levels of minerals and vitamin D in the diet. Bone meal

region, may be noted, and stiffness of the joints.

and dicalcium phosphate are ideal sources of both cal-

Secondary deficiency is usually seen in calves sucking cium and phosphorus but they are expensive and

the dam or grazing. There is again a stiff gait and

cannot be used in many countries. Ground limestone is

unthriftiness. Molybdenosis is characterized by severe

a useful cheap method of ensuring adequate calcium in

scours. Some calves become very lame with epiphyses

the diet, but its overuse can result in other mineral

that are painful to palpate and usually the distal ends deficiencies.

of the metapodial bones are enlarged. On radiography there is a thickened irregular epiphyseal plate, and the
Copper deficiency *(see Chapter 21)*

metaphyses are thickened. In some animals depigmentation of the hair occurs.

This is either primary due to a lack of copper in the diet or secondary when the dietary level is adequate but

Necropsy

there is a failure in digestion, absorption or metabolism of the copper. In calves, deficiency can be seen at a few

On post mortem there is usually emaciation with

weeks old although it is much more common when

anaemia seen as thin, watery blood and pale tissues.

three or four months old. This is because copper is

Where copper levels are low there are deposits of

stored in the liver and there is preferential absorption

haemosiderin in the liver, kidney and spleen. The limb

from the dam. Milk contains little copper although

bones may show evidence of rarefaction and fracture.

levels are high in colostrum. In the milk-fed animal

A thickening of the epiphyseal plates, particularly of the absorption in the small intestine is high (up to 80 per cent) but this falls as the animal becomes a ruminant. In the small intestine there may be villous atrophy. Histologically, the bones show osteoporosis.

menting with copper and so most cases arise in calves sucking their dams or at grass. Problems often occur in

Diagnosis

the spring or summer when the mineral content tends to be lower.

In practice the majority of cases are diagnosed because

Primary deficiency depends on the soil type and is of the area where the animals have lived, and most common on sandy soil, particularly where there is much rain and leaching, and on peat. Secondary deficiencies are an indication of the condition and it usually affects calves will be sucking a cow or at pasture. The signs give an indication of the condition and it usually affects are probably more numerous than primary. They can

several animals. Examination can involve looking for
be due to high molybdenum levels and this effect is
anaemia with a reduced erythrocyte count ($2-4 \times 10^{12}/l$;

Other Calf Problems • 255

normal, $5-10 \times 10^{12}/l$) and low blood haemoglobin (5–
been used to release copper slowly from an intrarumi-
8 g/100 ml; normal, 8–15 g/100 ml). Plasma copper levels
nal bolus. The use of pasture dressing annually with
are low (normal, $>15 \text{ mmol}/l$; deficient, $<0.9 \text{ mmol}/l$)
5.6 kg/ha (5 lb/acre) copper sulphate is effective. As
as are liver levels (normal, 100–200 ppm dry matter
there is a possibility of poisoning, animals should not
(DM); deficient, $<50 \text{ ppm DM}$). Cytochrome oxidase
graze the pasture until after heavy rain or three weeks
(normal, $>7.0 \text{ mmol}/g$ wet liver) and caeruloplasmin
after application. The copper supplementation of water
levels are low. Copper can also be estimated in the diet,
has been advocated (Farmer et al., 1982). Salt licks con-pasture and soil. Often
response to copper therapy
taining 0.5 per cent copper sulphate are safe in use but
gives an indication.

are not permitted in all countries.

Treatment

Hypomagnesaemic tetany of

*When therapy is undertaken it is important to confirm
calves (see p. 787)*

the presence of copper deficiency, as overdosing is toxic.

Oral administration of 1.5 g copper sulphate weekly is

*The condition occurs most commonly in calves on high-
very useful, but requires constant handling of the calves.*

milk or milk-substitute intakes that are receiving little

Parenteral administration of copper can overcome

other feed. It results from a hypomagnesaemia that may

the problem. Copper sulphate has been used at a dose

be associated with hypocalcaemia. The young calf has a

level of 200 mg/calf. A methionine copper complex can

serum magnesium level similar to the dam and receives

be administered as a deep intramuscular injection at a

extra magnesium in its colostrum. However, milk is

dose of 40 mg/calf, as can diethylamine copper oxy-

deficient in this element and if it constitutes most of the

quinoline sulphonate at a rate of 0.24 mg/kg body

feed then there will be a gradual fall in circulating magnesium by subcutaneous injection and copper edetate levels. This is partly allayed by the absorption as a subcutaneous injection of 50 mg. Experiments in sheep have shown high doses of copper methionine absorb magnesium very efficiently from the small and subcutaneously to be safer than calcium copper edetate large intestine in early life but this capacity reduces so and diethylamine copper oxyquinoline (Mahmoud & Ford, 1981) but it was considered that this might have been the result of the rapidity of absorption depending of age. Occasionally, hypomagnesaemia is seen in the young calf about two weeks old and this is due to poor of copper preparations in cattle showed copper edetate absorption, which can occur with diarrhoea, the feeding to be best with copper diethylamine oxyquinoline of liquid paraffin or the use of fibrous feed, which

sulphonate 19 per cent worse, aqueous copper methionate increases salivation and thereby causes the body to lose 36–48 per cent worse, and cupric sulphate produces magnesium. A calf requires 1–5 g daily, according to size giving the second-best result. The injections also cause a and growth rate.

local reaction, with copper diethylamine oxyquinoline sulphonate giving least damage, copper edetate pro-

Signs

ducing an intermediate reaction and copper methionate causing most swelling (Suttle, 1981a).

In the early stages there is hyperexcitability to stimuli with increased ear movements, interspersed with the ears being held back. There tends to be opisthotonus,

Prevention

ataxia and head shaking. The animal may have trouble

Prevention is to ensure that the level of copper in the drinking from a bucket on the ground. The temperature diet of dams and calves is at least 10 mg/kg dry matter is normal and the pulse rate rapid. Later on there are of feed. When deficiency has been determined it may

muscular fasciculations with jaw champing, frothing at the mouth and a spastic gait. Convulsions may occur. However, no such programme should be undertaken starting with the calf stamping its feet, pricking its ears, unless a sample of the animals has been checked to ensure blood copper levels are low. The timing of the first injection (in severely affected herds) is at about six weeks old, but subsequent injections need to be based on further blood sampling. Copper sulphate can be used as a drench at a level of 1.5 g weekly. Cupric oxide temperature is often raised to 40.5°C (105°F) due to needles have been used to alleviate hypocupraemia muscular exertion. If the convulsions are severe the pulse is often imperceptible, cyanosis develops and the

animal dies within about 30 minutes. In some cases given to calves in the form of magnesium boluses or in there are periods of relative normality between bouts molassed creep feed. of convulsions.

Vitamin A deficiency

Necropsy

On post-mortem examination there is usually extensive The condition results from a deficiency of the fat-haemorrhage and congestion of the organs including soluble vitamin A or its dietary precursor carotene. Sec-the aorta, mesentery, pericardium, gall-bladder and in-ondary deficiency can arise where there is sufficient tercostal walls.

vitamin A/carotene in the diet but it does not reach a normal tissue level due to a failure in digestion, absorption or metabolism. In calves the condition may result

Diagnosis

in skeletal changes, which can affect the brain or spinal

Diagnosis depends on the history of the feeding

cord. The condition can be congenital or postnatal and regimen used or presence of diarrhoea as well as the is often partly due to the nutritional status of the dam. signs present and serum magnesium levels (normal, Usually, a diet of green food will provide sufficient of 0.9–1.4 mmol/l, 2.2–3.4 mg/100 ml).

Clinical signs

the precursor carotene and hence vitamin A. Thus may occur at 0.12–0.33 mmol/l (0.3–0.8 mg/100 ml). problems do not occur at pasture until there are periods In animals that die the Ca : Mg ratio in the caudal of prolonged drought, which can cause deficiency in the vertebrae or rib bone is increased from a normal of calves of affected dams or beef calves about six months 70 : 1 often to over 90 : 1. The aspartate aminotransferase old.

and creatine kinase levels also tend to be raised

The condition is more common in housed animals fed because of the increased muscular activity. The main diets likely to be deficient in vitamin A such as straw,

differential diagnoses involve conditions resulting in cereals or sugar beet pulp. The dam's nutrition is important in that carotene in green food does not pass across the placental barrier until it is converted to vitamin A. course is usually longer) (see p. 733), arsenic, lead or mercury poisoning (but in all these there is colic and It can then be taken up by the fetus and stored in the diarrhoea and with lead there is blindness). Strychnine liver, as also can the vitamin A in water-soluble injections or fish oils. Colostrum is a major source of vitamin A may well result in night blindness.

A for the calf and the introduction of extra carotene or Encephalitis (which may be viral or bacterial in origin) vitamin A to the dam's diet precalving can be useful.

or meningitis may be difficult to determine. Clostridium

The vitamin A requirement for a pregnant cow is about

perfringens (welchii) type D produces apparent blind-80 iu/kg body weight daily and that for a calf is about

ness and a raised blood glucose level (normal,

40 iu/kg.

2.5 mmol/l, 45 mg/100 ml).

Calves that are fast-growing, stressed or in a high environmental temperature require more vitamin A.

Many factors that influence the vitamin A and carotene

Treatment and prevention

contents of feeds can lead to secondary deficiencies. Vit-

*Treatment should include the use of magnesium sul-
amins C and E help to prevent vitamin A loss, and the*

phate (50 ml of 25 per cent solution) subcutaneously

uptake of the vitamin is inversely proportional to the

and possibly calcium borogluconate intravenously.

phosphate present in the diet. The vitamin is not very

Magnesium given intravenously can lead to medullary

stable and so pelleting of the rations, storage at high

depression and cardiac embarrassment. If necessary

temperatures and rancidity all decrease the content of

the animal should be sedated with acepromazine or

the diet. Wood preservatives such as chlorinated naph-

xylazine. When the problem is the result of diarrhoea

thalenes inhibit carotene conversion to vitamin A and

then this should be rectified. The condition in the older prolonged oral use of liquid paraffin or other mineral calf is prolonged and there will be greatly reduced oils can produce a deficiency. Vitamin A is used to magnesium levels in bone, etc. Thus these animals will produce visual purple for the retina, normal epithelium need to be supplemented with 2–4 g magnesium oxide and bone, and for normal CSF absorption.

or 4–8 g magnesium carbonate daily.

Prevention involves the provision of roughage,

Signs (see p. 925)

usually as good quality hay, from ten days of age. This is difficult in suckler calves but can be overcome by

Congenital: Calves are born blind due to impingement feeding the cows with magnesium oxide (calcined magnesiumite) at a level of 60 g daily. Magnesium can also be increased CSF resulting in syncope with the calves

Other Calf Problems • 257

showing tonic–clonic convulsions, ventral flexion of the where convulsions are occurring. Subsequently, an ade-

head and neck, retraction of the eyeballs and tetanic closure of the eyelids. The calves are not blind. They may die during convulsions. In some outbreaks the allowances are doubled. Green feed or early-cut hay, affected calves develop severe diarrhoea and occasionally otitis media. good quality silage or dried grass should be given.

However, very high daily levels of vitamin A supplementation in the diet can lead to exostoses on the digits. Postnatal: One of the most common lesions is the presence and loss of epiphyseal cartilage. This produces lameness of large amounts of brown, bran-like scales in the coat and this is particularly seen in fast-growing animals. A reduction in growth rate may occur but is usually also the result of other deficiencies combining but dietary supplementation should be the primary aim.

with that of vitamin A. Classical xenophthalmia with thickening and whitening of the cornea is unusual.

When it does occur, it may be accompanied by serous

Iodine deficiency (see p. 301, 586)

ocular discharge. Nervous signs usually start with ataxia and weakness of the hind limbs and then the forelimbs.

This can be the result of a primary lack of iodine. Sec-

ondary deficiency is recorded following high intakes of

and can lead to fainting with animals showing tonic–

brassicac, high calcium ingestion, heavy bacterial con-

clonic convulsions for up to half a minute. The signs are

tamination of feed or water, a low level intake of linseed

similar to those for congenitally affected animals.

meal or other plants containing cyanogenetic glyco-

Night blindness is more likely to be seen in yearling

sides. Iodine deficiency occurs in most parts of the

cattle.

world where there is a high rainfall and there is little

exposure to oceanic iodine. Soils with a high calcium

content are likely to be deficient. The condition is

Necropsy

mainly seen in the newborn calf of a deficient dam, Following death it may be possible to see the constriction usually in suckler herds. Iodine forms part of the thyroxine, and a deficiency will result in the reduction of the optic nerve, or the cranial cavity or vertebral cord may be reduced in size leading to injury to the pituitary increasing the production of thyrotrophic spinal nerve roots. Histologically, there is squamous metaplasia of the interlobular ducts of the parotid salivary gland that is pathognomonic. The epithelium of the prepuce, reticulum and rumen shows hyperkeratosis. Calves born alive are prone to die if chilled, etc.

are prone to die if chilled, etc.

Signs

The liver may show focal necrotic areas.

Many of the affected calves are aborted or stillborn and usually there is evidence of thyroid enlargement

Diagnosis

(goitre). If the animal is born alive it will be weak and

Diagnosis depends on post-mortem findings, plus a

disinclined to suck. Occasionally, the gland will be felt history of a lack of green feed and the signs. The defecation pulsate. Very rarely areas of alopecia are apparent. Deficiency can be confirmed by determining plasma vitamin D levels. The cow is often slow in calving.

A levels (normal, 25 mg/100 ml; deficient, <10 mg/100 ml) and CSF pressure (normal, <100 mm H₂O).

Necropsy

The condition needs to be differentiated from hypomagnesaemia where the animal is not blind, lead poisoning where there is abdominal pain, tetanus where the thyroid glands are enlarged and heavier than usual (normal fresh weight 6.5 g; a weight greater than 14 g is suspect). Histologically, there is thyroid hyperplasia.

(Welchii) type D where there are high blood glucose levels. Bacterial and viral encephalitis and meningitis

Diagnosis

(see p. 251) usually result in pyrexia.

Diagnosis depends on the area or a diet containing goitrogenic plants. There is thyroid enlargement in the

Treatment and prevention

calves and several heifers or cows abort or produce still-

Treatment involves the parenteral administration of

born or weak calves. Plasma protein-bound iodine

aqueous vitamin A at a rate of 400 iu/kg body weight.

levels are low (normal, 24–140 mg/l). The plasma

The animals often respond quickly to treatment even

inorganic iodine can be used (optimum 100–300 mg/l;

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marginal 50–100 mg/l; low 20–50 mg/l; very low <20 mg/l).

from the cow's milk there is a need to supplement with

Most (90 per cent) of circulating iodine is bound as thy-

hay, straw and cereals. The only source of iron for veal

roxine and normal values are 36–89 mg/l (80–160 nmol/l).

calves on slats without roughage is the milk substitute

The thyroid weight is increased and the iodine content

and this needs to be supplemented. Levels less than

of the gland is low (normal, 15.6–39.0 mmol/kg DM;

19 mg soluble iron/kg DM of feed are likely to result in

deficient, 9.5 mmol/kg DM). Differential diagnosis is

problems.

mainly to eliminate other causes of abortion (see Chapter 37).

Signs

The main sign is a reduction in appetite followed by

Treatment and prevention

reduced weight gain. The mucous membranes tend to

Treatment is to ensure that the calf sucks and is kept

become pale, but death is extremely rare.

in a warm, draught-free environment. Thyroid extract

can be used at a dose of 1–2 mg/kg body weight (0.5–

Necropsy

1 mg/lb). Intravenous sodium iodide can be used at a

dose of 5–7 g for the young calf, but it is not without

Necropsy findings are of pale muscles with the blood

risk; potassium iodide can be used orally at about 3 g

thin and watery and clotting slowly. The liver tends to

per calf. Iodism (iodine poisoning) can sometimes

be enlarged and there is moderate anaemia. Diagnosis

develop (see p. 261).

depends on the history of the diet and signs. It can be

Prevention involves allowing the dams adequate

confirmed by haematological examination demonstrating a reduced erythrocyte count and low haemoglobin for pregnant and lactating cows. The level for calves value. The serum iron level is low (normally 30 mol/l, should be 0.12 mg/kg DM. Pouring on to the coat 7 ml of 167 mg/100 ml when born, reducing to 12 mmol/l, 5 per cent tincture of iodine weekly can be helpful. One 67 mg/100 ml at three weeks). The main differential diagnoses are those of copper and cobalt deficiency water or 800 mg potassium iodide per cow every two weeks by drench or added to the drinking water can work. Injections of iodized poppy seed oil can assist. A

Treatment and prevention

bolus of 3400 mg iodine with selenium and cobalt can release iodine for five to six months. Some fertilizers are high in iodine and seaweed meal can be given in the feed weekly to each calf as iron dextran or 0.5–1.0 g iron as

and typically contains 50–100 mg/kg DM iodine.

ferric polygalactofuranose. Vitamin B12 is also often used at levels of 5–10 mg/kg body weight. Prevention is by supplying milk substitute containing an iron con-

Iron deficiency

centration of 25–30 mg/kg DM. This will ensure that the animal has a normal appetite and growth and it will

Iron deficiency is not common in cattle and the primary help to produce pale meat suitable for veal (Bremner

condition is mainly seen in veal calves without access

et al., 1976). This is because the level is sufficient to give to roughage. The secondary condition usually follows

an acceptable blood haemoglobin without there being

heavy infestation with sucking lice such as *Haematopi-*

enough to produce much myoglobin. Most milk substi-

nus *eurysternus* and *Linognathus vituli* or after haem-tutes for calf rearing contain considerably more iron.

orrhage. The primary condition can occur in veal calves

or others fed predominantly raw milk or unsupple-

mented milk substitute. The calf has only sufficient iron

Selenium/vitamin E deficiency

for about three weeks after birth and milk is a poor

(see p. 302)

source of iron. In the case of veal calves there is an attempt to maintain iron levels low to keep the meat white. Over half the iron in the body is in the form of haemoglobin with small amounts present in myoglobin in muscular dystrophy, also known as white muscle or fish flesh disease. It can be seen at any age after birth. Vitamin E and/or selenium can be deficient and result in enzymes used for oxygen utilization. The normal Selenium deficiency is mainly dependent on the area where crops are produced. Soils derived from granite or pumice are deficient. Alkaline soils encourage selenium absorption by plants. The condition appears to be and solid feed (Holman, 1956). The calf's daily iron requirement is 50 g and as only about 2–4 g are received increased cost of bought-in feeds causing farmers to use

more home-produced crops for their animals. The
However, it is fully conscious with normal appetite,
accepted level of selenium in feeds is 0.1 mg/kg DM.
normal temperature and usually normal respiratory
Selenium is mainly used by the body in the production
rate, but the heart rate may be raised. In many cases the
of the enzyme glutathione peroxidase.

gait is abnormal and it moves by rotation of the hocks.
Vitamin E deficiency is much more dependent on the
In some cases the affected muscles are swollen and firm
type of crop grown and its storage, etc. Vitamin E levels
on palpation. It has been shown that there is increased
tend to be high in green pasture, silage, dried grass or
susceptibility to infectious diseases such as calf pneu-
kale. Adequate levels of the vitamin are also present in
monia due to delayed lymphocytic response.

cereal grains, well-cured fresh hay, maize silage and
brewers' grains, but deficiencies can occur on poor

Necropsy

quality hay, straw or root crops unless there is a suitable
supplement provided. Vitamin E tends to deplete with

On necropsy of calves with the sudden death syndrome storage. Calf diets high in unsaturated fatty acids, as can there are often no macroscopic lesions. Other cases may occur where cod liver oil, fishmeal, soya bean meal or linseed oil are fed, may become deficient due to their a slight pallor of the myocardium. Histologically, lesions oxidation, resulting in rancidity and the destruction of not otherwise apparent can be detected with a haema-vitamin E. Storage of grains when wet or with propi-toxylol basic fuchsin–picric acid method and these are onic acid can also reduce the vitamin E level. Normal considered to be peracute myocardial degeneration. In levels for growing cattle are considered to be 150 mg of acute muscular dystrophy there are localized streaks in a-tocopherol, and for the calf, milk substitutes should the diaphragm and skeletal muscles. In the latter they contain antioxidants and 300 iu/kg DM a-tocopherol. tend to be bilaterally symmetrical white or grey areas The condition affects muscles, particularly cardiac, in the muscles. In the heart there may be cardiac hyper-

skeletal and diaphragmatic. Deficiency can occur in trophy and myocardial degeneration with pulmonary suckler calves sucking mothers with low selenium or congestion and oedema. Histologically, there is no vitamin E levels or in artificially reared calves on deficient diets. The condition results from unsaturated fatty acids entering the muscle cells where they accumulate. In muscular dystrophy there is usually no cardiac involvement but the skeletal muscles show bilateral grey or white areas. They are oxidized to lipid peroxides, which result in degeneration and calcification. It is believed vitamin E helps prevent lipid peroxide formation within the muscle cells whereas selenium compounds with many

Diagnosis

unsaturated points are known as polyunsaturated fatty acids and these are particularly common in vegetable oils, which therefore predispose to peroxide formation. Diagnosis depends on the area and diet provided as

well as the signs. Decreased glutathione peroxidase levels occur (normal >23 iu/ml RBC) and the normal level of selenium in the blood is 0.63 mmol/l. There are

Signs

raised plasma creatine phosphokinase and aspartate

The signs vary in degree and in the sudden death

transaminase levels. The blood vitamin E level normal

syndrome the calf appears perfectly healthy but while

range is 3.0 to 18.0 mmol/l and it will be low in deficiency.

drinking or normally within 30 minutes of feeding the

Otherwise liver and kidney levels of selenium can be

animal will suddenly collapse. Death is usually within a

examined (normal, 3 and 30 mmol/kg DM). Response to

minute of collapse. Mortality is 100 per cent.

treatment can be determined as a means of diagnosis.

Acute muscular dystrophy is again sudden in origin.

The animal becomes dull and lies in lateral recumbency.

Treatment

There is respiratory distress, a heart rate often elevated

to 150 – 200 beats/minute and irregular. The rectal tem-

Therapy can involve the use of vitamin E and/or sele-

perature is normal, the calf is fully conscious and has normal eye reflexes. Most calves die within 6–18 hours. DL2-a-tocopherol acetate is about 6 iu/kg body weight and the mortality approaches 100 per cent.

Selenium can be injected as 0.1–0.15 mg/kg sodium selenite. The most common form seen is subacute muscular dystrophy and the morbidity is variable between about 10 and 40 per cent of calves. The signs depend on the muscles affected. The animal may stand stiffly; it is reluctant to move and when it does it may have a stiff gait. The calf is often weak and will not stand for long. The diet should provide sufficient vitamin E and selenium. Cows with calves at foot or in late pregnancy can be given a combined vitamin E/selenium injection to Treatment and prevention

supplement their calves.

Treatment involves oral medication with zinc sulphate at a level of 2 g weekly, or 1 g weekly by injection is

Prevention

useful. Any calcium oversupplementation should be

corrected and fibrous roughage should be reduced. A

For prevention, growing calves should be given a sup-

diet containing 50 p.p.m. zinc should prevent the con-

plement at the rate 0.1 p.p.m. selenium of the total

dition. Weekly oral medication with 0.5 g zinc sulphate

ration and 150 mg/head of α -tocopherol daily. Cows

can be helpful. Long-term control can be obtained with

should receive a supplement of vitamin E during the

zinc-containing fertilizer.

last two months of pregnancy. Injections of selenium

and vitamin E can be used; selenium bullets or soluble

glass intraruminal boluses are also of value. Pastures

Furazolidone poisoning (see p. 941)

can be top-dressed with fertilizer containing sodium

selenite 75–150 g/ha (1–2 oz/acre) or foliage dusting or

Furazolidone was a common form of prophylactic medi-

spraying can be undertaken at 17.5 g/ha (1/4 oz/acre).
cation in calves, which can result in problems of toxic-
Analysis of pasture should be undertaken to determine
ity. However, its use is now banned in many countries.
that toxic levels of selenium are not produced; this can
There are two syndromes, one of which (the acute)
occur at levels of 0.5 mg/kg. Selenium can be added to
involves overdosing at levels of 20–30 mg/kg body
the drinking water.

weight and the classic condition results from long-term
feeding of low levels, often 2 mg/kg. Both are usually the

Zinc deficiency

result of poor mixing, which allows some animals in a
group to receive more than the others. Mortality in both
cases is high and the chronic form may be seen several
This can be either primary due to a lack of zinc, or sec-
days after furazolidone feeding ceases.

ondary due to impaired uptake. It is usually seen in
calves from six to ten weeks old, particularly in the
period after weaning, and most commonly they are

Signs

housed. Usually, calves do not show signs on diets containing 40 ppm zinc, but it is probable that calcium and In the acute form there are nervous signs with hyper-highly fibrous diets reduce zinc availability and perhaps excitability, including muscle tremors, arched back and low copper levels reduce uptake. A congenital skin condition possibly circling, convulsions and death normally within a few days. When the chronic condition occurs there in requirement (see p. 181).

tend to be necrotic lesions and haemorrhages in the mouth and lower gut. This results in melaena or dysentery.

Signs

Signs usually occur about two weeks after the deficient

Necropsy

diet is introduced. The main signs are of poor growth with possible stunting. There is alopecia and paraker-
After death few lesions are seen in the acute condition
atosis often affecting the limbs, muzzle, vulva, anus and
but when chronic there are haemorrhages and necrosis

tail head. There are fewer lesions on the main part of in the alimentary tract. Haemorrhages are present the body. In some cases any wounds or abrasions will on the peritoneal and pleural surfaces and there is take longer to heal. Most animals do not die but skin decreased myelopoiesis in the bone marrow.

biopsies show increased thickness of all skin components and the stratum corneum contains nucleated epi-

Diagnosis

dermal cells.

Diagnosis depends on the signs and feeding the compound and can be confused with bracken poisoning (see

Diagnosis

p. 946) or anthrax (see p. 717).

Diagnosis is partly on the lesions and a biopsy shows parakeratosis. Normal plasma zinc levels are

Treatment and prevention

9–18 mmol/l (80–120 mg/100 ml). Serum alkaline phosphatase, albumin and amylase levels fall whereas serum

Little therapy can be given in either case but in the globulin levels rise. The calves usually start to respond

acute form furazolidone feeding should be stopped at to therapy in about a week.

once. Noise should be kept to a minimum and excite-

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ment should be avoided. Sedatives such as acepro- contain high levels of thiaminase. Amprolium is also a mazine, xylazine or magnesium sulphate can be helpful.

specific thiamine antagonist and has also been used

In the chronic form little can be done, except perhaps

experimentally. Molasses toxicity results in a similar

to give blood transfusions. Prevention, where allowed,

problem due to a fall in the proprionate levels. Thi-

involves the correct dosage of furazolidone being

amine is naturally synthesized in the rumen. It forms an

offered to calves and it should be thoroughly mixed. If

essential component of several enzymes used in gluco-

given with milk substitute from a bulk container the

lysis in the brain. Deficiency in thiamine results in

milk must be constantly agitated. Furazolidone in the

increased blood pyruvate levels and a decrease in the

micronized form or combined with diethyl sulphoxide

lactate : pyruvate ratio as well as a depression of the or other nitrofurans will reduce the risk. Therapy with erythrocyte transketolase level. This causes an interference with normal carbohydrate metabolism and the whether medicinal or prophylactic, should not be cerebral cortex in particular requires the oxidative repeated.

metabolism of glucose.

It is possible that thiamine deficiency might have a direct metabolic effect on the neurones, particularly

Iodism (see pp. 302, 823)

in the calf which is very dependent on the pentose pathway of metabolism in which the transketolase

The overuse of sodium or potassium iodide in therapy enzyme limits the rate of activity. Thiamine pyrophosphate is a coenzyme for several carbohydrate metabolic or ringworm can lead to iodism. The signs depend partly reactions and it is associated with transketolase in the on the form of administration. If intravenous the animal

pentose pathway of glucose oxidation. There tend to be can show considerable discomfort with dyspnoea, marked cerebral oedema and cerebral necrosis and the staggering and tachycardia. When given subcutaneously signs are mainly the result of an increase in intracranial there may be swelling following injection for about two pressure.

days and local discomfort for about two hours after The morbidity is usually low but occasionally up to administration. In the oral form the coat becomes stary, 25 per cent. However, mortality can be 25 to 50 per cent with a scaly skin and often a fine, white dandruff. There if not treated early, with higher levels in young cattle is excessive lacrimation and nasal discharge with, in (six to nine months) than older ones.

some cases, a degree of inappetance. If problems arise in treatment from the intravenous route then the iodine

Signs

should be given subcutaneously or orally. When signs occur following oral administration, treatment should In acute cases there is a sudden onset of nervous signs

be discontinued.

including blindness, muscle tremors, particularly of the head and neck, head pressing, jaw champing and frothy salivation. Animals tend to be hard to handle and in the

Cerebrocortical necrosis

early stages signs may be intermittent. Although the

(CCN, polioencephalomalacia)

animal appears blind, and the menace reflex is absent, the palpebral and pupillary reflexes are present. The

Aetiology

convulsive signs soon become continuous with the

animal becoming recumbent. The signs are then of

It is a deficiency of thiamine caused by endogenous

opisthotonus, nystagmus, optic disc oedema, often stra-

thiaminase. Thiaminase has been found to be produced

bismus and clonic–tonic convulsions which become

by Clostridium sporongenes and certain Bacillus spp.

worse when the animal is stimulated. The temperature

which can be found in cases of CCN. However, this does

is normal, the ruminal movements are normal but the

not mean they are the only factors (see p. 903).

heart rate is variable. Calves often die in one to two days although older animals show signs for a longer period. Recovery following therapy may well take two

Occurrence

to four days or longer.

It is a sporadic condition which can occasionally occur

The signs of the subacute form last for a few hours to as outbreaks. Most animals affected are fast-growing, several days and include blindness, head pressing and well-nourished animals between 6 and 18 months old. standing. The condition will resolve in some cases.

It can occur following deprivation of food followed by

However, in an outbreak of CCN some of the animals good grazing or feeding with concentrate.

show anorexia, partial impairment of eyesight and a

Similar syndromes can be produced experimentally

mild depression. Almost all of the subacute animals

by feeding large amounts of bracken or horsetail which recover within 24 hours of therapy.

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Although recovery may occur, some animals may still

(10)

Haematology virtually normal although total remain blind. The longer the time between onset of and differential counts may show a mild stress signs and therapy, the less favourable the prognosis. reaction.

When cattle remain dull and anorexic after three days'

(11)

Increased cerebrospinal fluid pressure, 200– treatment they are unlikely to recover and should be 350 mm saline (normal 120–160 mm saline). slaughtered.

(12)

Histology – bilateral necrosis in cerebral cortex (bisect brain longitudinally, put one half in buffered formalin, the other is deep-frozen).

Necropsy

(13)

Green fluorescence of brain when exposed to Most of the animals do not show any gross changes long-wave ultraviolet light.

in the body other than the brain. There is usually

Differential diagnoses are shown in Table 18.2.

increased intracranial pressure with a yellowing and compression of the dorsal cortical gyri. The cerebellum tends to be compressed into the foramen magnum and

Treatment

recovered animals show decortication of the motor area

Thiamine hydrochloride should be administered intra- and occipital lobes. Histologically there is bilateral

venously at a dose of 10 mg/kg (5 mg/lb) BW, and the

necrosis of the dorsal occipital and parietal cerebral

dosage should be repeated every three hours or so for

cortex and also, occasionally, the thalamus, basal

five treatments. A response will occur in 24 hours if

ganglia, lateral geniculate bodies and mesencephalic

animals are caught in the early stages, otherwise recovery is slowly progressive over several days. Multi-

vitamin injections are often used but although they are

Diagnosis

suitable for follow-up therapy, in the initial stages insuf-

ficient thiamine will be administered unless very large

Diagnosis can be made from the following:

doses are given.

(1)

History – age of animals, a change in feeding and

*Nursing is important and the cattle should be pre-
the condition of the animal.*

sented with wholesome food including at least 50 per

(2)

Signs – blindness, normal palpebral and pupillary

*cent good quality roughage. Rumen liquor from cattle
reflexes; normal ruminal movements, normal*

*on predominantly roughage diets may be helpful. The
temperature but otherwise many nervous signs.*

use of dried brewers' grains can help the conditions as

(3)

Blood pyruvate and lactate levels are increased.

they contain high levels of thiamine and others of the

(4)

Urine pyruvate levels increased.

(5)

Erythrocyte transketolase activity reduced

(Table 18.1).

(6)

Pyruvate kinase levels are much increased.

Table 18.2

Differential diagnoses.

(7)

Thiamine levels in erythrocytes, blood and plasma may be in the normal range.

Disease

Differential diagnoses

(8)

Blood creatine phosphokinase (CPK) levels may occur.

Listeriosis

Unilateral facial paralysis, pyrexia

(9)

Thiaminase levels increased in rumen liquor and

Lead poisoning

Abdominal pain, diarrhoea, no

in faeces.

pupillary reflex, no ruminal

movements

Coenuriasis

Slow onset, circling

Molasses poisoning

Similar to cerebrocortical necrosis

Table 18.1

Differences in thiamine levels within certain tissues

but history of feeding large

of the body in animals with or without cerebrocortical necrosis.

quantities and also glucose

(After Edwin et al., 1979.) TPP = total plasma protein.

levels fall whereas thiamine

levels remain normal

CCN (\pm SEM)

Not CCN (\pm SEM)

Amprolium poisoning

Similar to cerebrocortical necrosis

but history of feeding it

Liver dry (mg/g)

2.5 \pm 0.43

11.1 ± 2.11

Bracken or horsetail

Similar to cerebrocortical necrosis

Heart dry (mg/g)

2.5 ± 0.56

13.2 ± 2.12

poisoning

but highly unlikely to cause such

Brain dry (mg/g)

1.8 ± 0.37

7.7 ± 1.52

a manifestation other than

Erythrocyte

172

15

experimentally

transketolase

Haemophilus somnus

Infection, but usually pyrexia and

(% TPP effect)

neutrophilia

Other Calf Problems • 263

B vitamin group. Levels of 0.5–1.0 kg/300 kg BW

passage of small amounts of urine, often blood-tinged, (1–2 lb/6 cwt) have been suggested.

or the attempts are unproductive. Calculi may be present on the prepucial orifice hairs. There is usually evidence of mild to severe colic with kicking at the

Control

belly, paddling movements and tail swishing. In most

The precipitating factors for CCN are still not known,

untreated cases with complete urethral obstruction,

which makes it difficult to recommend preventive

there will be perforation of the urethra or bladder

measures. As the condition is the result of endogenous

rupture. When either takes place there is usually a

thiamine activity, provision of extra thiamine is of

period of relief from abdominal pain. When bladder

limited value. Most natural feeds contain thiamine at a

rupture has occurred the urine enters the abdomen,

level of 2 ppm and this, plus the vitamin synthesized in

which becomes distended and there is a fluid thrill

the rumen, is normally sufficient. Provision of adequate present on percussion. In those with urethral perfora- amounts of roughage should prevent the condition and tion, urine tends to dribble under the skin, causing a level of 1.5 kg roughage per 100 kg BW is suggested.

ventral abdominal distension, which will start to progress anteriorly. In most cases there is some toxemia and possibly uremia and this is seen as

Urolithiasis

inappetance, with increasing dullness of the animal, which will ultimately become comatose and die.

Urinary calculi are either organic or inorganic. The organic type are less common and form casts or urinary

Necropsy

deposits. The inorganic ones tend to be crystalline and are more common. The condition is usually seen in

At necropsy there is usually some degree of cystitis, calves that are housed, with milk substitute as their often with urinary deposits present in the bladder.

main source of feed or in weaned growing animals fed

When the bladder ruptures there is much fluid in the

high levels of concentrates. Some pastures are problem
abdomen and in those cases of urethral perforation
areas, which can be due to high plant oestrogen,
there will be erosion in the area of the calculus and
oxalate or silica levels. Most calculi in housed animals
urine, possibly with cellulitis, present subcutaneously.
contain calcium or magnesium ammonium phosphate
The position of the calculus can be ascertained by the
although struvite and oxalate deposits occur at times.
passage of a catheter.

At pasture, carbonates of calcium, magnesium and
phosphorus are most common. Vitamin A deficiency

Diagnosis

has been suggested as a precipitating factor both
indoors and when cattle are grazing. The urinary pH has
The diagnosis depends on the history, particularly of the
an influence and phosphate and carbonate calculi form
area and feeding as well as the sex of the animal and
more readily in an alkaline than an acid urine (adult
signs. If there is bladder rupture then the fluid can be
ruminant urine is alkaline with a pH of 7 to 9). Binding

aspirated from the abdominal cavity. It is often difficult of the calculi occurs with mucoprotein in the urine and to determine that urine is present without its odour and this is seen more frequently when oestrogens from appearance. Urinary crystals may be present on pre-plants or growth promoters are present, or the ration is pucial hairs and these should be analysed. The main pelleted.

differential diagnoses are ascites, intussusception and Urolithiasis can occur in all animals fed a predispos-constipation.

ing diet regardless of sex; however, the condition is mainly seen in the male because signs are not normally

Treatment

observed unless some form of urethral blockage occurs.

More cases are found in castrated than entire animals.

Treatment of the condition is primarily by surgery. If

Calculi can lodge anywhere in the urethra but occur

the animal is nearing slaughter and there is no bladder

most commonly at the sigmoid flexure of the penis with

rupture or urethral perforation then casualty slaughter

the region of the ischial arch being the second most is best. Otherwise, treatment is usually only successful common site.

in the early stages and all treated animals and others in the group must be carefully examined for several days subsequently. It may be possible to perform a urethro-
Signs

tomy and remove the calculi. Provided the stones are Most of the signs are associated with partial or com-
distal to the ischial arch then the provision of a ure-
plete blockage of the urethra. This is seen as frequent
throtomy in the perineal region may overcome the
attempts to urinate, which may be accompanied by the
problem and, if it proves impossible to remove the
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calculi, the opening can be made permanent. Medical
available at pasture and areas likely to produce urolithi-
treatment can include hyoscine butylbromide injected
asis are best grazed by female cattle.

intravenously or intramuscularly at a dose of 20–
40 mg/animal, or 5–10 ml of protein-free pancreatic

extract possibly repeated once or twice. Acetylpro-

References

mazine can give useful results acting as a smooth muscle

relaxant. Withdrawal of concentrates may assist the

Bremner, J., Brockway, J.M., Donnelly, H.T. & Webster, A.J.F.

condition and the provision of salt water following

(1976) Anaemia and veal calf production. Veterinary

relief of the blockage is useful.

Record, 99, 203–5.

Butson, R.J. (1994) Bovine septic arthritis: a review of current and future treatment regimes. Cattle Practice, 2, 315–21.

Edwin, E.E., Markson, L.M., Shreeve, J., Jackman, R. &

Prevention

Carroll, P.J. (1979) Veterinary Record, 104, 4–8.

Farmer, P.E., Adams, T.E. & Humphries, W.R. (1982) Copper

Prevention is partly dependent on feed alteration, and

supplementation of drinking water for cattle grazing

precipitation of phosphate can be avoided by having a

molybdenum-rich pastures. Veterinary Record, 111, 193–5.

correct ratio of calcium to phosphorus, which should be

Holman, H.H. (1956) Changes associated with age in the

at least 1.2 : 1, but levels up to 2.5 : 1 have been suggested. The concentration of magnesium in the diet
Journal, **112**, 91–104.

should be kept low and this means that the maximum

McCoy, M.A., Smyth, J.A., Ellis, W.A. & Kennedy, D.G. (1997) amount of magnesium oxide that should be added to

Stillbirth/perinatal weak calf syndrome. *Cattle Practice*, **5**, the diet is 200 g/t (1/31–4.

2 lb/ton) of feed. In the concentrates,

up to 3 per cent salt has been recommended and it is

Mahmoud, D.H. & Ford, B.J.H. (1981) Injection of sheep

thought to have an ionic effect rather than just causing

with inorganic injections of copper. *Veterinary Record*, **108**, 114–17.

diuresis. Such diets should only be used where there is

Suttle, N.F. (1981a) Comparison between parenterally

always free access to water. The addition to feed of

administered copper complexes of their ability to alleviate

urinary acidifiers such as ammonium chloride or phos-

hypocupraemia in sheep and cattle. *Veterinary Record*, **109**, phoric acid can be helpful. In animals at pasture the use

304–7.

of salt in the water can reduce the concentration of

Suttle, N.F. (1981b) Effectiveness of orally administered cupric silicic acid in the urine, thereby preventing silica calculi

oxide needles in alleviating hypocupraemia in sheep and

formation. Adequate water supplies must always be

cattle. Veterinary Record, 108, 417–20.

Growing Cattle

Chapter 19

Endoparasites

S.M. Taylor and A.H. Andrews

Introduction

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ment of parasitic disease, to inquire whether the two

Nematodes

267

preconditions have existed and to involve their separa-

Parasitic gastroenteritis (PGE)

267

tion as part of the therapeutic and prophylactic advice

Parasitic bronchitis (husk, hoose)

272

given.

Stephanurosis (kidney worm)

274

Bunostomosis

275

Haemonchosis

275

Trematodes

276

Nematodes

Fasciolosis

276

Paramphistomosis (stomach fluke disease, intestinal

Parasitic gastroenteritis (PGE)

amphistomiosis)

279

The term itself is currently specifically associated with

Schistosomosis (bilharziosis)

279

Cestodes

280

the presence of large numbers of nematodes in the abo-

Taenia saginata

280

masum and intestines rather than any other endopara-

Echinococcus granulosus and hydatid cysts

281

sites. The nematodes in the abomasum are generally

Protozoa

282

considered to be the primary pathogens, with those in

Coccidiosis

282

the intestines playing a lesser but synergistic role.

Neosporosis

283

In temperate areas the predominant worms in the

Toxoplasmosis

284

abomasum are those of the genus Ostertagia, with O.

Sarcosporidiosis (sarcocystosis)

284

ostertagi the most important and numerous. In the small intestine *Cooperia oncophora* and *Nematodirus helvetianus* are commonest.

Introduction

There are two common forms of ostertagiosis, type I and type II, and since they are the result of different

Although cattle of all ages may become infected with

manifestations of the bionomics of O. ostertagi they will many species of parasites, clinical disease caused by parasite described separately.

asitism is mainly observed in groups of animals under

18 months of age, especially when two preconditions

TYPE I OSTERTAGIOSIS

coincide:

This form of the disease is characterized by a profuse

(1)

The availability of large numbers of the infective

watery diarrhoea in calves at grass. The faeces, because

stages of the parasite (a variable that is usually

of the grass diet, is usually green. There is rapid loss

dependent on the relationship between the bio-

of weight in severe cases, coupled with a slight hypo-

nomics of the parasite and suitable maturation

albuminaemia due to protein loss and in chronic cases

conditions).

this can eventually result in submandibular oedema.

(2)

The presence of susceptible cattle grazing on the

*It is most common in late summer and autumn in
contaminated area.*

northern temperate areas.

*When these two conditions are fulfilled, the resultant
simultaneous maturation of large numbers of parasites*

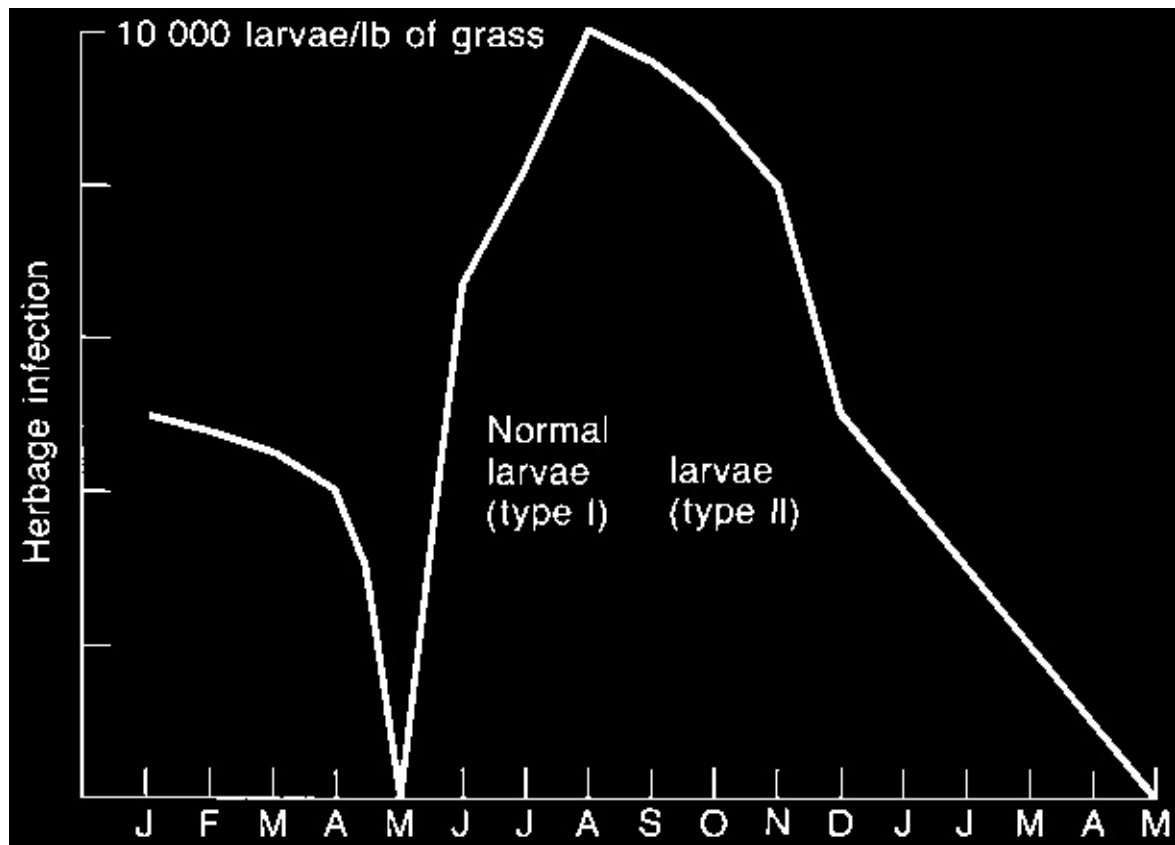
Aetiology and epidemiology

in a specific host organ produces severe tissue disruption and the consequent signs associated with the par-

*The direct cause is the ingestion over a relatively short
asite involved. The gastrointestinal tract, lungs and liver
period of large numbers of the infective larvae of O.*

are the organs most commonly affected. It is prudent,

*ostertagi. The presence of such large numbers of larvae therefore, when
considering the diagnosis and treatment is a result of several epidemiological
interactions, a*



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Parasitic gastroenteritis normally occurs when calves or non-immune older cattle are grazed on pastures on which a large number of infective larvae are present.

Typical cases occur in dairy herds where autumn-borne replacement calves are put out to graze for the first time in April or May. The pastures used are frequently close inhibiting

to the farmhouse to enable both ease of inspection and rehousing should weather conditions deteriorate. For

these reasons the same fields frequently have the same use each year. The calves remain on the fields until midsummer, excreting eggs that initiate the midsummer larval increase. At that point, when grass in such paddocks requires resting, the farmer has aftergrass available after conservation for silage or hay. The calves

Fig. 19.1

Normal annual pattern of worm larvae on pasture in northern temperate countries.

anthelmintic treatment. Shortly afterwards, by which time the grass in the original fields has recovered slightly, spring-born calves are put out on to it. If no prophylactic measures are taken, the inevitable result of severe type I ostertagiosis ensues a few weeks later advice on treatment and prophylaxis is to be offered.

in the latter calves, caused by ingestion of massive

The annual pattern of fluctuations of infective larval numbers of infective larvae by fully susceptible cattle

numbers on calf grazing was described by Michel over a relatively short period of time.

(1969), and can be seen in Fig. 19.1, which shows that the number of infective larvae in northern temperate

Pathogenesis

areas is lowest in May and June but rises to a peak in late August and September. Summarized, the pattern

Ingested infective larvae of *O. ostertagi* develop in the arises from the following sequence of events. Calves put

gastric glands in the abomasal mucosa, emerging 18–21

out to graze in April or May, on grazing that has been

days later as adults. Whilst in the mucosa their presence

used for cattle (and especially calves) during the pre-

produces distension of the parasitized acini, which in

ceding year, ingest some of the infective larvae remain-

turn causes several pathological and biochemical

ing on the pasture from the contamination produced in

lesions. Firstly, the hydrochloric acid-producing parietal

the previous summer. These infective larvae develop

cells in the acini are destroyed and replaced by rapidly

into adults in approximately three weeks and egg-laying

years in the British Isles the maximum number of infective larvae is found in southern England in late July, and albumin can leak outwards into the lumen. As a result in the west of Scotland and Northern Ireland in mid to late August. Wet summers produce an earlier peak but bacteriostatic and bacterial overgrowth of the damaged numbers of infective larvae decrease more quickly than wall results.

normal due to more rapid depletion of the numbers in faecal pats and to dilution due to the more abundant

Signs

grass growth under these conditions. Conversely, in dry summers the build-up is delayed; release of larvae from

When all the above events occur the intestinal metabolism does not take place under the autumn rainfall is also affected and the classical signs of acute fall and the larval contamination is maximal thereafter. diarrhoea, inappetance and weight loss commence.

TYPE II OSTERTAGIOSIS

of infective larvae remaining on pasture can be sufficiently high to cause normal type I disease from 4 to 6 weeks after going to grass. This form of the disease is usually found in yearlings in

the late winter or spring following their first season of grazing. Affected cattle can be housed or outwintered.

Nematodirosis in calves

Aetiology and epidemiology

*Parasitic gastroenteritis due to large infections of the nematode *Nematodirus battus* has recently been*

As with type I disease the direct cause is the simultaneous observed. Normally recognized as a parasite of sheep,

*neous maturation of large numbers of *O. ostertagi*. The it has recently been observed to be able to be trans-circumstances surrounding the event require some*

mitted by cattle, both on farms where annual alternation of sheep and cattle has taken place and even where disease is not when infective larvae on the pasture are cattle only are kept, and has caused severe outbreaks most abundant.

of diarrhoea in calves.

Infective larvae present on pasture after September and ingested from that time until the following spring undergo a change in their normal parasitic develop-

Cooperiosis in calves

ment, which results in a period of delayed development

In the last decade, as a result of the widespread use of

at the early fourth larval stage while within the abo-

suppressive regimes with avermectins and moxidectin

masal wall. The behavioural change has been shown to

that are more effective against Ostertagia than Cooperia be more common in some strains of O. ostertagi than

ria species, the prevalence of Cooperia oncophora, the others and to be brought about by either cold or desiccation - commonest of that genus in northern Europe, has

cation in their preparasitic exposure (Armour et al. , increased in comparison to Ostertagia species. In faeces 1969). In the late autumn, calves that have not had

samples taken during the first two-thirds of the grazing

adequate prophylactic treatment may harbour many

season on farms where intermittently applied aver-

thousands of such larvae but relatively few normally

mectins have been routinely used for prophylaxis,

developing larvae or adult worms. Type II ostertagiosis

Cooperia species eggs can form up to 90 per cent of the results when these inhibited larvae resume their development, with *Ostertagia* species forming the

remainder in the late winter or spring, the emerging larvae

majority of the remainder. In such circumstances large

burdens can produce the same lesions as those causing type I

Cooperia oncophora worm burdens can accumulate in disease.

a short time when hatching conditions are favourable.

These can result in outbreaks of acute diarrhoea and

ATYPICAL FORMS OF PARASITIC

loss of condition, and are rather similar to *Nematodirus*

GASTROENTERITIS

C. battus infections in lambs in spring, although in calves they usually occur in July or early August. If left

Parasitic gastroenteritis in beef herds

untreated calves normally recover after a few weeks

This is not usually a problem in spring-calving herds,

and become immune to *Cooperia oncophora*, but the

since most of the infective larvae are consumed by the

loss of condition can be prevented by prompt

adult cows, which normally have enough immunity to

treatment.

prevent a high percentage of worms establishing and producing eggs. The calves that might transmit the

Parasitic gastroenteritis in adult cattle

infection are too young to consume much grass in the early part of the season. As a result the peak of infec-

Although uncommon, since most cattle acquire immature larval numbers does not develop until September

nity by the age of 18 months, occasional individual cases

or October when most calves are weaned, treated and

can occur. Bulls that are grazed on calf paddocks and

housed. Conversely, in autumn-calving herds in which

cows, which due to debilitating intercurrent diseases

calves are weaned in the following spring and grazed in

such as fascioliosis may have some of their normal

the same manner as dairy replacements, the same epi-

immune reactions depressed, can both suffer from type

demiological picture can result, i.e. type I ostertagiosis

II ostertagiosis.

will occur in the absence of preventative measures.

Diagnosis

Early-season type I ostertagiosis

Affected animals invariably present with diarrhoea,

In areas where climatic conditions allow autumn-born

which may be present to a greater or lesser degree in

calves to be put out in March or early April, the number

all members of the group. Younger animals are fre-

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Table 19.1

Differentiation between type I and type II ostertagiosis.

Type I

Type II

Seasonal incidence

Predominantly July–November;

February–May

occasionally April–May

History

Usually in calves at grass for the first time, heavily

Always in calves that grazed on heavily stocked

stocked (8–10/ha). Occasionally in beef yearlings

pasture during the previous autumn, usually in

during the second year's grazing, and in individual

younger members of group. Can also occur in cows and bulls transferred from hill herds to more bulls grazing calf paddocks. Occurs usually when intensive systems

housed or within 3 weeks of being put to grass

Clinical signs

Profuse green diarrhoea; will only usually recur

Intermittent profuse diarrhoea. Recurs every 2 once approx. 10 days after treatment. Rapid loss weeks until supply of inhibited fourth stages is of body weight. Morbidity high, mortality low exhausted. Morbidity low, mortality high

Laboratory findingsa

Not usually anaemic

Mild anaemia

PCV > 0.3

PCV 0.22–0.26

RBC > 7 \times 10⁶/mm³

RBC 5.6 \pm 10⁶/mm³

Hb > 10 g/100 ml

Hb 8.4 \pm 0.5 g/100 ml

Plasma pepsinogen 2–5 iu

Plasma pepsinogen 2–8 iu

Post mortem

pH abomasal contents

pH abomasal contents

>5.0

>5.0

>50 000 adult

>50 000 adult

O. ostertagi

O. ostertagi plus large numbers of

Severe abomasal reaction

immature fourth stages

Severe abomasal reaction

a PCV, packed cell volume; RBC, red blood cell; Hb, haemoglobin.

quently the most severely affected. There will be a

(3)

Plasma gastrin. The plasma concentration of the

history that indicates grazing on potentially infected

hormone gastrin has been shown to rise at the time of

pasture during the preceding four to eight weeks in the

patency in experimental infections and to peak at levels case of type I ostertagiosis or during the previous late between 500 and 1000 pg/ml. Although still under investigation it may be used as a substitute for plasma

Specific diagnosis (see Table 19.1) can be assisted by pepsinogen in the future.

the following tests.

(4)

Post mortem findings. In both type I and type II

(1)

Faecal egg counts. Despite the dilution of faeces ostertagiasis the abomasum (providing the animal caused by diarrhoea, the nematode egg counts will be has remained untreated) will contain large numbers of in excess of 1000 eggs/g. Counts may be higher in less adult *O. ostertagi*. The number can vary from 50 000 to severely affected calves that have not yet become

1 000 000 in extreme cases. In type II disease pepsin severely diarrhoeic. In older animals and in some indigest of the abomasal mucosa or incubation in luke-

viduals in type II cases the faecal egg counts may be low warm normal saline will reveal large numbers of inhibited fourth stage worms. The pH of the abomasal contents will be raised to pH 4.5.

other tests should be borne in mind.

(2)

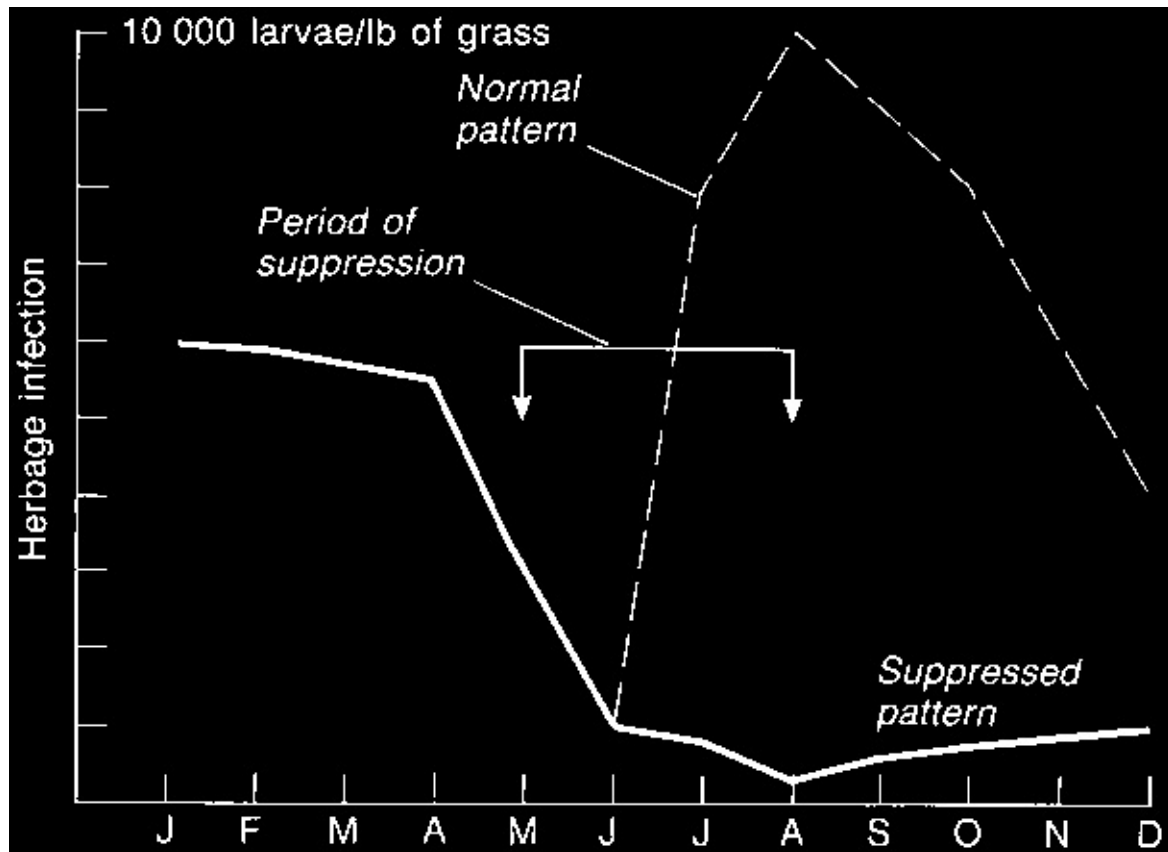
Plasma pepsinogen. In healthy calves the normal

Treatment (see Chapter 60)

level of plasma pepsinogen is less than 1 international unit (iu) of tyrosine. In affected calves the level will normally be greater than 3 iu, and in very severely diseased anthelmintics currently available such as levamisole, individuals up to 4.5 iu. It should be pointed out that in Type I disease can be treated with almost any of the benzimidazoles or avermectins. Type II disease requires adult cattle the normal pepsinogen level can be 1.5–2 iu, almost that found in calves in the prepatent period the use of some of the modern longer acting benzimidazoles or pro-benzimidazoles or avermectins such as of type II ostertagiasis. Plasma pepsinogen levels in

ivermectin, doramectin, abamectin, eprinomectin or adults therefore should be carefully evaluated.

moxidectin. The avermectins and moxidectin are much



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the most effective in removing both adult and inhibited although not ovicidal have an even longer half-life and fourth stage parasites as a result of their persistence and will inhibit further infections for at least two weeks. On slow excretion. Levamisole is much less effective for the other hand, levamisole has a rapid excretion rate

type II disease, as it has a poor efficiency against inhibition and is also not ovicidal. If reduction of contamination of worms due to its rapid excretion (see p. 1021), and as well as nematode removal is intended, cattle treated with benzimidazoles can vary in efficacy depending on the time of treatment in relation to the onset of inhibition, at least overnight, if not for 24 hours. After this first treatment of the worms, i.e. in the early and late stages of inhibition in early to mid July, and depending on the availability of grass and its previous grazing history, the metabolism, and therefore absorb more anthelmintic; in calves may require no further treatment until housed in the middle of the inhibition period they absorb less, and late autumn, although in practice many are treated four to six weeks after the first movement. Although highly persistence necessary to kill the worms.

effective and the most economical method of prophylaxis when carried out carefully, it should be pointed out

that although excellent for control of gastrointestinal

Prevention

nematodes it is not completely effective in the preven-

As for most parasitic diseases, prevention is more cost-

tion of lungworm infection caused by Dictyocaulus

*effective than treatment. Since there are no vaccines yet
viviparus.*

available for gastrointestinal nematodes, numerous

schemes that combine grazing management and

Suppressive strategies: These have been introduced

anthelmintic therapy have been developed. The

gradually since the mid 1970s and are also based on

methods fall into three types: (i) evasive; (ii) suppres-

knowledge of the epidemiology of the infections.

sive; and (iii) dilution.

Unlike the evasive systems, which allow natural infec-

tion and pasture contamination to take place in spring

Evasive strategies: The basis of this category (also

and early summer, the basis of these methods is to sup-

called the 'Weybridge' system) was first enunciated by

press egg production during that period (Fig. 19.2). If

Michel (1968) at the laboratory of the same name.

pasture contamination is prevented or reduced, the

Using the knowledge of the epidemiology of parasitic

summer increase of infective larvae is of such propor-

gastroenteritis that had then become available, Michel

tions as to cause a negligible risk of severe parasitism.

pointed out that if susceptible cattle were removed

Suppression is carried out by two different methods: (i)

from infected to uninfected grazing just before the

repeated anthelmintic treatments with standard prepa-

summer increase in infective larval numbers, serious

rations, the interval between which is determined by the

parasitism could be avoided. When combined with

pharmacokinetic properties of the chemical used; cur-

anthelmintic treatment at the time of movement the

rently most of the anthelmintics used are avermectins

system reduces the level of nematode worm egg con-

or moxidectin: or (ii) the use of a device, usually an

tamination on the clean grazing. In essence, therefore,

intraruminal bolus, which has a continuous slow release

the system involves taking no prophylactic action until

of the active anthelmintic for periods from 60 to 140 early July. At that time, pasture which has not been pre-days. Such boluses are usually administered by balling-viously used for cattle grazing that year is available, after grass has been taken for silage making or some other form of conservation.

The calves are given anthelmintic treatment before being moved to it. The timing of the treatment can vary with the pharmacokinetics of the anthelmintic used and its effect on nematode eggs if contamination of the aftergrass is to be avoided. For instance, the longer acting benzimidazole anthelmintics (e.g. oxfendazole, albendazole, fenbendazole) are ovicidal within a few hours after treatment and their slow rate of excretion means that cattle are protected from infection with susceptible parasites for approximately 30 hours after treatment. They can therefore be put back safely onto the contaminated grazing for 24 hours after treatment before transfer to the aftergrass. The same technique can be applied with avermectins and moxidectin, which

Fig. 19.2

Rationale for anthelmintic suppression.

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gun at or just before susceptible calves are put out to sufficiently large to cause disease. In general, calves grass in the spring, although recent experiments have at grass from midsummer to autumn are those most shown that they can also be effective when given in frequently clinically affected, but heavy infections in midsummer to prevent infection in calves grazing pre-animals of any age previously uninfected will produce viously contaminated fields. The length of prophylactic signs. It is most prevalent in dairy-type calves, but is also activity varies between different climatic areas and common in weaned beef calves. The range of signs can nematode species, e.g. in south-eastern areas of the UK vary from occasional coughing to severe respiratory dis-suppression for eight weeks after being put to pasture tress, and is a reflection of the number of infective in spring may be sufficient, but in wetter western areas larvae ingested during a relatively short period. it may require to be prolonged for a further five weeks

and if lungworm infections are present on the farm for

Aetiology and epidemiology

up to 15 weeks.

The immediate cause of clinical symptoms is the inges-

Dilution strategies: Originally described in New

tion two to four weeks previously of large numbers of

Zealand as the Ruakura method (McMeekin, 1954) and

*infective larvae of *D. viviparus* by non-immune cattle.*

further amended by Leaver (1970), the methods consist

Experimentally, severe infections can be induced by a

of the grazing of paddocks in relays by groups of calves

single administration of larvae at a rate of between 25

followed by groups of older cattle. The basis of the tech-

and 50 larvae/kg body weight. Typically, an affected calf

nique is that the greater consumption of infective larvae

weighing 300 kg will have ingested between 7500 and

by the immune or partially immune adults will delay

15 000 L3 (third stage larvae). This is a much smaller

and reduce the build-up of infective larvae on the pad-

infection than is required to cause parasitic gastroen-

docks. Initially, no anthelmintic treatments were given,

teritis by *O. ostertagi*, and is an indication of lesser but in modern practice the method is frequently com-margin for error involved in ensuring adequate

bined with either of the evasive or suppressive

prophylaxis.

techniques.

The epidemiology is more complex and infections are

The advantages and disadvantages of each of the

also much less predictable than are those of gastroin-

three preventive methods are summarized in Table 19.2.

testinal nematodes, principally because at present not

all details of larval survival and transmission are known.

Infections are more prevalent in wetter areas, especially

Parasitic bronchitis (husk, hoose)

those in the west of the British Isles. One of the major

Dictyocaulus viviparus is almost the sole cause of

differences between *D. viviparus* and gastrointestinal severe pulmonary helminth infections in cattle. Occa-nematodes that has an influence on the unpredictabil-

sionally other parasites are found, but rarely in numbers

ity of infections is that the female worm produces eggs

Table 19.2

Advantages and disadvantages of the prophylactic methods for gastrointestinal parasites.

Method

Advantages

Disadvantages

Evasive

Low labour cost

Does not protect against

Low anthelmintic cost

lungworm

Not likely to lead to anthelmintic

Reduces the flexibility of pasture

resistance in nematodes

usage in the late summer

Suppressive

If carried out for long enough

Higher labour or anthelmintic

will reduce the chances of

costs especially in the case of

lungworm infection

slow-release boluses

More likely to lead to

Allows more flexible use of

anthelmintic resistance in

grazing

target nematodes

Dilution

Lower anthelmintic costs

Requires excellent fencing and

high labour costs and is generally

used only in well organized dairy

farms

Is not effective for prevention of

lungworm infection

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containing fully developed larvae, which are passed in

Necropsy

the faeces. These become infective within a much

The pathological progression during maturation pro-

*shorter time than the eggs of *Ostertagia* species and*

gresses from alveolitis and bronchiolitis to bronchitis

hence in optimal conditions can produce a rapid

and severe emphysema, sometimes with superim-

increase in their numbers. In addition to rapidity of

position of a secondary bacterial pneumonia. If left maturation to infectivity, larvae are also dispersed from untreated after patency even moderate infections can faecal pats by some of the following means: (i) ascending progress in severity due to aspiration of eggs and ingesting the common faecal fungus *Pilobolus* and being pro-hatched larvae back into previously unaffected alveoli expelled into the air on discharge of the sporangium, and smaller bronchioles, resulting in bronchitis and to be carried by wind to adjacent areas; (ii) although pneumonia.

unverified, there is evidence that earthworms or coprophagous beetles may act as transport hosts; (iii) also unverified, it has been reported that the European
Diagnosis

hare (*Lepus europaeus*) can be infected, although experiments have shown that the smaller blue hare (*L. timidus*), common in Ireland, is refractory to infection. Affected cattle invariably present with varying degrees of respiratory abnormality, and since similar signs can In addition to these means of dispersal it has been

occur as a result of infection by a variety of pathogens, shown that infective larvae can remain viable in soil as careful differential diagnosis is advisable. The grazing well as on pasture over the winter, and that small history and time of the year in northern temperate numbers of adult worms and hypobiotic larvae can areas invariably involve cattle grazing established survive and overwinter in infected animals despite their pasture previously used by other cattle plus the appearance of clinical signs between July and October. Since to mature and propagate larvae during the following the caudal lobes of the lung are more frequently spring.

affected, adventitious lung sounds are usually more apparent from these areas, in contrast to those from viral pneumonias, which usually affect the cranial and

Pathogenesis

medial lobes. Confirmation of the diagnosis by identification of larvae in faeces or sputum samples can only

mucosa, moult to the L4 stage of their life cycle and take place after patency, which takes place approximately 25 days after infection, although it should be noted that in adults that are suffering from a re-infection the infection usually does not reach patency. are observed in the host. After that period, larvae break out of blood vessels into alveoli and small bronchioles,

Treatment (see p. 1027)

after which they moult to become young adults, and as they increase in size they ascend the bronchiolar tree In order to kill the worms and larvae present in the lung towards the large bronchioles and bronchi. Larvae are tissues, anthelmintic treatment is essential. The most first found in faeces from the 25th day after infection. effective are the avermectins and moxidectin; levamisole and some of the more recently developed benzimidazoles or pro-benzimidazoles, such as

Signs

oxfendazole, fenbendazole, albendazole and netobimin, Clinical signs appear during the second week after are also effective, but the incidence of alveolar epithelial infection and their severity depends on the number of lialization is more common after their use, since they developing worms. The signs range from occasional to have little persistent efficacy and reinfection can occur repeated coughing, with a noticeably increased respiration rate in the worst affected. By the third week, Severely affected animals may also require antibiotic severely affected cattle do little else except stand in therapy to control secondary bacterial pneumonia, and a characteristic head extended position with rapid if anorexia has been present rehydration with electrolytes may also be helpful. In most outbreaks a range Although most calves, if not severely affected, will of severity is observed and it is frequently advisable to recover after treatment, a small percentage will suffer

house the worst affected cattle, especially if climatic a relapse of clinical signs in the absence of fresh infections would be stressful even to healthy calves. In addition, because treatment of severe cases can exacerbate signs farmers should be warned of the possibility. autopsies reveal an oedematous rubbery lung with alveolar epithelialization observed microscopically.

In outbreaks involving adult dairy cows care should be

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taken that the anthelmintic used does not prevent sale time of turn-out to grass, and intraruminal boluses of milk products for a prolonged period until a withdrawal designed to release anthelmintic either intermittently or holding time long enough to allow excretion of the drug continuously for periods up to 140 days. The level of immunity stimulated depends on the number of larvae ingested from pasture and, since this can be highly variable is eprinomectin, the use of which requires no period of milk withdrawal from sale.

able, some cattle in a group may have inadequate resistance to future infection should they be exposed to it. As a result, careful monitoring is necessary after ces-

Prevention

sation of anthelmintic treatment and some thought

There are two major methods available, the first relying required to plan cattle husbandry during the subsequent on inducing immunity and the second on suppression year and then as adults.

of infections.

Stephanurosis (kidney worm)

Vaccination (see p. 1011): Immunity can be stimulated

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by the use of a live vaccine, which takes the form of two doses of 1000 infective larvae irradiated by gamma irra-

This is mainly an infestation of pigs but it can infect

diation. Calves should be two months old before vacci-

calves and is due to *Stephanurus dentatus*. The adult is nation and the doses separated by four weeks and

up to 45 mm in length.

preferably both doses given before going to grass in

The condition is found in the tropical and subtropical spring or before the time of earliest challenge in cal countries of Africa, East and West Indies, Brazil, outdoor calves. Although the vaccine induces excellent Hawaii, Philippines, Australia and southern Europe protection against clinical disease it does not completely prevent all worms from natural infections where pigs are kept, but in calves adult worms do not develop. The larvae cause damage to the liver completing their life cycle, so that the parasite can be maintained at a very low level on pasture grazed by vaccinated cattle. If the farmer neglects to vaccinate, aberrant larvae can encapsulate anywhere in the host site numbers can quickly increase sufficiently, if weather conditions are favourable, to affect non-immune cattle eggs produced in the pig form hatch to produce first stage larvae. These develop to third stage infective

larvae if there is warmth and moisture. The larvae are

Suppression: On farms where calves have suffered lung often ingested by earthworms and these act as a vector damage due to viral pneumonia, vaccination may be for infection. The infective larvae are otherwise susceptible to cold or drying. Transmission is either by of the existing lesions. Under these circumstances it ingestion, which may include earthworms, or by skin has been found that regular anthelmintic treatments penetration. In the host they pass to the liver where throughout the grazing season can suppress infection they remain for a considerable time before going to sufficiently to minimize the danger of clinical disease other parts of the body.

and at the same time allow some immunity to be induced by the natural ingestion of infective larvae from

Signs

pasture which are subsequently killed before completion of their life cycle (see p. 1027). The method has also

They are mostly of anaemia, ill thrift and ascites. Many

been successfully applied to normal healthy calves, but infestations are without signs.

it requires to be carried out and monitored carefully, since there are potential problems in its use. Firstly, the Necropsy

length of time during which anthelmintic suppression is Necrotic lesions with thrombosis in the mesenteric required may vary between areas with different climatic blood vessels and hypertrophy of the mesenteric lymph patterns and farming practices. In general in the British nodes are likely to be found. In a few cases there may Isles it is necessary to provide anthelmintic cover until be haemorrhages and abscesses in the lungs and mid August. The methods of application require that the kidneys.

anthelmintic must be extremely effective against all stages of the parasitic life cycle of the nematode. Those Diagnosis

anthelmintics used fairly successfully have been avermectins, either as injections or pour-ons administered at This will depend on the presence of calves grazing areas

intervals after going to grass that are dependent on the with infected pigs. An immunodiffusion test can be particular pharmacokinetics of the drug used and the used. There is a marked eosinophilia.

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Treatment and control

suck blood and cause haemorrhagic anaemia and hypoalbuminaemia, and small numbers (about 2000) in Fenbendazole at high doses may be effective. Calves comparison to other gastrointestinal nematodes, other should not graze pastures recently used by pigs. than Haemonchus placei, will cause very severe disease and death.

Bunostomosis

Bunostomosis in cattle is caused by the hookworm

Diagnosis

Bunostomum phlebotomum. It mainly affects cattle up to one year old and causes a range of signs from unrest It is difficult to diagnose when prepatent and mild and mild abdominal pain to diarrhoea and eventually anaemia is the main sign. When patent it should be dif- anaemia depending on the stage of the infection and the

ferentiated from other parasites that cause haemorrhagic anaemia, e.g. *H. placei* and *F. hepatica*, and those tropical and subtropical regions than in temperate; this

deficiencies such as cobalt or copper (Chapter 21) that distribution has become especially marked during the adversely affect erythropoiesis.

last decade as the worm, which does not occur in large numbers, seems especially sensitive to some of the

Treatment and control

modern anthelmintics and may now be absent from those areas where frequent suppressive anthelmintic

The parasite is sensitive to modern anthelmintics and treatments have been applied.

can be controlled by their suppressive or strategic use.

Other measures in housed cattle should be the provision of dry bedding and its replacement when soiled.

Aetiology and epidemiology

Once infected and treated, cattle normally develop a

Bunostomum phlebotomum parasitizes the small intestine. strong immunity to the parasite.

tine of cattle. Adults can reach 3.0 cm in length and are characterized by a large buccal capsule with lateral

Haemonchosis

cutting teeth and a tooth-like structure in its base, which is the duct of an oesophageal gland. The worms suck

Haemonchosis is manifested by haemorrhagic anaemia blood, which can induce anaemia and in large numbers and diarrhoea, and is caused by the presence in the abo-cause diarrhoea.

masum of *Haemonchus placei* (synonym *H. similis*). As The eggs from adult females pass out with faeces.

with bunostomosis it is mainly pathogenic in immature

Depending on climatic conditions, a parasitic infective cattle, since infection in the first three years of life

larva, which is susceptible to desiccation, will develop

usually produces a strong immunity to reinfection. It

under optimal conditions of adequate moisture and

also has a similar distribution, being commonest in sub-

warmth within one week. The infective larvae can infect

tropical and tropical countries, but present in most tem-

susceptible cattle in either of two ways.

perate countries and able to cause disease during any unusually warm periods of weather.

- By penetration of the skin after which it enters the bloodstream and passes to the lung, emerging into the alveoli and after further moulting up the

Aetiology and epidemiology

bronchial tree to the pharynx, where it is then swal-

Adults of *H. placei* suck blood from the surface of the lung and passes to the intestine to mature.

abomasum. They are fairly large in comparison to other

- By direct ingestion after which it burrows into the abomasal nematodes and measure 3 cm in length when intestinal wall during development to emerge as an fully grown. In the female the ovaries and intestine are adult.

spirally intertwined to give the worm its 'barber-pole'

The prepatent period is eight weeks. The worm flourishes in conditions of adequate moisture and warmth and thus is generally a subtropical problem, but disease

capsule.

has been recorded in countries as far north as Scotland, Female H. placei are very prolific egg producers and where it has been seen in cattle maintained indoors on can lay up to 10 000 eggs daily. The eggs are passed in damp straw bedding.

faeces and undergo a typical trichostrongyline development to infective larvae. The time taken to reach this stage varies considerably between temperate and sub-

Signs

tropical climatic areas. In the former, it may take Heavy percutaneous larval infections cause restless-several weeks in contrast to four days under suitability, stamping and itching. Once in the intestine adults ble conditions. The conditions for development of

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Haemonchus spp. have been studied in some detail, and Treatment and control it has been concluded that they require a temperature Anthelmintic treatment is the primary consideration in excess of 18°C and rainfall of 5.3 cm/month for and H. placei is susceptible to levamisole, avermectins, maximum translation,

but can develop provided the
moxidectin and modern benzimidazoles as well as the
mean minimum temperature is not less than 10°C. As a
flukicides rafoxanide, clorsulon and nitroxynil. This
result of the combination of fecundity and rapid infec-
should be coupled with movement to uninfected
tive development in favourable conditions, pasture can
pasture. Prevention can be achieved using similar
become quickly contaminated with huge numbers of
methods as those described for ostertagiosis.

infective larvae. In the subtropics acute infections can
therefore be found in susceptible young cattle, whereas
in temperate areas infections are more likely to be

Trematodes

chronic. Under adverse conditions in the tropics, e.g.
dry seasons, infective larvae can undergo hypobiosis in

Fasciolosis

a similar manner to *Ostertagia* spp., and resume development shortly before the
rainy season commences.

Fasciolosis in cattle is a chronic wasting disease caused

Once ingested, larvae undergo two moults before

by the presence in the liver and bile ducts respectively becoming adult in 26–28 days, when infections become of immature and adult trematodes of the genus *Fasci-*
patent. After the first moult, the L4 larvae and subse-
 ola. The disease is found in vast areas of the world, with quent adults suck blood, leaving haemorrhagic spots on
 the smaller *F. hepatica* (3.5 ¥ 1 cm) in temperate counthe abomasal epithelium, and the resultant blood loss is
 tries and the larger *F. gigantica* (7.5 ¥ 1 cm) in tropical the major pathogenic sequelae. However, in large infec-regions the commonest species. Calves and yearlings
 tions the pH of the abomasal contents may be increased
 are most commonly affected but any age of animal may
 in the same way as in ostertagiosis, with the resultant
 be susceptible. Although it may take place at any time
 digestive disturbance, raised plasma pepsinogen, possi-
 of the year, infection is most prevalent during autumn
 ble bacterial overgrowth and diarrhoea. Once patent,
 in temperate areas, with the resultant effects of disease
 peak egg production from females is normally reached
 becoming apparent in winter and spring.
 between six and seven weeks after infection and may
 continue for a further six to eight weeks. Cattle, unlike

Aetiology and epidemiology

sheep, develop resistance to the worm and unless

immunologically compromised by intercurrent illness

F. hepatica: Multiplication of the parasite is partially or malnutrition the population decreases rapidly there-climatically regulated, since its life cycle involves an

after and the animal remains fairly resistant for the

intermediate host that requires adequate moisture and

remainder of its life.

a suitable ambient temperature. The intermediate hosts

are various species of snail of the genus Lymnaea, which are found on wet mud surfaces. In the British Isles, L.

Signs

truncatula is the species involved. It prefers a neutral or There are two types of clinical picture. The first occurs

slightly alkaline habitat, but can survive in fairly acid

in severe primary infections and presents severely

conditions such as peaty hills, although under such con-

anaemic, weak and sometimes diarrhoeic young cattle.

ditions individual snails remain small and the popula-

The second is found in chronic lesser infections and typ-

tion low. At its maximum it can have a shell length of

ically causes weakness, lethargy, weight loss, a less acute

1 cm. The snail population fluctuates, being least in anaemia, hypoproteinaemia, submandibular oedema winter and greatest in June and July, especially in years and anasarca.

when late spring rainfall has been above average, when large numbers accumulate and are therefore available for transmission of *F. hepatica*.

Diagnosis

Briefly summarized the life cycle of *F. hepatica* is as Diagnosis can be confirmed if the infection is patent by

follows. The adult parasite in the bile ducts is a hermaphrodite and can be self- or cross-fertilized. Large numbers of eggs are produced by each parasite, which level may be raised. The grazing and past parasitic pass from the ducts to the gall-bladder and are subsequently passed into the intestine on contraction of that causes of anaemia or diarrhoea such as fasciolosis. Eggs are then passed in the faeces, but will not

sis, bunostomosis, copper or cobalt deficiencies,
hatch unless moist and until an ambient temperature of
babesiosis, coccidiosis, bacillary haemoglobinuria and
10°C is reached. Hatching at temperatures between
malnutrition.

22 and 26°C takes nine days, but under normal

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temperatures in the months of May and June in the

F. gigantica: Although similar in many respects to *F.*

hepatica it is not found in western Europe, but is wide-released miracidium must

locate and penetrate the foot

spread within tropical and subtropical climatic regions.

of *L. truncatula* within three hours. Once in the snail, The intermediate hosts are
also snails of the genus

the parasite undergoes multiplication through stages of

Lymnaea, but the species are more aquatic than those

sporocyst and rediae and reaches the cercarial stage

transmitting *F. hepatica* and thrive in swamps, drainage after a minimum of two
months, although in colder tem-channels and artificially flooded areas. As a
result the

peratures it may take up to 16 weeks or should winter

epidemiology is closely tied to the periods of maximum

intervene complete development may cease until the rainfall, the snails being infected and development in following spring. Under natural conditions within the the snail completed within these periods. Cercariae are British Isles, cercariae from summer infections are shed towards the end of the wet season and encyst on released from snails between late August and October, herbage and even on the water surface at the start of and in some areas from overwintered infections in the dry season.

April or May. On release from the snail, which must

The parasitic life cycle is similar to that of *F. hepatica* take place in conditions of wetness, the cercariae

but each phase takes slightly longer with the result that

swim to the nearest plant, encyst on its leaves and

patency of primary infections take three or four weeks

become the infective stage, termed metacercariae,

longer than *F. hepatica*, eggs being found in faeces from several hundred of which can arise from one miracid-13 to 16 weeks after ingestion of metacercariae.

ium. Metacercariae on herbage can survive for several

months and therefore maintain the infection from one

year to the next.

Signs

Cattle become infected by ingestion of grass on which metacercariae are encysted. The metacercariae In severe infections there is a progressive loss of weight excyst in the small intestine, the wall of which they with anaemia and occasionally oedema. Subclinical penetrate to reach the peritoneal cavity where they infections cause a reduction in milk yield and reduction move to the liver and invade its capsule. At this point in the growth rate of young cattle.

the flukes are very small (about 1 mm) but as they burrow through the liver parenchyma they gradually
Diagnosis: The disease is characterized by a gradual increase in size and in primary infections in cattle will loss of condition that is exacerbated if the quantity and reach the bile ducts after six to eight weeks, when they quality of feeding are suboptimal. A chronic anaemia mature and become egg-laying adults 10–12 weeks after develops and the packed cell volume (PCV) drops to ingestion. In response to the presence of fluke in the

approximately 20 per cent.

Erythrocytes are

liver parenchyma, the liver of cattle produces a more hypochromic but normocytic, and while the leucocyte intense fibrous reaction than that of sheep, and the count may be slightly raised, the percentage of resultant cirrhosis is much more severe and is longer eosinophils in a differential count is greatly increased lasting. The liver cells of cattle do not regenerate as do and may consist of up to 20 per cent of the total white those of sheep, and the result of even moderate cell count. In cows the total milk yield and the non-fat infection in cattle is a more fibrous liver parenchyma solids proportions may be reduced. than in most other species.

Diagnosis can be confirmed by the presence of fluke

Within the bile ducts, the adult flukes suck blood eggs in faeces samples, but detection is not always as and the spines on their cuticle cause multiple small simple as in sheep, especially in adults, since even in haemorrhagic lesions. In cattle, the resultant fibrous

patent infections excretion of eggs is intermittent.

reaction within the bile ducts is also more severe than

Examination of plasma shows a hypoalbuminaemia,

in sheep and the bile duct walls thicken and become

and during the migration phase the plasma glutamate

gradually calcified thereafter (Fig. 19.3). The result of

dehydrogenase (GLDH) is raised. Once the epithelium

the fibrous reaction during a primary infection in both

of the bile duct walls becomes diseased, the plasma

bile ducts and parenchyma of cattle shortens the life

concentration of gamma-glutamyl transpeptidase (GT)

span of adult flukes in the ducts to a maximum of 1.5

risks and this is a useful diagnostic indicator.

years and reduces the chances of flukes from subse-

quent infections being able to complete their life cycle.

Cattle therefore become largely resistant to further

Treatment (see pp. 1026, 1028)

infections, most of which do not become patent, and the

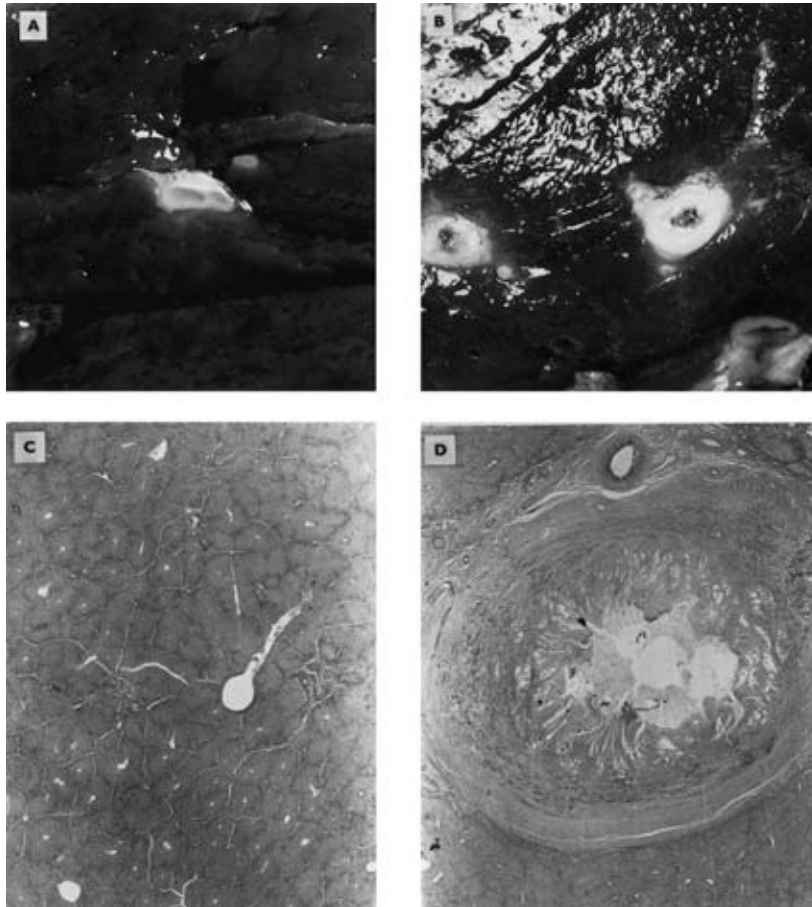
few flukes that do so may take much longer than normal

Affected cattle are treated by administration of a

to reach patency and survive in bile ducts for a rela-

fasciolicide. Of those currently used for cattle the comparatively short time.

monest are triclabendazole, oxyclozanide, rafoxanide,



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Fig. 19.3

*(A) Normal and (B) liver fluke
infected bovine liver. (C, D) Microscopic
sections of the same stained to show
fibrous tissue.*

nitroxynil, albendazole, netobimin and clorsulon. All in an attempt to give a more rapid reduction in the will remove more than 90 per cent of adult flukes from prevalence of the parasite.

the bile ducts, but they have variable efficiencies against

- Prophylactic use of anthelmintics to reduce both the immature stages migrating through the liver. The the number of flukes in cattle and the number of most effective is triclabendazole, which will remove eggs excreted. This is by far the commonest practice and involves treatment of cattle once or twice Rafoxanide, nitroxynil and clorsulon are effective annually. In the British Isles such treatments take against six-week-old flukes at normal dose rates and at place in the autumn and late spring.*

increased dose levels affect those four weeks old.

- If the areas of snail habitat are delineated and small Albendazole, netobimin and oxyclozanide at normal and they can be drained. This is usually not cost-dose rates remove only adult flukes from the bile*

effective for fluke control alone if the sites are ducts and are ineffective against immature flukes. All extensive.

require withholding of meat and milk for human con-

- Small snail habitats can be fenced off during the sumption for variable periods dependent on their time of greatest infection.

pharmacokinetics (see p. 1026).

- Snail habitats can be treated with molluscicide.

There can be environmental objections since many which were formerly in use were toxic to many

Prevention

species in addition to snails. The most satisfactory

There are four methods available that are used individually in suitable circumstances or can be combined in the UK is used in many tropical countries.

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Paramphistomosis (stomach fluke

Diagnosis

disease, intestinal amphistomiosis)

Most disease occurs with immature flukes and so it is

Aetiology and epidemiology

not easy to detect in the live animal but it can be missed

at post mortem. Often other parasitic infestations are

The cause of acute paramphistomosis in Africa is

present, which again causes paramphistomosis to be

Paramphistomum microbothrium. Other Paramphisto-

overlooked. However, the history and area where the

mum spp. and Cotylophoron spp. are found in rumi-cattle are grazing can give some indications. There is a

nants in Africa and tropical and subtropical regions.

reduction in the red and white blood cells and a hypoal-

In Asia, Calicophoron spp. are found and one species

buminaemia. Differential diagnoses include copper and

occurs in Africa. Carmyerius spp. are seen in Africa, cobalt deficiencies, liver fluke, parasitic gastroenteritis,

India, Pakistan and the Middle East.

Johne's disease in the adult and poisoning.

It has been recorded in a severe form in Australia,

India and the USA. The life cycle is indirect and similar

to that of Fasciola. It involves aquatic snails that are Treatment (see p. 1028)

adapted to a variety of locations. Disease occurs only

Few of the modern trematocides have been found to be where there is a massive concentration of the infected effective against paramphistomosis. Oxclozanide at planorbid snails. The metacercariae are ingested and 18.7 mg/kg has variable efficacy from approximately 50 the immature flukes develop in the duodenum, where to 90 per cent. Niclosamide at 100 mg/kg is highly effective they may be seen in the mucosa. The flukes are 3–4 mm against intestinal paramphistomes but ineffective long and 1–2 mm wide. They also develop in the abo- for adults. Brotianide at 55 mg/kg is effective against masum but are less common. They then migrate adult flukes but less so against the immature stages. through the abomasum to the rumen and reticulum, where again few are found. The prepatent period is very variable, being between one and a half to four months.

Control

Most outbreaks occur in the late summer, autumn or Drainage of low-lying areas and destruction of the soil early winter when pastures are being contaminated will considerably reduce infestation. The most suscep-

with cercariae. Although all ages of cattle are susceptible animals, i.e. calves and immature cattle, should be kept away from likely snail habitats. Routine treatment against *Paramphistomum* before they mature and can infect snails is of use.

Fewer infestations occur in the adult, suggesting some degree of immunity.

Signs

Schistosomosis (bilharziosis)

Acute paramphistomosis is due to a massive infestation

Aetiology and epidemiology

in calves. There is a persistent fetid diarrhoea without

There are several parasites in the genus *Schistosoma*.

blood or mucus, anorexia, weakness, marked loss of

Schistosoma bovis is found in African, Asian and south-conditions and then recumbency. In some cases there is

in European cattle. *Schistosoma mattheei* occurs in

submaxillary oedema and a pallor of the mucosa. Death

cattle in central and southern Africa and can infect

occurs in about one to two weeks after signs develop.

man. *Schistosoma indicum* is found in India and

Pakistan as is also *S. spindale*, which can also occur in Chronic: In heavy infestations of adult flukes there is a south-east Asia and Indonesia. *Schistosoma japonicum*

chronic loss of weight, a dry coat and anaemia.

occurs in the Far East. *Schistosoma nasale* is again found in India and Pakistan and is unusual in that the adult occurs in the mucosal veins of the nose. Several Necropsy

minor species occur involving *S. curassoni*.

In the acute case there is severe inflammation of the Various species of the parasite are widespread and duodenum and abomasum as the parasite penetrates the adults live in veins. Unlike most flukes, *Schistosoma* deeply into the mucosa and sometimes the muscularis spp. are not hermaphroditic and the longer male carries layer, and there is haemorrhage. There is muscular the long slender female, which is up to 25 mm long, in atrophy, subcutaneous oedema and fluid accumulation a gyroectoplasmic canal. Infection can be via the skin in the body cavities. Large numbers of small, immature, or gut. After penetrating the skin the schistosomulae

flesh-coloured flukes are present in the lumen. In less (young flukes) pass via the lymphatic system to the right acute cases the duodenum is thickened and congested. side of the heart. They then go via the lungs into the cir- There is fluid in the abdominal cavity. culation and then to the liver. They mature in the portal

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Table 19.3

Intermediate snail hosts of Schistosoma spp.

cally spiral. As the eggs hatch quickly they need to be fixed in 10 per cent formalin.

Species

Intermediate snail host genus

Differential diagnosis involves coccidiosis, trypanosomosis, poisoning and Johne's disease.

S. bovis

Bulinus and Physopsis

S. indicum

Indoplanorbis

S. japonicum

Oncomelonia

Treatment (see p. 1028)

S. mattheei

Bulinus

Trichlorophan, an organophosphorus compound, can

S. nasale

Lymnaea and Indoplanorbis

be used parenterally, or niridazole or thioxanthane derivatives such as becanthane or hycanthane may be used. Treatment must be performed carefully otherwise a large number of dead schistosomes will produce veins before migrating to their final area, usually the emboli and lead to hepatic infarction or portal mesenteric veins, but they are also found in the urinary occlusion.

bladder veins in the case of S. mattheei and in the nasal mucosal veins in S. nasale. When infection is oral the Control

gut is penetrated and the immature stages pass to the liver via the blood.

Immunity to the disease occurs but varies with the

Once mature, eggs are produced that can hatch

Schistosoma species. Control involves keeping cattle within minutes if conditions are right. The free-away from areas frequented by the intermediate host.

swimming miracidiae penetrate snail hosts producing Molluscicides can be of some benefit. Some vaccines sporocysts, daughter sporocyst and cercariae but there are under development, e.g. a recombinant glutathione are no redia. The cercariae must infect the final host *S-transferrase* derived from *Schistosoma bovis* has been within one or two days or they die off as there is no shown to reduce both egg output and parasitic establishment. There are various genera of snails involved depending on the species of *Schistosoma* in the next year or two. (Table 19.3). All the snails like mud except for *Oncomelania* spp., which are aquatic.

Cestodes

In many areas of Africa most of the cattle are infected but there are very few signs. Young cattle are

Taenia saginata

more likely to be ill.

Taenia saginata is a cyclophyllidean tapeworm, for which Signs man is the final host. It is transmitted to man by the

ingestion of the intermediate larval stage in under-

The main signs are of diarrhoea, which may contain cooked beef. Although the larval stage can be found in blood and mucus and is intermittent. There is a loss of any age of cattle, viable cysts are found mainly in cattle condition and reduced resistance to other diseases.

up to two years of age. The presence of the parasite in the

*Death occasionally occurs. Infestation with *S. nasale* muscles of cattle does not cause clinical signs unless the causes a mucopurulent nasal discharge with dyspnoea.*

animal is very heavily infected and the myocardium is

Chronic cases develop nasal granuloma.

involved, but because it is a zoonotic infection its presence can cause significant losses to the beef industry,

Necropsy

especially in developing countries. In countries where sanitation standards are high it is a decreasing problem

In severe infestation there are small grey-white

that on average is found in 0.5 per cent of bovine car-

granules in the gastrointestinal tract with petechial

casses, with occasional heavy infections occurring under

haemorrhages and ecchymosis. The ileocaecal body unusual circumstances. In those areas of Africa, Asia may be enlarged and haemorrhagic. Granulomas may and parts of Latin America where human sanitation also be scattered through the liver parenchyma as well methods are poor or non-existent and local customs as the lungs and bladder. In *S. nasale* infestation there favour ingestion of raw or undercooked beef, the prevalence may be nasal granulomas. lence may be up to 20 per cent.

Diagnosis

Epidemiology

The signs and grazing history help but it is difficult to

The adult tapeworm inhabits the intestines of man. Its diagnose unless faeces are taken or nasal discharge if *S.*

scolex has no hooks, and apparently moves up or down

nasale is present. The eggs are large and characteristic-the intestine depending on whether the host has eaten

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recently or not. When the host's stomach is empty the

- If lightly infected they are frozen to a temperature

scolex moves back up the intestine towards the duode-

of -10° for a minimum of 10 days, after which any

num, and these movements produce hunger pains and previously viable cysticerci will have been killed.

vague intestinal discomfort. Fully grown the tapeworm

- Heavily infected carcasses are usually condemned may reach a length of 8 m. Mature egg-filled (about as unfit for human consumption and rendered after 25 000) proglottids are shed from its posterior end and heat treatment into animal feedstuffs in some countries. The thermal death-point of cysticerci is 57°C nocturnally. Anal pruritis and occasional diarrhoea are usually the only signs apart from the aesthetic and to at least that temperature if the possibility of hygienic conditions involved.

infection is to be avoided.

The eggs are resistant to external conditions. They are inactivated within two weeks if desiccated, but are known to survive for 16 days in raw sewage, 71 days in

Echinococcus granulosus

liquid manure, 159 days on pasture and 200 days in

and hydatid cysts

sewage sludge. No normal method of handling sewage

There are two species of the taeniid tapeworm E. gran-

can inactivate them other than total incineration or

ulosus. The first is E. granulosus granulosus: the adult extensive heat treatment. Extended periods of sludge

is found worldwide in the intestine of dogs and many

digestion with or without heat may affect viability but

wild Canidae but not the red fox. The intermediate

technical proof is lacking. Chemical disinfectants other

form, the hydatid cyst, is found in domestic and wild

than some containing copper at very high pH (11.5) are

ruminants, man and pigs, but not horses and donkeys.

not ovicidal.

The second is E. granulosus equinus, which is almost

Two-thirds of outbreaks in developed countries have

exclusively found in Europe: adults parasitize the dog

been shown to be associated with the spreading of

and the red fox, and horses and donkeys are the exclu-

sewage sludge on agricultural land. The remainder have

sive intermediate hosts.

been presumed to be caused by spread of seagulls
Cattle therefore are affected by hydatid cysts of *E.*
feeding at sewage plants and passing viable eggs
granulosus granulosus. The majority of hydatid cysts
through them on to grazing land, or by accidental flood-
cause little apparent disease as they are situated in the
ing of pasture by contaminated effluent streams from
liver or lung, their presence only becoming disclosed
sewage plants. Migrant labour from less developed
at abattoirs. Occasionally, they develop in sites where
countries and infected stockmen have been implicated
their gradually increasing size causes pressure on vital
in some isolated local multiple infections.

organs., the resultant disease being dependent on the
Once ingested, the egg hatches and the liberated
organ and system affected.

oncosphere travels via the bloodstream to striated mus-
cular tissue, including the myocardium. It develops in
the muscle and reaches infectivity three months after

Aetiology and epidemiology

ingestion, when it is recognizable as a whitish fluid-

filled cyst with a diameter of approximately 1 cm,
The adult worms parasitize the small intestine of the
known as a cysticercus. It becomes enclosed in a thin
dog. They are small worms, consisting of four segments
fibrous capsule. Cellular immune reaction around it
of length 0.6 mm. Large numbers may be present in
gradually increases and the capsule becomes thickened,
infected animals, each releasing one gravid segment
eventually leading to the death of a high proportion of
daily containing many eggs, which are resistant to
the cysticerci within 18 months and resistance to further
normal external climatic conditions and remain infec-
infection, although some may survive for several years.
tive for up to two years. After ingestion by cattle or
After death the contents of the cysts become caseous
other hosts the oncosphere penetrates the wall of the
and finally calcified. The masseter, tongue and heart
intestine and is carried via the bloodstream to the liver
muscles were generally considered preferential sites
or other organs where it lodges and starts to grow to
and are still examined carefully during meat inspection

become a hydatid cyst. Growth is relatively slow and at abattoirs, in preference to damaging more expensive cuts of meat by the multiple incisions that would otherwise be necessary. In restricted sites the cyst assumes the shape of the

space available, and daughter cysts may be formed and spread to other organs. If growth takes place in the omentum or mesentery, cysts can become very large.

Control (see p. 1028)

There is usually little cellular reaction around cysts in cattle. Since most cattle in developed countries are slaughtered in abattoirs there is little chance of further treatment in one of two ways:

spread unless casualties are fed to farm dogs or packs

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of hounds. In underdeveloped countries or where carcasses are not disposed of, wild Canidae ingesting the

*occurs three or four weeks after groups of purchased
offal become infected; the brood capsules from within
calves are mixed. In North America it has also been
the hydatid cyst evaginate, the scolex attaches to the
associated with the sudden stress of extreme tempera-
intestinal wall and reaches adulthood and patency
ture reductions in midwinter.*

*some six weeks later. Infection in dogs is usually
After ingestion, the oocyst is disrupted, releasing
symptomless.*

*eight sporozoites each of which penetrates an intestinal
epithelial cell and develops into a trophozoite. Multiple
fission within the trophozoite produces a schizont,*

Treatment and control (see p. 1028)

*which after a few days ruptures the cell to release a
Affected cattle are not treated specifically for hydati-
large number of merozoites, which in turn invade other
dosis, principally because of the difficulty of diagnosis,
epithelial cells. There are up to four generations of
but also because anthelmintics are only now becoming
schizogony depending on the species involved. The*

available for treatment of infected human beings.

location of the cells invaded is important for the

Activity against larval forms has been claimed for

pathogenicity of the species, e.g. *E. zuernii* and *E. bovis* praziquantel and albendazole, although most cysts are

both invade cells in the lower ileum and later schizogony and gametogony (the term used to describe the

Prevention is directed to ensuring that dogs do not

sexual stages) take place in the epithelial cells of the

become infected, either by regular treatment, the most

caecum and colon. The gametocytes are formed deep in

effective anthelmintic being praziquantel, or by pre-

the epithelium and their rupture causes severe disruption

venting the ingestion of infected offal.

tion of the lining of the caecum with resultant haemorrhage

rhage into the lumen. After gametogony, oocysts are

formed that subsequently are passed in faeces. They are

Protozoa

not immediately infective but require to undergo sporulation,

in which the sporocysts and sporozoites are

Coccidiosis

formed ready for further infection. The last process requires adequate moisture and warmth and takes

Bovine coccidiosis almost invariably affects groups of 24–72 hours. The total life cycle from ingestion to cattle less than one year old, although it does occasionally occur in older animals. It is manifested by enteritis, days for E. bovis. Oocyst production during infection diarrhoea and in severe cases dysentery. Not all with a single species lasts for five to twelve days, but members of the group are equally affected and light may be prolonged in multiple species infections. The infections are self-limiting; severe infections can be fatal oocysts are very resistant to external conditions and will if untreated. The disease is caused by the ingestion over survive for up to two years in suitable environments a short period of large numbers of oocysts of Eimeria with adequate moisture. They can resist moderate frosts species by non-immune cattle; the greater the infective down to -8°C for two months, but are susceptible to

dose, the more severe the signs produced. *Eimeria* are drought, high temperatures and the chemical action of

species-specific, and although 13 species have been isolated from cattle, *E. zuernii*, *E. bovis* and *E. alabamensis* are much the commonest and most pathogenic. They are

Signs

found everywhere in the world where cattle are farmed.

Cryptosporidiosis is mentioned on pp. 204 and 286.

The presenting signs of diarrhoea or dysentery accompanied sometimes with severe straining in a group of calves leads to suspicion of coccidiosis.

Aetiology and epidemiology

Diagnosis

The presence of large enough accumulations of oocysts to cause disease is the result of farming practices that Examination of faeces samples for the presence of large allow groups of cattle, which frequently have been numbers of oocysts can confirm the diagnosis. Care stressed by transport and by mixing with others, to should be taken in interpretation of oocyst counts since ingest food or water contaminated by faeces. It can

small numbers are present in the faeces of many normal calves, and in severely affected diarrhoeic calves the main oocyst production phase may have passed or their numbers be misjudged due to the dilution factor of liquid faeces. Occasionally, calves may exhibit nervous signs, the reasons for which are as yet obscure, and viral

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diseases that have cerebral affects should be considered. as a ubiquitous parasite of cattle and dogs that also

affects many other species of animals. Although it is the type species, recently, another species, *N. hughesi*, has been isolated from horses. Currently little is known of

Treatment (see p. 1029)

its development in animals that are naturally infected. Two considerations need to be borne in mind. Firstly,

The dog is the intermediate host, and is also a final host

severely affected calves will require individual treat-

in prenatal infections. Infection can be both vertically

ment with an anticoccidial and, if deemed advisable,

transmitted from dam to calf in utero and lactogenically, fluid therapy with electrolytes and injections of anti-naturally by ingestion of food and water contaminated

biotics to control secondary bacterial infections. Antic-

*with dog faeces containing *Neospora caninum* oocysts,*

occidial treatments include injections of sulpha drugs or

or from cow to cow. The mechanisms of repeat con-

the oral administration of sulphadimidine at 140 mg/kg

genital transmission are unknown at present.

body weight daily for three days, or amprolium at

10 mg/kg for four or five days. Amprolium has been

Signs

withdrawn from sale in many countries because of pos-

sible carcinogenesis, and it should be remembered that

It is a major cause of abortion in both dairy and beef

overdose with amprolium, which is a thiamine antago-

cattle, and has been observed in almost all countries

nist, has also been associated with production of cere-

in the world. Cows of any age can abort from three
brocortical necrosis (CCN), so care should be taken to
months of gestation to full term, although most abor-
give the correct dose in those areas where it is available.

tions occur at 5–6 months. Foetuses can be born alive

Secondly, prophylactic treatment for other calves in
or may die in utero and be mummified or reabsorbed.

the group should be assessed, depending on the

Calves that are infected may be born underweight and
numbers involved. Lasalocid at 1 mg/kg body weight
with neurologic symptoms such as ataxia, decreased
per day, monensin included in the diet at 16.5 ppm of
reflexes and exophthalmia.

the diet or decoquinate at 100 ppm, either continuously
to suppress all infection or for two cycles of medication
for a week separated by a week when it is removed, can

Necropsy

protect from the infection and allow sufficient immu-

Tachyzoites and tissue cysts are found intracellularly in
nity to be stimulated. A long-acting intraruminal bolus
affected cattle. Tachyzoites measure 6 × 2 microns.

consisting of 1.6 g baquiloprim plus 14.4 g sulphadimi-

Tissue cysts are oval, 102 microns long, and are found
dine is also effective, and sulphadimidine itself can also
in the central nervous system and retina. Although
be used prophylactically at 35 mg/kg, but it should be
infection can be found in many organs, the commonest
borne in mind that as light infections are self-limiting
site is the brain. The characteristic lesion is a focal
such treatments may not be cost-effective.

encephalitis with necrosis and non-suppurative

Other measures should be taken to reduce the
inflammation. Hepatitis can also be found in epidemic
chances of infection, i.e. removal of food contaminated
abortions.

with faeces and better siting of feeding and water
troughs so that faecal deposition on food and in water
is made less likely. If the problem is a recurring event

Diagnosis

with each batch of calves, thought should be given to a
Ante-mortem diagnosis is by serologic methods.

short period of anticoccidial treatment after two weeks

Several methods of assay of serum antibody are available, e.g. ELISA and indirect fluorescent antibody. A PCR (polymerase chain reaction) diagnosis of antigens immunity has been allowed to take place. A potent is under development.

anticoccidial of a novel chemical type, toltrazuril, has been used experimentally in this manner with great success and may constitute a significant future advance

Treatment and control

in the prophylaxis of the disease.

At present treatments are largely experimental.

Sulphonamides, pyrimethamine and clindamycin have

Neosporosis (see p. 584)

been used in infected dogs, and decoquinate has been

shown to kill N. caninum tachyzoites in cultures. Dogs Aetiology

should not be allowed to eat aborted fetuses or fetal

The cause is Neospora caninum, a coccidial protozoan

membranes, and their faeces should be prevented from

discovered within the last decade, and now recognized

contaminating bovine feedstuffs.

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Toxoplasmosis

a seronegative status much more rapidly than do sheep,

which are as a result a more reliable indicator of the

Toxoplasmosis is a protozoan zoonosis that can infect

actual prevalence of the infection.

any species of vertebrate. It is mainly recognized as

being most pathogenic for sheep, however it occasion-

ally causes human illness, and it can cause significant

Diagnosis

disease in cattle, although abortion is not as common a

result as in sheep (see p. 895).

At autopsy or in abattoirs the most common findings

are multiple necrotic granulomatous foci in internal

organs. Histopathological examination is necessary for

Aetiology and epidemiology

confirmation. In vivo, cattle are presented with vague pyrexia disorders sometimes affecting the central

*The parasite involved is *Toxoplasma gondii*. It has a*

nervous system. If pregnant cows are affected, stillborn

two-host life cycle with both sexual and asexual stages.

or weak calves that die within a few days of birth may

The definitive hosts are cats, both domestic and wild,

result. Such calves frequently manifest nervous signs

which become infected by eating raw infected meat

such as tremors of the head or neck, teeth grinding or

from the intermediate host, which can be any other

convulsions.

species of vertebrate but is frequently a rodent. Once

Serological diagnosis can be made using a variety of

ingested the infective cysts are digested, each releasing

tests such as indirect immunofluorescence or enzyme-

large numbers of bradyzoites. They penetrate intestinal

linked immunosorbent assay (ELISA), or the Sabin-

epithelial cells and undergo a coccidia-like series of

Feldman dye test. Because of its ease of use, an ELISA

schizogonies and finally gametogony in the small intes-

using both anti-IgM and anti-IgG for detection of

tine to produce oocysts in faeces within one and a half

both acute and chronic infection is currently being

weeks after ingestion of cysts. The cat then excretes

developed.

oocysts for approximately two weeks. Oocysts remain viable for 17 months on pasture and if they contaminate animal feed any animal consuming it becomes infected.

Treatment and control

Within the intermediate host, development is asexual. The oocyst wall is disrupted, releasing eight sporozoites which reach the lymph and blood vessels by the use of pyrimethamine and sulpha compounds after penetrating the intestinal epithelium. The tachyzoites, as they are now called, are spread throughout the animals have been disappointing compared with those body to muscles, heart, lungs, liver, uterus and central in mice and humans. These drugs affect tachyzoites but nervous system, where they penetrate cells and multiply asexually. It is during this phase that the intermediate host becomes ill. After each multiplication the

monensin have shown prophylactic activity in sheep, infected cells rupture, releasing 8–16 tachyzoites for but there are no reports of its use in cattle (Buxton further spread. After some days of such divisions the *et al.* , 1987). Other preventative measures include pre-host develops some resistance to the organism and the vention of cat faecal contamination of animal food and spread of tachyzoites ceases, but is replaced by the for-adequate destruction of infected carcasses.

mation of slow-growing cysts, each of which eventually contains large numbers of bradyzoites. Normally these remain until ingested, but should the host's immunity

Sarcosporidiosis (sarcocystosis)

be reduced by other disease the invasive process can

Aetiology

recommence. The tissue cysts are susceptible to freezing and cooking to 70°C for 30 seconds and therefore

The cause is *Sarcocystis*, a coccidial protozoan that has consumption of adequately cooked infected meat is

a predator–prey life cycle. The indirect hosts are dogs, harmless.

cats and man and each may contain several species of

The organism is ubiquitous. Blewett (1983) reviewed sarcocyst, each one specific for a different intermediate the serological surveys that have been carried out in host. Sarcocystis hirsuta is present in the ox and cat domestic animals and came to the estimate that anti-species, S. cruzi in the ox and dog and S. hominis in body was present in 6.5 per cent of horses, 12.5 per cent the ox and man. In the final host the Sarcocystis spp. of cattle, 23.5 per cent of pigs, 30 per cent of sheep and undergo an enteric cycle with oocysts in the cells 40 per cent of cats. He also postulated that different producing sporocysts in the faeces. On entry to some animals vary in susceptibility to infection and in the rate intermediate hosts those infected via the dog undergo of loss of antibody, and concluded that cattle return to schizogamy in blood vessel endothelial cells before

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passing to the muscle. Those of the cat are not patho- ineffective. The difficulty with any treatment is the genic and are found in the oesophagus, and throughout maintenance of sufficient concentrations of the drugs the tissues of the intermediate host produce micro- in the cerebrospinal fluid, and relapses are usually

scopic cysts. If cattle are grazing and ingesting a few ascribed to failure to do so.

oocysts this probably results in a strong immunity. Signs develop about 26 days after infection (see p. 895).

Control

This is difficult but involves keeping carnivores away

Signs

from cattle. Infection in dogs and cats can be avoided

Usually no signs occur but, following ingestion of by feeding them cooked meat.

massive doses, calves may develop anorexia, fever, emaciation and anaemia. Adults can suffer pyrexia, emaciation and abortion.

References

Armour, J., Jennings, F.W. & Urquhart, G.M. (1969) Inhibition Necropsy

of Ostertagia ostertagi at the early fourth larval stage. II. The There are usually petechial and ecchymotic haemor-influence of environment on host or parasite. Research in

rhages throughout the body, especially in the heart,

Veterinary Science, 10, 238–44.

Blewett, D.A. (1983) The epidemiology of ovine toxoplasmo-brain, liver, lungs, kidneys and muscle. Some animals

sis. I. The interpretation of data for the prevalence of anti-show emaciation, lymphadenopathy, anaemia and body in sheep and other host species. *British Veterinary Journal*, **139**, 537–45.

ditis. Histologically, schizonts can be found in the

Buxton, D., Donald, K.M. & Finlayson, J. (1987) Monensin and endothelium throughout the body and haemorrhages,

the control of experimental ovine toxoplasmosis: a systemic lymphatic infiltration and oedema occur in the tissues effect. *Veterinary Record*, **120**, 618–19.

and organs.

Leaver, J.D. (1970) A comparison of grazing systems for dairy herd replacements. *Journal of Agricultural Science, Cambridge*, **75**, 265–72.

Diagnosis

McMeekin, C.P. (1954) Good rearing of dairy stock. *Bulletin*, This is usually based on post-mortem findings.

Department of Agriculture, New Zealand, No. 228.

Michel, J.F. (1968) The control of stomach worm infections in young cattle. *Journal of British Grassland Society*, **23**, Treatment

165–73.

Michel, J.F. (1969) *The epidemiology and control of some*
Pyrimethamine at 1 mg/ml and trimethoprim at
nematode infections. Advances in Parasitology, 7, 211–82.

5.0 mg/ml alone or in combination with sulphonamides

Urquhart, G.M., Armour, J., Duncan, J.L., Dunn, A.N. &
have been shown to inhibit the development of S.

Jennings, F.W. (1987) *Veterinary Parasitology. Longman* *neurona in cell*
cultures. Sulphonamides alone were

Scientific, Harlow, pp. 1–286.

Chapter 20

Respiratory Diseases

A.H. Andrews

Shipping fever (transit fever, pasteurellosis)

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induced with the participation of infectious agents. The

Infectious bovine rhinotracheitis (IBR)

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infection has been thought to be primarily viral, mainly
due either to parainfluenza III (PI3) virus or infectious
bovine rhinotracheitis (IBR) virus, followed by sec-

Shipping fever (transit fever,

*ondary infection with either *Pasteurella multocida* or **pasteurellosis)***

Mannheimia haemolytica (Pirie, 1979). *Mannheimia*

haemolytica biotype A serotype 1 is considered to be

Epidemiology

the most common isolate although biotype T strains

The condition is an important respiratory problem in

*have also been found. *Mannheimia haemolytica* biotype*

groups of weaned suckled calves that have been sold,

A produces a heat-labile cytotoxin that is ruminant-

often via a market, hence the terms transit fever or ship-

specific destroying leucocytes (Sherwen & Wilkie,

ping fever. They are usually over six months old and

*1982). *Pasteurella multocida* infection is only isolated under two years. During part of the sale transactions*

occasionally. Recently, it has been proved experimen-

they will probably have been mixed and grouped with

*tally that *M. haemolytica* biotype A1 can produce the*

other batches of animals of similar age during transport,

disease as a primary agent in non-immune calves (Shoo,

at the market or following arrival at the finishing farm.

1989). Disease does depend on administering the
Most cases occur within the first month of their entry
organisms into the lungs either intratracheally (Friend
to the fattening unit and most cases are seen at least 10
et al., 1977; Gibbs et al., 1983) or intrathoracically days after arrival (Andrews
et al., 1981). The disease (Houghton & Gourlay, 1984; Panciera & Corstvet,
is characterized by an illness of sudden onset with
1984). However, the exact mechanism by which the
dullness, pyrexia and anorexia. It is an acute exudative
organisms enter the lung and result in lesions is not yet
bronchopneumonia with toxemia as well as much
known. Other evidence of *M. haemolytica* involvement
fibrin present in the exudate and often accompanied
is that calves previously exposed naturally to the infec-
by fibrinous pleurisy (Blood & Radostits, 1989). The
tion are much more resistant to experimental and
condition is common in North America, Europe and
natural pneumonic pasteurellosis (Confer et al., 1984).
Britain. It is particularly prevalent in the American
Although *M. haemolytica* can be isolated from the
cattle feedlot industry and is the largest cause of mor-

nasal passages it is only in small numbers when calves
tality in that system. In a Colorado survey 75 per cent
are healthy and unstressed. The organisms can be
of all clinical disease in yearling cattle and 64 per cent
present in low numbers in the tracheal air but are not
of all post-mortem diagnoses were due to respiratory
considered to be normal inhabitants of the lungs (Grey
disease (Jensen et al., 1976). The morbidity and mortal-
& Thomson, 1971). However, as the calves are trans-
ity rates vary considerably but a level of up to 35 per
ported, move to market and then enter the feedlot, the
cent and a mortality of 5–10 per cent of those affected
number of *M. haemolytica* biotype A1 increases in the
or 0.75–1 per cent of the susceptible population is often
nasal tract, the tracheal air and then the lungs, where
quoted (Blood & Radostits, 1989). More recently in
bronchopneumonia can result (Frank & Smith, 1983).
Britain, morbidity rates of 73–100 per cent with a mor-
Thus management factors are important. The effects
tality of 0–8 per cent of those affected and an average
of stress are extremely difficult to quantify and have

mortality of 4 per cent have been reported (Andrews, not been successfully undertaken experimentally to 2000).

produce transit fever. However, transportation of yearling cattle does result in a rise in plasma fibrinogen levels, which is indicative of stress (Phillips, 1984).

Aetiology

Increased fibrinogen levels also occur following confinement in unfamiliar surroundings and deprivation of food and water. It is considered to be stress or management

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Respiratory Diseases • 287

denly introduced to a diet consisting of large amounts

Necropsy

of cereal are more prone to respiratory problems

Death is usually the result of anoxia and toxæmia (Wilson et al., 1985).

although occasionally in young cattle there is septi-

The environment tends to be important in that

caemia. Usually, over one-third of the lung shows disease seems to be related to the season of the year marked consolidation and the ventral parts of the apical (Andrews, 1978), fluctuations in daily ambient temperature and cardiac lobes are most involved. Initially, there is lung congestion and then hepatitis with exudate and, in 2.0–3.3 mm in diameter (MacVean et al., 1986). Calves some animals, emphysema. Bronchitis and bronchiolitis are usually catarrhal, often with serofibrinous minimum levels of nasopharyngeal bacteria at a relative humidity between 65 and 75 per cent. However, much pleural effusion.

bacterial counts tend to rise at humidities on either side of this range (Jones & Webster, 1981).

The interrelationship of different micro-organisms

Diagnosis

and transit fever still needs further elucidation.

However, cattle entering feedlots with low serological

This depends on the history of age, recent movement, titres to IBR, bovine respiratory syncytial virus (RSV) weaning or housing. The signs also help with severe and Mycoplasma dispar were at greater risk of being respiratory signs involving the anteroventral parts of treated for BRD (Rosendal & Martin, 1986). It has also the lungs and pyrexia. Post-mortem findings of lung been shown that IBR, PI3, bovine virus diarrhoea consolidation and pleurisy are present, and impression (BVD) and RSV are all associated with acute respiratory smears show bipolar staining organisms with methylene tory disease (Martin & Bohaz, 1986).

blue. Mannheimia haemolytica or P. multocida may In many outbreaks of transit fever it is likely that the be isolated from nasal swabs in live animals or lungs stress of movement plus mixing with other calves, the at post-mortem and their antibiotic sensitivity introduction to housing and a new diet are sufficient to determined. Serology for antibody rise can be under-initiate disease. The main organism involved is usually taken, as also can haematology but often the findings

a Mannheimia species, especially M. haemolytica are variable.

biotype A1. Spread of infection is optimized by crowded conditions in transport and markets. It is also probable that as animals go down with disease and pass

Differential diagnosis

on the infection, the organism increases in virulence.

Differential diagnosis includes enzootic pneumonia

This can be seen in some outbreaks in which disease can (see p. 242) but this usually occurs in younger calves be traced from individual animals to other members with a different history. Infectious bovine rhinotracheitis (see p. 289) usually shows mainly upper respiratory signs and a conjunctivitis. Fog fever (see p. 866) is mainly apparent in older cattle at grass after a pasture move in the autumn. Parasitic bronchitis (see p. 272) is present in cattle following grazing. Acute broncho-peracute form is unusual but results in sudden

Signs (see p. 921)

move in the autumn. Parasitic bronchitis (see p. 272) is present in cattle following grazing. Acute broncho-

The peracute form is unusual but results in sudden

pneumonia would be almost identical except for death with no premonitory signs.

bacteriological isolation. Contagious bovine pleuro-

In the acute form the animals are dull and inactive pneumonia is severe in all age groups of cattle and the with excessive oculo-nasal discharges, which may be affected animals suffer severe pain and toxæmia with mucopurulent. There is usually anorexia although a high mortality (see p. 868).

the cattle still drink, and a marked fever (40–41°C, 104–106°F). There is rapid (40+/minute), shallow breathing and a soft, productive cough, which tends

Treatment

to increase with exercise. On auscultation there are bronchial sounds over the anterior and ventral parts of Therapy for shipping fever is given in Table 20.1. Treatment of the lungs, which become louder as the condition continues. In some cases squeaks and high-pitched sounds some improvement within one to three days of initiation should begin early. Most cattle will usually show are heard, together with a pleuritic rub. Later signs can

ing treatment. Complete recovery may take four to include dyspnoea with marked abdominal breathing seven days. Where severe outbreaks occur it may occasionally be necessary to use mass medication by water animals. There is usually a favourable response to as most ill cattle still drink well. Anti-inflammatory therapy.

agents are often useful in promoting a speedy recovery.

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Prevention and control

ducing the calves hopes that by selling them at market he will obtain a better price than by selling direct. In Although the disease of transit fever is sporadic, many addition, the first farmer will be little concerned about of the conditions that predispose to the problem can be the subsequent disease status of his calves once they defined. This should mean that methods of prevention have left his farm for sale. This makes the possibility of should be relatively easy to institute. Ideally, the calves vaccination prior to movement difficult.

would be weaned at least two weeks prior to leaving the
 As cattle go through markets, disease prevention
 farm of birth (Andrews, 1976). They would be intro-
 tends not to be practised because many of the factors
 duced to the type of feed to be provided on the rearing
 enhancing the likelihood of a disease outbreak are
 farm. In addition, they would be batched in groups that
 outside the control of the purchasing beef rearers. Thus
 would go direct from the farm of birth to the rearing
 they cannot control the time before moving, the trans-
 farm. In reality this does not happen as the farmer pro-
 port of the animals from the farm of origin, their mixing

Table 20.1

Therapy for shipping fever.

Drug

Method of

Dose

Duration

administration

(mg/kg)

(mg/lb)

Amoxycillin trihydrate

s.c., i.m.

7

3

Daily for 3–5 days

Ampicillin trihydrate

s.c., i.m.

5–10

2.5–5

Daily for 3 days

Baquiloprim and

oral

4–8

2–4

Once or repeat

sulphadimidine

36–72

18–36

after 48 hours

Cefquinome

i.m.

1

0.5

Daily for 3–5 days

Ceftiofur

i.m.

1.1

0.5

Daily for 3–5 days

Chloramphenicol (some countries)

i.m.

10

5

3–4 times daily for 3 days

Danofloxacin

i.v., i.m.

1.25

0.6

Daily for 3–5 days

Enrofloxacin

s.c.

2.5

1.25

Daily for 3 days

s.c.

7.5

3.75

Once

Erythromycin

i.v., i.m.

5

2.5

Daily for 3 days

Florfenicol

i.m.

20

10

2 (48 hours apart)

s.c.

40

20

Once

Marbofloxacin

i.v., s.c.,

2

1

Daily for 3–5 days

i.m.

Oxytetracycline

i.v., s.c., i.m.

10

5

Daily for 3 days

Oxytetracycline (long-acting)

i.m.

20

10

Usually once

Penicillin/dihydrostreptomycin

i.m.

(20–30 000 iu/kg)

(10–15 000 iu/lb)

Daily for 3 days

20

10

Penicillin (long-acting)

benzathine

i.m.

5

2.5

Usually once

procaine

i.m.

6

3

Daily for 3–5 days

Spectinomycin

i.v., i.m.

10–20

5–10

Daily for 3–4 days

Spiramycin

i.m.

20

10

Daily for 2–3 days

Sulphadimidine

i.v., s.c., oral

150

75

Daily for 3 days

Tilmicosin

s.c.

10

5

Usually once

Trimethoprim and sulphadiazine

i.m.

4

2

Daily for 3–5 days

20

10

Trimethoprim and sulphadoxine

i.v., i.m.

3

1.5

Daily for 3–5 days

12.5

6

Tylosin

i.m.

4–10

2–5

Daily for 3–5 days

Mass medication

Oxytetracycline

oral

5

2.5

Daily for 5 days

Sulphamidine

oral

100

50

Daily for 5–7 days

Trimethoprim and sulphadiazine

oral

25

12.5

Daily for 5–7 days

Tylosin

oral

Daily for 5 days

All animals should be provided with good-quality feed and adequate ventilation. Corticosteroids, e.g. betamethasone, dexamethasone, are often found to help in reducing the level of exudation. NSAIDs may be useful in reducing inflammation, and Mannheimia antisera may also be helpful.

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with other cattle or the conditions present at market.

however shown only a slight decrease in morbidity of

Likewise they will probably have little direct say as to

disease, with slight improvement in relapse rates and

how the animals are transported or their mixing with

response to treatment (Bateman, 1988; Jim et al., 1988).

other groups before their entry to the rearing farm. It

is only once the cattle reach the rearers that control

measures can be undertaken. Obviously, they can

Infectious bovine rhinotracheitis (IBR)

ensure that the animals are kept in as satisfactory an environment as possible and are only lightly stocked.

Aetiology

This is best done by having the animals outside but this is resisted by most farmers because of the incon-

Infectious bovine rhinotracheitis (IBR) is a highly venience and extra labour required. The new ration for infectious and contagious disease of cattle. It is caused the cattle can also be introduced slowly.

by a virus known as bovine herpes virus 1 (BHV 1). It The problem is worse in North America where calves is generally considered that there are several strains of often travel vast distances. Here various precondition- the virus, although often the differences between the ing programmes are undertaken. It is advised that strains are not easily shown in the laboratory. The calves should be fed prior to weaning and kept in the disease is seen in North America, Europe and Africa. It same place once the dams are removed. Following is considered that the outbreak of severe respiratory weaning, regular checking of the calves several times a

disease that occurred in Great Britain in the mid-day should be practised. When animals are travelling 1970s was due to a new strain being imported. Several they should be given adequate periods of rest and European countries are undertaking eradication campaigns, many of which are progressing satisfactorily. They should also be well bedded during transit. Some farmers inject multivitamin preparations before moving and vaccines can be used, including those against Mannheimia and PI until the disease is no longer present.

3 virus.

In many countries antibiotics are used following the While the disease is primarily one of cattle, it can also move. These have given variable results mainly due to infect deer, goats and pigs. The main sources of infection between animals are nasal discharge (or eye discharge) because most problems occur about seven to ten days

charge), when it is affecting the respiratory system, and after entry to the feedlot or farm. When used, it is probably best to give long-acting preparations and initiate therapy when the first animals show disease. Use of reproductive tract (see p. 579).

antibiotic medication in the feed or water is a common practice in North America and while it appears to

Epidemiology

reduce mortality it has no effect on morbidity.

In most cases of respiratory infection disease is spread by aerosol. The genital form of the disease is contracted

Vaccination

venereally. As respiratory infection is spread via the air,

If it is considered that *M. haemolytica* and at times *P.*

it can spread by direct contact between the animals or

multocida form an important part of the disease syn-

where the cattle are in the same air space, i.e. there is

drome of transit fever then it might be possible to

air contact. This can only be avoided by ensuring the

obtain some protection by vaccination. Various vaccines extend from floor to ceiling and there is no contact with other animals either at the front of the pens or with are available in Britain.

other groups on either side. However, generally there In North America various *Pasteurella bacterins* and is a need for close sustained contact between groups. viral vaccines have been used to assist with the control This is why disease is often slow to spread within and of transit fever. Their efficacy appears to be low and between groups. It often takes two to five weeks to literature reviews suggest that at present there is little spread in a group. However, unexplained outbreaks of evidence to show the efficacy of such vaccines under IBR do occur on isolated farms.

feedlot conditions (Myers, 1984).

Recently in North America several new vaccines

Latent infection: There is a problem with BHV 1 in that have become available. One of these is a *M. haemolyticus*-it is able to become a latent infection. Thus once an

ica vaccine/bacterial extract. This has been shown to be animal is infected with

a strain of the virus or vaccinated effective to controlled challenge provided two doses with a live viral vaccine, the virus may remain in the of vaccine have been administered prior to challenge animal for the rest of its life without it showing any signs (Sherwen & Wilkie, 1988). Response in a field trial has of illness. The site of latency is arguable but the virus

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can be found in the trigeminal nerve of clinically normal (2)

Subacute disease. This is commonly seen in adult cattle. However, if the animal is stressed at all, such as cattle and often is of only short duration. There is a when moved or calving, or becomes ill, the virus may marked rise in temperature lasting only a day or two be shed. Shedding can also occur in bulls when mating. (40°C, 104°F). Often there is a marked drop in milk This shedding may be, but is often not, accompanied yield. Again the lining of the eyes is reddened with dis- by any signs of disease. Corticosteroid injections may charge and this also involves reddening of the lining of

promote virus shedding. The level of this latent infection is variable but can be up to 10 per cent of clinically normal animals. In addition, while most of these cattle is rapid and shallow. The animals recover in 10 to 14 days. will show antibody levels to the BHV 1 virus, some do

not. Thus all cattle in herds where disease occurs have
(3)

Acute disease. The signs are as in the subacute form but the temperature tends to be higher (40–41°C, 104–106°F). These signs are particularly present in way of knowing whether or not disease is present is to growing cattle between six months and two years old. look at the results of milk or blood tests. If a blood test shows an antibody titre (positive response) to BHV 1 and there is some respiratory distress. The discharge

in cattle other than calves it means that the animal(s) from eyes and nose tends to become profuse, yellow, have been exposed to infection and could possibly be thick and purulent. The lining of the nose may show carriers. If a blood test from an animal is negative it grey areas, which consist of dead tissues that smell and generally means that the animal is unlikely to have been are shed. The signs tend to last a lot longer before exposed to infection, but it does not completely rule out recovery occurs.

the possibility that the animal is a latent carrier of disease. Thus tests on individual animals are of limited (4)

Peracute disease. The animals develop very high significance; however, on a herd basis repeated negative temperatures (42°C, 108°F) with eye and nasal dis-tests indicate that it is highly unlikely infection is charge, respiratory distress, cough and then death in present. When tests show several animals with antibody about 24 hours.

levels in the herd then infection is present. The calf that

Some cattle may die owing to complications and this receives colostrum from its infected mother will also is particularly so in the six month to two year age group. show a positive antibody level to the test although not Here mortality may reach 10 per cent or more. necessarily being infected. The antibody provided from However, it is usually only about 1 per cent. Some cattle the colostrum will usually remain for one to six months, keep a stertor (snoring breathing) for many months. depending on the amount received from the mother. Some become 'puffers' with bouts of respiratory signs including distress when breathing, loss of appetite and Incubation period: The incubation period for the cough. These usually have a secondary bronchopneumonia and die in weeks or months after losing condition. Often they become recumbent before death. occurs about 10–20 days after introduction of infected Characteristically, there is a sudden outbreak of or susceptible animals. Longer incubation periods do

disease involving the respiratory tract. This will initially occur. This is quite likely when it is considered that involve the group of cattle to which infection has been latent carriers may not be shedding virus at a particular time. It will then tend to spread round all the

other groups and ages of cattle. This can often take a few weeks or many months, dependent on the strain of

Signs

virus, the level of infection and the degree of exposure to infection of each group. Characteristically, an outbreak in a group reaches a peak at about two or three weeks. It is usually seen in cattle over six months old but weeks after its start and is over between the fourth and sixth week. can occur at a younger age. The signs vary considerably

but typically they tend to be worst in animals from about six months to two years old.

Reproductive form: In most cases the reproductive and respiratory forms are not seen together.

(1)

Mild disease. This is just a conjunctivitis, i.e. slight reddening to the lining of the eye and eyelids with some

(1)

Infectious pustular vulvovaginitis. This is an infection discharge, usually watery and clear (see p. 921). This condition of the vulva and vagina often lasting several weeks. often occurs when the disease strain is mild or degree It results in the discharge of pus from the vulva, usually of infection is low, or the animal is resistant.

in small quantities. There is reddening of the lining and

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pustule presence. In some cases infection causes irritation mucous membranes with a mucoid discharge to large tion in the vagina resulting in frequent urination and areas being covered by a necrotic layer of exudate possibly increased straining (see p. 628).

(Pirie, 1979). In some cases there is pulmonary emphysema and secondary exudative bronchopneumonia,

(2)

Infectious bovine penoposthitis. Bulls show a

which may be purulent or necrotizing. Histologically, small amount of purulent discharge from the prepuce. the mucous membranes show acute catarrhal inflammation and in some cases the epithelium and larynx with haemorrhages and small necrotic areas. tend to be infiltrated with neutrophils, lymphocytes,

(3)

Endometritis. Infection of the uterus, with dis- plasma cells and macrophages. In naturally occurring charge, poor conception rates and short returns to infections, inclusion bodies appear to be absent. oestrus, can occur if semen is infected.

Abortion: Aborted fetuses often show marked autoly-

(4)

Abortion (see p. 579). This is becoming increas- sis and focal necrotic hepatitis. ingly common and occurs some weeks or months after the animal is infected. Abortion is usually at six to eight

Alimentary form: Epithelial necrosis of the turbinates, months of pregnancy and often the placenta is retained

oesophagus, rumen and abomasum. Inclusion bodies following abortion. In some cases of infection calves are often evident in the surviving epithelium.

born weak at the normal time.

Nervous form: A non-suppurative encephalitis occurs

Generalized/alimentary form: This is seen in young particularly affecting the cerebral cortex and internal newborn calves and is recorded mainly in America. It capsule.

occurs when calves receive little or no immunity in the colostrum and are then subjected to infection. The animals show a severe temperature reaction, they do

Diagnosis

not eat and they salivate. The lining of the nose is red.

This will depend on the type but in the alimentary form

The eyes may show a conjunctivitis. In addition, the

it will depend on a history of IBR in the herd with virus

lining of the mouth and the soft palate (at the back

present in the faeces or nasal swabs and possible

of the throat) are reddened with mucus present. The

necrotic areas on the turbinates. The nervous form is

opening to the trachea, i.e. the pharynx, is also red-
difficult to differentiate except that there is probably an
dened with much discharge present. There is usually
IBR outbreak in the herd. At post-mortem examination
severe respiratory distress with a bronchopneumonia.
the turbinates may show necrotic lesions.

Some calves show severe diarrhoea and dehydration.

In the respiratory form the history is that a new
animal or group has entered the herd. In most cases

Central nervous system/encephalitic form: This is seen several animals will be
affected and besides varying

in calves under six months old and involves brain signs.

degrees of respiratory signs there will usually be a

The animals show incoordination with bouts of excite-
conjunctivitis with copious, initially serous, ocular dis-
ment and depression. In other cases there are convul-
charge. There may be necrosis of the nasal mucosa but
sions, bellowing and blindness with salivation. This
this is absent in the mouth. The virus can be isolated in
nervous form is rare.

upper nasal or ocular swabs and is detected by fluo-

rescent antibody staining. Otherwise paired blood

Other infections: Reports of infections of the udder and samples will show a rise in titre to the enzyme-linked

intestinal tract have been made. However, these are

immunosorbent assay (ELISA) test. Other tests used

rarely the only systems affected.

include the serum neutralization test, indirect haemag-

glutination test, complement fixation test and virus neu-

tralization test. Bulk milk testing will indicate the status

Necropsy

of a dairy herd.

Respiratory form: In uncomplicated cases, lesions are

restricted to the upper respiratory tract terminating at

Treatment

the upper bronchi. Inflammation of the muzzle and the

nasal cavities varies from some congestion and petechi-

There is a considerable divergence of opinion as to

ation with muroid exudate, to a fibrinopurulent exudate

whether or not to use antibiotic treatment. If the

with necrosis of the nasal mucous membranes. The sub-

disease appears to be uncomplicated, and this is

mandibular and retropharyngeal lymph nodes tend to be unusual, then there would appear to be little point in being swollen and oedematous. There is a laryngotracheo-therapy. If, however, as in most cases, there is second-bronchitis, which varies from a mild congestion of the ary lung involvement, then therapy is justified. At the

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start of an outbreak all cases should be isolated as soon as possible because although it may not stop spread can be used in an IBR eradication campaign. Live vaccines, as other vaccines, can present a possible danger if of the disease, the first animals are often clinically the contaminated with other pathogens, such as BVDV. worst affected. If antimicrobial agents are to be used, Beef calves can be vaccinated about two weeks or so all ill calves should be treated for three to five days with before they are weaned. One vaccine is temperature any one of a number of drugs, including penicillin and sensitive and so only replicates in the upper parts of streptomycin, oxytetracycline, ampicillin, trimethoprim the respiratory tract. However, the vaccine can still pro-

and sulphadiazine, sulphadimidine, sulphamethoxypyridine and sulphapyrazole. The animal should be given will preclude their export or their sale to AI centres.

good, wholesome feed and it should be encouraged

Another vaccine does replicate in organs other than the to eat and drink. Some farmers have vaccinated their lungs and produces a good systemic immunity. The vaccines with live vaccine after the start of an outbreak, provide effective immunity but they do allow the with good results, but to be successful infection must replication and re-excretion of the IBR virus, which can not have become established. It should be remembered thus spread infection to non-vaccinated animals.

that for effective protection, vaccines should be intro-

Several countries within the EU have successfully duced before the onset of infection. Several compounds eradicated BHV1 (IBR), including Denmark, Finland have been found that are active against herpes viruses.

and Sweden. An EU-approved national compulsory

However, many are toxic, but one drug, acyclovir, has

eradication scheme is being undertaken in Austria.

been shown to be safe and may in the future be tried in

*Control programmes are being pursued in the Nether-
animals.*

*lands, Belgium, France and Germany. A programme for
the monitoring, screening, eradication and accreditation
of freedom from IBR is being undertaken in the UK*

Control

by Cattle Health Certification Standards (CHeCS).

*Management: It is best to keep a closed herd. However, in America, and also
Britain, infection has been found*

References

*in closed herds. Any new animal entering a known un-
infected herd should be blood tested prior to entry. If*

*Andrews, A.H. (1976) Factors affecting the incidence of pneu-
the test is negative the animal should be isolated for a
monia in growing bulls. Veterinary Record, 98, 52–5.*

month and then retested. The use of corticosteroids

*Andrews, A.H. (1978) Some factors influencing respiratory
may allow detection of virus in swabs of carrier animals.*

disease in growing bulls and the effect of treatment on

If the tests are all negative the animal can be allowed

liveweight. In Respiratory Diseases in Cattle. Seminar in to enter the herd. The risks then of the introduction of

CEC Programme of Coordination of Research in Beef

infection are small. If the animal suffers a respiratory

Production, Edinburgh, 8–10 November 1977 (ed. by W.B.

problem while in isolation the second test should not

Martin), pp. 169–80. Martinus Nijhoff, The Hague.

be until two or three weeks after the end of the episode.

Andrews, A.H. (2000) Calf pneumonia costs! Cattle Practice, Some farmers may need to go to these lengths to keep

8, 109–14.

their pedigree herds free from disease, because many

Andrews, A.H., Cook, G.L., Pritchard, D.G., Morzaria, S.P.

& Gilmour, N.J.L. (1981) Observations on a respiratory

European countries will not accept exports unless they

disease outbreak in weaned suckled calves. Veterinary

are shown to have a negative titre for IBR. The same

Record, 108, 139–42.

conditions at present govern the entry of bulls to some

*Bateman, K.G. (1988) Efficacy of *Pasteurella haemolytica**

artificial insemination (AI) centres.

vaccine/bacterial extract in the prevention of bovine respiratory disease in recently shipped feedlot calves. *Canadian Vaccination* (see p. 1010): *An inactivated multicompo-Veterinary Journal*, **29**, 838–9.

nent vaccine has been available for a long time. The

Blood, D.C. & Radostits, O.M. (1989) *Pneumonic pasteurel-*

vaccine could be given as doses two to four weeks apart

losis of cattle (shipping fever pneumonia). In *Veterinary*

with a third injection at 10–12 weeks old. Its efficacy has

Medicine, 7th edn, pp. 663–73. Ballière Tindall, London.

at times been questioned. Subsequently, live IBR vac-

Confer, A.W., Panciera, R.J. & Fulton, R.W. (1984) *Effect*

cines have become available. They are given by

of prior exposure to *Pasteurella haemolytica* antiserum on experimental pneumonic pasteurellosis. *American Journal*

intranasal inoculation or injection and should be

of *Veterinary Research*, **45**, 2622–4.

administered 24–48 hours after entry to the farm.

Frank, G.H. & Smith, P.C. (1983) *Prevalence of Pasteurella* Ideally, calves should be vaccinated 10 days prior to any

haemolytica in transported calves. *American Journal of movement*. A gene-depleted live vaccine is available in

Veterinary Research, **44**, 981–5.

some countries and allows control of disease. The

Friend, S.C., Thomson, R.G. & Wilkie, B.N. (1977) Pulmonary immunity produced allows differentiation from that

lesions induced by *Pasteurella haemolytica* in cattle.

produced by the wild strain. This means the vaccination

Canadian Journal of Comparative Medicine, **41**, 219–23.

Respiratory Diseases • 293

Gibbs, H.A., Allan, E.M., Selman, I.E. & Wiseman, A. (1983) in Ontario feedlot cattle. *Canadian Journal of Veterinary*

Experimental bovine pneumonic pasteurellosis. Veterinary

Research, **50**, 351–8.

Record, **113**, 144.

Myers, L.C. (1984) Questions on the efficacy of cattle

Grey, C.L. & Thomson, R.G. (1971) *Pasteurella haemolytica* in vaccines. *Journal of the American Veterinary Association*, the tracheal air of calves. *Canadian Journal of Comparative* **184**, 5–7.

Medicine, **35**, 121–8.

Panciera, R.J. & Corstvet, R.E. (1984) Bovine pneumonic

Houghton, S.B. & Gourlay, R.N. (1984) Bacteria associated

pasteurellosis: model for Pasteurella haemolytica and Pas-with calf pneumonia and their effect on gnotobiotic calves.

teurella multocida-induced pneumonia in cattle. American Research in Veterinary Science, **37**, 194–8.

Journal of Veterinary Research, **45**, 2532–7.

Jensen, R., Pierson, R.E., Braddy, R.M., Saari, D.A.,

Phillips, W.A. (1984) *The effect of assembling and transit*

Lauerman, L.H., England, J.J., Keyvan, F.A.R., Collier,
stresses on plasma fibrinogen concentration of beef calves.

I.J.R., Horton, D.P., McChesney, A.E., Benitez, A. &

Canadian Journal of Comparative Medicine, 48, 35–41.

Christie, R.M. (1976) Shipping fever pneumonia in yearling

Pirie, H.M. (1979) Respiratory Diseases of Animals, pp. 68–70, feedlot cattle.
Journal of the American Veterinary Medical 71–4. Notes for a Postgraduate
Course, Glasgow Veterinary

Association, 169, 500–506.

School.

Jim, K., Guichon, T. & Shaw, G. (1988) Protecting feedlot

Rosendal, S. & Martin, S.W. (1986) The association between calves from
pneumonic pasteurellosis. Veterinary Medicine, serological evidence of
Mycoplasma infection and respira-83, 1084–7.

tory disease in feedlot cattle. Canadian Journal of Veterinary Jones, C.R. &
Webster, A.J.F. (1981) Weather induced changes Research, 50, 179–83.

in airborne bacteria within a calf house. Veterinary Record, Sherwen, P.E. &
Wilkie, B.W. (1982) Cytotoxin of Pasteurella 109, 493–4.

haemolytica acting on bovine leucocytes. Infection and MacVean, D., Franzen,
D.K., Keefe, T.J. & Bennett, B.W.

Immunity, 35, 91–4.

(1986) Airborne particle concentration and meteorological

Sherwen, P.E. & Wilkie, B.W. (1988) Vaccination of calves with conditions
associated with pneumonia incidence in feedlot

leukotoxic culture supernatant from Pasteurella haemolyt-

calves. American Journal of Veterinary Research, 47, ica. Canadian Journal of Veterinary Research, 52, 30–6.

2676–82.

Shoo, M.K. (1989) Experimental bovine pneumonic pas-

Martin, S.W. & Bohaz, J.G. (1986) The association between

teurellosis: A review. Veterinary Record, 124, 141–4.

serological titres in infectious bovine rhinotracheitis virus, Wilson, S.H., Church, T.L. & Acres, S.D. (1985) The influence bovine viral diarrhoea virus, parainfluenza III virus, respi-of feedlot management on an outbreak of bovine respira-

ratory syncytial virus and treatment for respiratory disease

tory disease. Canadian Veterinary Journal, 26, 335–41.

Chapter 21

Trace Element Disorders

N.F. Suttle

Introduction

294

most common being malnutrition, which is likely to be a

Differential diagnosis

294

whole herd problem. A preliminary check must be made

Definitions

294

of the quantity and quality of available feeds (Sinclair & Cobalt disorders

295

Suttle, 2001). Trace element disorders usually afflict only

Copper disorders

298

a minority of the herd but a preliminary 'audit' of the

Iodine disorders

301

likely mineral inputs and needs may narrow the range of

Selenium disorders

302

possible culprits (Suttle & Sinclair, 2000). Responses to

Manganese and zinc disorders

305

a broad mix of micronutrients would confirm this line of

investigation. Depending on the history of the farm or

area, it may be beneficial to attempt an earlier definitive

Introduction

diagnosis by using a specific trace element supplement,

but proof of responsiveness amongst an affected minor-

*The full impact that trace element disorders can have
ity may require unconventional statistical techniques
on the health of cattle was seen when Europeans
which take into account initial variation in performance
attempted to carry their methods of animal production
(Suttle, 2000). There may be cross-over between lines of
to the New World and to the Antipodes. The geologi-
investigation: correction of a gut parasite problem might
cally young soils bordering Australia, and forming the
improve trace element status, while improving the major
Florida peninsula and the interior of New Zealand's
nutrient supply of a herd (by improving ration digestibil-
North Island, were a graveyard for many an animal
ity) may lower status and increase the risk of trace
until the therapeutic effect of newly discovered essen-
element disorders. Seasonal and annual variations in
tial trace elements became known. Crop and pasture
incidence mean that it will always be necessary to review
growth was also limited by some of the deficiencies but
the need for any trace element intervention from time to
these were the more easily diagnosed because they pro-*

time. Overall, following such a scheme should improve
duced characteristic foliar abnormalities. The animal's
the precision of diagnoses.

needs were more difficult to delineate because they
lacked clinical definition and persisted where the more
modest needs of the plant had been met. Since those

Definitions

pioneering days, trace element disorders have been
described in many areas of the world and they continue

Four terms will occur frequently in descriptions of the
to appear when new methods of production are intro-
sequence of events that occur when cattle are deprived
duced, but it is important to keep them in perspective.

of essential trace elements, and these require definition:

- Depletion: the reduction of body stores of the

Differential diagnosis

element.

- Deficiency: the presence of subnormal concentra-
tions of the element in blood or tissues.

Trace element disorders carry more than their fair share

- Disorder: the malfunction of trace element-

of blame for poor cattle performance, due to the non-dependent body processes (subclinical disorder).

specificity of most clinical signs, the pressures on practi-

- Disease: the presence of visible, clinical*

tioners to offer a quick diagnosis and the alacrity with abnormalities.

which clients accept a diagnosis that may seem to exonerate them from blame. Figure 21.1 presents a scheme

They are particularly useful when describing the

for differential diagnosis of some common problems. Of

trace element status of cattle biochemically, and avoid

the three main lines of investigation shown, trace ele-

the confusion caused by using the one word 'deficiency'

ments are only part of the least probable explanation, the to describe all four stages, and as a 'tag' for interpret-294

Trace Element Disorders • 295

NON-SPECIFIC DISORDER

(Ill-thrift, infertility, retained

placenta)a

INFECTION

MALNUTRITION

MICRONUTRIENT

Possible

(Microbial or

(Energy,

DEPRIVATION

cause

parasitic)

E/protein, P)

(Mineral or vitamin)

PATHOGEN

Preliminary

MINERAL/

AUDIT

FEED AUDIT^b

check

VITAMIN AUDIT^c

(WBC, APR, FEC, etc.)

Multi-mineral/vitamin

Broad spectrum

Preliminary

More feed and/or

mix (introduce,

antibiotic or

intervention

change E : P in feeds

provide more or

anthelminthic

reformulate)e

Definitive

Identify

Metabolic

Mineral/vitamin

test

pathogen (s)

profile

profiled

Sustainable

Sustainable

Targeted

Specific trace

drug

cropping, grazing &

intervention

element supplement

programme

feed purchase policy

DIAGNOSIS

Fig. 21.1

Scheme for the differential

a

diagnosis of trace element disorders with

Firstly, rule out 'exotic' (e.g. plant, agrochemical or industrial poisons) or common (poor oestrous detection, large calf) factors.

non-specific signs; proceed vertically with

b See Suttle (2002) for fuller details.

a given line of investigation only while test

c See Suttle & Sinclair (2000) for fuller details.

results are positive. WBC = white blood

d See Table 21.1 for best indices of trace element status.

e

cell count; APR = acute phase proteins;

Some untreated animals must be left to prove efficacy and allow specific follow-up FEC = faecal egg count.

tests to be made.

ing indices of mineral status which rarely reflect onset molecules, methyl-(Me) and adenosyl-(Ado) Cbl, which of disorder or disease. Throughout this chapter, use will have contrasting functions in the body. MeCbl acts as a be made of 'marginal bands' for diagnostic criteria coenzyme to methionine synthetase and is linked to (Table 21.1), which reflect the uncertainty of detecting folate metabolism, through the use of methyltetrahy- the onset of health-limiting conditions. The following drofolate as the methyl group donor. Deficiencies of descriptions of specific disorders concentrate on the this coenzyme can theoretically impair methionine more commonly encountered deficiencies of cobalt synthesis and the bioavailability of folate and cause (Co), copper (Cu), iodine (I) and selenium (Se).

formiminoglutamic acid (FIGLU) to appear in urine.

AdoCbl enables propionate to be used for gluconeogenesis via succinate and the tricarboxylic acid (TCA)

Cobalt disorders

cycle, acting as coenzyme to methylmalonyl-CoA

isomerase: insufficiency causes methylmalonic acid

(MMA) to accumulate.

Aetiology

The anorexia and anaemia that are successive

The only known functions of Co arise from its place at

debilitating consequences of deprivation may reflect

the centre of the corrin ring of two cobalamin (Cbl)

the dysfunction of AdoCbl and/or MeCbl (Underwood

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Table 21.1

Marginal bands for commonly used biochemical indices of micronutrient status in the diet, serum or tissues of cattle: mean values falling within bands indicate a possibility of benefit from trace element supplementation; the more extreme the value and the more parameters affected, the greater the possibility becomes; values below (above for Fe:Cu and thyroid weight) bands indicate a probable benefit.

Cu

Co or vit B12

I

Mn

Se

Zn

Dieta (mg/kg DM)

5–15

0.04–0.06

0.08–0.3

10–15

0.03–0.06

10–20

Serumb (mmol/l)

3–8

0.25–0.5 ¥ 10⁻³M

0.08–0.20

0.09–0.11

0.13–0.26B

6–9

Tissuec (mmol/kg DM)

150–300nL

0.2–0.4L

9.5–15.8 ¥ 10³T

11–13H

0.6–1.3nL

765–1070R

a Higher values are appropriate for: antagonist-containing diets (for Cu, Cu:Mo < 1–3, Fe:Cu > 50–100; Cu:sulphur < 0.0025); for I, with high goitrogen intake); for low agonist intake (e.g. for Se, low vitamin E intakes); for all

elements when the digestibility of the ration is high.

b The ranges are for adults; newborns often have different norms (e.g. 3–9 mmol/l then being adequate for serum Cu).

c Superscript letters indicate sample origin: B = whole blood, H = heart, L = liver, M = milk, R = rib, T = thyroid; for the latter, an organ weight range of 0.3–0.8 g FW/kg LW is marginal; for the newborn (n), ranges may again differ, L Se being up to 3¥ that in the adult and L Cu 7–10¥ higher.

Divide values by 3.3 (L) or 4.0 (H,T) for approximate fresh weight basis.

Conversion from mmolar to mg units requires multiplication by: 63.5 for Cu, 58.9 for Co, 126.9 for I, 54.9 for Mn, 79.0 for Se, 65.4 for Zn and 1355

for B12; division by these factors converts mg to mmol units.

& Suttle, 1999). Loss of appetite was initially linked

Clinical signs

to Adocbl dysfunction and increased blood propionate

All ruminants require a dietary supply of Co for the

concentrations, but disturbances of rumen fermenta-

synthesis of the essential vitamin B

tion (McDonald & Suttle, 1986; Kennedy et al.,

12 by rumen micro-

organisms. Cattle are less susceptible to a lack of Co

1991) and the accumulation of other metabolites

than sheep, but when they succumb, they develop essen-

(e.g. MMA, Rice et al., 1989; succinate, Kennedy et al., tially the same clinical abnormalities in the same order:

1991; homocysteine, Stangl et al., 2000) may also affect anorexia, loss of body condition, muscular wasting and

appetite. Loss of appetite would compound the effect

finally an anaemia that is both normochromic and

of a diminishing supply of the glucose precursor,

normocytic. In the early stages of the disease, the hair

propionate. The anaemia of Co deficiency may reflect

coat can become rough and discoloured (Judson et al.,

the role of MeCbl in DNA synthesis and red cell

1982) and may be repeatedly licked. Appetite may

maturation.

become depraved (pica). Eventually, the skin becomes

The basic cause of cobalt-responsive disorders is a

thin and fragile and the mucous membranes pallid. The

simple nutritional deficit that can be traced to

clinical picture is thus one of a 'pining' or 'wasting'

impoverished soils in which crops or pastures have

disease, indistinguishable from many other causes of ill-

grown. Interactions can occur in the soil that leave

thrift. Cobalt-deprived cattle may be more susceptible

most of the Co adsorbed in unavailable forms onto

to infections than normal cattle (MacPherson et al.,
manganese oxides, particularly at alkaline soil pH with
1987a).

soil Mn >1 g/kg (Suttle, 2000). If extraction with 0.43 m

There is a neurological disease that affects cattle in

acetic acid yields <0.3 mg Co/kg air-dry soil, deficiencies Australia when they
graze *Phalaris tuberosa* pastures

may develop. The diet should contain >0.05 mg Co/kg

called 'Phalaris staggers'; it can be prevented by Co

dry matter (DM) (Underwood & Suttle, 1999). Inter-

dosage but not by administration of vitamin B

actions in the animal have been postulated because

12 and is,

therefore, not a classical 'deficiency' syndrome. The

Co can be incorporated into various analogues of

response to Co may arise through the neutralization of

B12 by the rumen microbes. There is thus competition

for Co in the rumen between different forms of the

Suttle, 1999).

vitamin. Cobalamins are selectively absorbed in associ-

ation with intrinsic factor but if analogues find their way into the bloodstream or tissues, they might com-

Necropsy

plicate the assessment of vitamin B12 status and compromise functions of the vitamin. However, protective

At necropsy, cattle suffering from Co deprivation show mechanisms would probably have evolved, if they are the hallmarks of starvation. There is little body fat needed, against analogues so copiously produced in the except occasionally in the liver, which may be pale rumen.

and friable due to fatty infiltration; this condition is,

Trace Element Disorders • 297

however, far less common in cattle than in lambs or goat shows better fit at low than at high levels (Mitsioulis kids. In severely affected, anaemic individuals, bone et al., 1995; Suttle, 1995); values between 0.06 and marrow hypoplasia and splenic haemosiderosis will be 0.08 mg Co/kg DM can be regarded as 'marginal' found.

(Underwood & Suttle, 1999).

The diagnosis of Co disorders from the presence of abnormal metabolites in the blood and urine was devel-

Diagnosis

oped in sheep and non-ruminants and extended to

Cobalt-responsive disorders are best diagnosed through cattle. Quirk & Norton (1988) found that heifers

responses in growth or health to Co or B12 supplemen-

grazing pastures low in Co (0.036 mg/kg DM) remained

tation in controlled trials (e.g. Duncan et al., 1986). Bio-healthy and excreted no MMA or FIGLU in urine,

chemical confirmation of subclinical or clinical disease

despite low plasma B12 concentrations (96 pg/ml). After

from the assay of plasma B12 is more difficult in cattle

calving, milk yield was unaffected but very low B12

than in sheep. Plasma B12 concentrations appear to

concentrations in the milk (42–86 ng or 31–63 pmol/l)

respond feebly to Co supplementation in cattle. Oral

caused depletion of plasma B12 in their calves (59–

doses of Co ten times higher on a body weight basis

74 ng/l) and growth retardation, which was accompa-

than those that increase plasma B12 in sheep were

nied by increased urinary excretion of FIGLU. They

apparently ineffective in cattle (N.F. Suttle & J. concluded that MMA was less reliable than FIGLU as Brebner, unpublished data). High plasma B12 concentrations (>500 ng/l) are rarely found in cattle yet they are relatively insensitive (30 mmol/l). Assay of plasma MMA has been advanced as a measure of functional B12 status in sheep, with concentrations >5 mmol/l taken to be abnormal (Rice et al., 1987): as yet plasma MMA there are exceptions (e.g. Duncan et al., 1986). The assay has not been tested for the confirmation of Co dys-of B12 by the more favoured radioisotope dilution function in cattle but a marginal band of $5\text{--}15$ mmol/l (RID) method is susceptible to interference from non-specific binders (Millar et al., 1984; Schulz, 1987a, b); teine are found after prolonged Co deprivation (Stangl et al., 2000). plasma (transcobalamins), which show quantitative dif-

ferences from man (Schulz, 1987a) as well as seasonal

Treatment or prevention

anomalies (Millar et al., 1984). Since binding affects the recovery of B12 after deproteinization of plasma or

Although there are clearly differences in B12 metabolism, it can impair assay by both microbiological and
lism between cattle and sheep, there is no evidence that
RID methods. The results of assays of B12 in bovine
these affect the methods of treating and preventing dis-
plasma can vary substantially between laboratories
orders. The injection of B12 as cyano- or hydroxycobal-
(Schulz, 1987b) and assays of the analogue component
amin, 2–6 mg/50 kg liveweight, provides protection for
by difference, using specific and non-specific binders,
several weeks, even though responses in plasma B12
are particularly prone to error. Extraction of the
soon dissipate. Slow-release, injectable preparations of
vitamin at alkaline pH overcomes the problem and
B12 are being developed in the Antipodes.

modified assay kits are now available (Stangl et al.,

Heavy cobalt oxide pellets given orally, in pairs or

2000).

singly with a 'grinder', improve the B12 status of growing

Assays of vitamin B12 in skimmed milk and liver are

and adult cattle for several months (Judson et al., 1981; far more reliable than those in plasma and values for

Quirk & Norton, 1988), even when a change in formu-

milk and liver from the same individual correlate rea-

lation halves their Co content (Judson et al., 1997b); the sonably well with each other, particularly at low levels

soluble glass bolus can also give sustained protection

that have diagnostic significance; milk B

<

12

0.5 nmol

(Allen et al., 1985). The effectiveness of infrequent oral (678 ng/l) indicates a possibility of ill health (Judson

doses of Co in anthelmintics has not been tested but

et al., 1997b) (Table 21.1). By contrast, the relationship weekly doses of 35–70 mg Co without anthelmintic are

between plasma and liver B12 values is poor, particularly

said to be effective (Underwood & Suttle, 1999). Cobalt

at low levels (Judson et al., 1997a). Diagnostic thresh-supplementation via the drinking water can be prac-

olds for liver B12 in sheep have long been extrapolated
tised where there is a piped water supply (MacPherson,
to cattle; thus values <0.10 mg/kg fresh liver have been
1983). Use of 'protected' or chelated forms for oral use
taken to indicate moderate to severe deficiency and
is contraindicated because Co must be given in forms
values >0.19 mg (>140 pmol)/kg have been regarded as
which can be rapidly incorporated into vitamin B12 by
normal. There is a curvilinear relationship between Co
rumen microbes. Evidence that volatile fatty acid
and vitamin B12 concentrations in bovine liver which
(VFA) production by rumen microbes is influenced by

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Co deficiency (McDonald & Suttle, 1986; Rice et al., been suggested that they
may reflect direct effects of

1989; Kennedy et al., 1991) suggests a possible advan-Mo on the central control
of appetite (Phillippo et al., tage for the oral route over the injection route, since
1987b). It is, however, premature to discount the role of
only the former meets the needs of the microbes for B12.

Cu (Suttle, 1988; Underwood & Suttle, 1999) and other
Comparisons between methods have rarely been con-
factors that influence Cu status should not be ignored.

ducted but one at least showed no advantage in calf

Copper absorption in cattle is as sensitive to Mo inhi-

growth for oral Co versus injected B12 (Judson et al., bition as it is in sheep and the equations derived for

1981).

grazing sheep to predict Cu absorption (Suttle, 1983b)

Administration of Co on a group basis via licks con-

should give an approximate weighting for cattle. High

taining 0.1 per cent Co or mineral mixes containing up

S intakes from the diet (Suttle, 1983a) or drinking water

to 0.4 per cent Co may provide an inexpensive means

(Smart et al., 1986) also suppress Cu absorption. Poor of prevention but could not be guaranteed to protect

Cu absorption, and hence disorders, will therefore be

all individuals. The best long-term strategy on certain

more common on lush, immature, S-rich swards than on

soil types will be to apply Co salts, such as hydrated

brown, mature swards and in wet than in dry seasons.

CoSO₄, at 2–3 kg/ha, every three to four years.

Copper is also absorbed better from hay than from

Responses may, however, be short-lived on sandy soils

fresh grass of similar S content (Suttle, 1983b). Ensiled

*and negligible on calcareous or recently limed soils con-
grass is a good source of absorbable Cu while S con-
taining >1 g Mn/kg DM because most of the Co
centrations are low (about 2 g/kg DM), but availability
becomes 'fixed' in the soil.*

probably falls rapidly as concentrations rise to 3 g/kg

There is virtually no risk of overdosing with cobalt,

DM if cattle share the characteristics of sheep (Suttle,

but this should not be used as an excuse to overfeed Co

1983a). High iron (Fe) concentrations also lower Cu

to animals whose capacity to synthesize and absorb B12

status in cattle (Bremner et al., 1987; Phillippo et al., is strictly limited.

1987b; Gengelbach et al., 1997), are often raised in

spring and autumn pastures and have been implicated

in incidents of hypocuprosis (Jarvis & Austin, 1983;

Copper disorders

McFarlane et al., 1990). There is debate over whether

(see also Chapter 18)

or not the Fe ¥ Cu antagonism requires the presence of

moderate amounts of dietary S (Suttle, 1988).

Aetiology (see p. 254)

The principal determinants of hypocuprosis in

Most Cu-responsive disorders in cattle are induced:

grazing cattle are therefore raised soil Mo, alkaline soil

they are induced in the rumen where the anaerobic

pH (which encourages Mo uptake), sward immaturity,

degradation of sulphur(S)-rich, fibrous feeds leaves

high rainfall (or irrigation), heavy fertilizer N use and

little of the ingested Cu in a soluble form. After

high soil ingestion, rather than the Cu content of the

weaning, less than 10 per cent of the total Cu input is

soil or herbage, on all but the most impoverished, sandy

absorbed during passage down the gastrointestinal tract

soils.

and the percentage can be reduced to as little as 1–2 per

Shortage of absorbable Cu leads to (i) rapid deple-

cent by small increments in dietary Mo (Underwood &

tion of liver stores, (ii) a lowering of caeruloplasmin Suttle, 1999). Again it is transformations in the rumen

synthesis and a drop in plasma Cu (i.e. deficiency) and that are pivotal: extrapolating from sheep, it appears

(iii) a reduction in cuproenzyme activities in the ery-

that the progressive substitution of S²⁻ for O²⁻ in molyb-

throcyte and tissues (dysfunction). Although cytochrome c oxidase creates thiomolybdates, which complex Cu and

oxidase and superoxide dismutase have been the most

bind it to the solid phase. Surplus trithiomolybdate

studied, others such as lysyl oxidase and dopamine

(MoOS₃) is absorbable and capable of changing the dis-

hydroxylase will almost certainly follow the non-

tribution of Cu in the blood and tissues (Underwood &

ruminant pattern in showing disorder (Suttle, 1988).

Suttle, 1999). Just how important the systemic effects of

Effects on growth, cardiac function and bone develop-

thiomolybdates are under normal grazing conditions

ment cannot be attributed to a particular enzyme defi-

has yet to be determined. The 'tell-tale' appearance of

ciency with certainty. Even the depigmentation once

abnormal plasma fractions, such as TCA-insoluble Cu,

attributed to diminished tyrosinase activity must be

has been restricted to experimental situations, involv-

open to other explanations now that it is known to be

ing high levels of dietary Mo (35 mg/kg DM; Wang et al., produced by Co as well as Cu deprivation (Judson et al., 1988), and they may serve as slow release pools of Cu

1982). Connective tissue defects, including those in the
in the long term (Underwood & Suttle, 1999).

ligamentum nuchae, are probably due to lysyl oxidase

Most cases of Cu-responsive growth retardation in
deficiency leading to defective cross-links in elastin.

cattle are associated with abnormally low ratios of

Digestive disturbances may result from disruption of

Cu : Mo in the herbage (<3.0; Phillippo, 1983) and it has

mitochondrial respiration and villous atrophy, but

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enhanced inflammatory reactions to gut parasites

the clinical signs, like the signs themselves, are non-

cannot be ruled out in grazing animals (Underwood &

specific. The earliest and most dramatic lesions are

Suttle, 1999).

likely to be those affecting the epiphyseal growth plates

in the costochondral junctions and metatarsal/

metacarpal bones. These can become overgrown to the

Clinical signs

point that they spill over to leave islets of collagen distal

The sequence and severity of signs associated with a

to the plate in various states of irregular calcification
lack of dietary Cu in cattle (hypocuprosis) depend upon
and fibrotic replacement (Suttle, 1988). Gross degener-
the rate and stage of development at which it occurs.
ative changes are also seen in connective tissues such as
There are no definitive reports of teratogenic or neu-
the ligamentum nuchae. In severe cases showing
rological effects, comparable with swayback in sheep
diarrhoea, villous atrophy in the duodenal and jejunal
(cf. Richards & Edwards, 1986). In the growing calf, loss
regions is likely to be seen (Suttle, 1988) but is
of hair colour, growth retardation and changes in
indistinguishable from that caused by gut parasites.
metatarsal conformation are the earliest signs of abnor-
The anaemia of Cu deficiency in the bovine is
mality followed by diarrhoea and finally anaemia.
similar to that of Fe deficiency, i.e. macrocytic and
Black hair usually turns grey and red/brown hair
hypochromic. Cardiac lesions including fibrosis of the
becomes light brown. The 'foxy brown' discoloration of
myocardium have been reported in natural outbreaks

the Friesian is not attributable to copper deprivation only once, over 40 years ago, in Australia. While cardiac (Mee, 1991). Cardiac hypertrophy has been reported in hypertrophy was seen in some experimentally depleted experimentally depleted calves and this may be an early calves, which took a long time to develop clinical signs; manifestation of the myocardial degeneration that can it was not present in others that were depleted more cause sudden death after prolonged deficiency in the rapidly (Suttle, 1988).

field (Suttle, 1988). Addition of small amounts of the Cu Attention switched from the lesions that underlie antagonist, molybdenum (Mo), to the diet (2 mg/kg the expected and familiar clinical signs to those which, DM) accelerates rather than changes these clinical though less spectacular, occur earlier and may underlie manifestations of disorder. At much higher Mo concentrations (>10 mg/kg DM), animals may develop diarrhoea (Fell et al., 1985; Fell, 1987) found basement membrane immediately upon exposure to the antagonist

brane defects in the pancreatic acinar cell, the duodenal (Suttle, 1988).

enterocyte and kidney tubule, pointing to perhaps a

Infertility has long been associated with Cu deprivation in cattle but there is little published evidence that

common early failure of proteoglycan organization.

It remains to be seen whether Mo can cause distinct

deficiency leads to impaired reproduction unless Mo is

tive histological or ultrastructural changes at central

involved (Phillippo et al., 1982, 1987a). Molybdenum-

sites such as the pineal body which may be crucial to

induced deficiency is associated with delays in the onset

the development of disorder (Phillippo et al., 1987a)

of oestrus, impaired conception rate and anoestrus:

and whether or not these are Cu-dependent.

these abnormalities have yet to be induced experimen-

tally by other antagonists of Cu, such as Fe (Phillippo

Diagnosis

et al., 1987a). Copper-responsive infertility in cattle given Mo, therefore, may result from the direct or indirect suppression of the release of luteinizing hormone

Since none of the clinical signs of Cu deprivation given

above is specific, diagnosis must be supported by (Phillippo et al., 1987a).

biochemical tests showing subnormal tissue Cu status.

Anaemia is a late sign of Cu dysfunction in growing

Because availability has such a major effect on Cu calves (Suttle, 1988) and may be associated with Heinz uptake, herbage or dietary Cu concentrations alone are body formation in the periparturient cow (Black, 1981).

almost worthless and should be accompanied by meas-

The in vitro viability and/or function of neutrophils ures of Mo and S and predictions of available Cu

from Cu-deficient cattle is poorer than that of cells from

(Suttle, 1983a). In green swards, which contain enough

normal cattle (Suttle, 1988) but exposure to Mo can sulphur to potentiate the Mo antagonism, Cu : Mo ratios increase leucocyte counts (Gengelbach et al., 1997);

<3.0 indicate a risk of hypocuprosis. Even then, other there is no evidence yet for impaired resistance to infec- factors such as initial Cu reserves, other dietary antag- tions in vivo (Arthington et al., 1996).

onists (Fe and ingested soil) and the rate of animal pro-

duction may determine the outcome of events. Genetic variation in bovine Cu metabolism is far less

Necropsy

pronounced than that found in sheep (Underwood &

The pathology of bovine hypocuprosis is largely

Suttle, 1999). Fe : Cu ratios in the range 50–100 indicate

unhelpful because the histological changes underlying

the possibility of a Cu-responsive disorder; these and

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other guidelines for the biochemical assessment of risk

viding protection for several months (Judson et al.,

are summarized in Table 21.1.

1985; Rogers & Poole, 1988). Soluble glass boluses are

The conventional criteria of Cu status in the animal,

likely in time to give equally sustained protection and

i.e. liver and blood Cu concentrations, are most depend-

are particularly suited to the extensive grazing situation

able when herbage Mo concentrations are low (<5 mg/

(Allen et al., 1985; Judson et al., 1985; Givens et al., kg DM). Under these circumstances the ranges

1988). During periods of supplementary feeding or

150–300 mmol (10–20 mg) Cu/kg DM for liver and 3–9 mmol (0.2–0.6 mg) Cu/l for plasma can be regarded as supplements commends itself. Where food supplements ‘marginal’, except for the newborn (Table 21.1): as group are not given, a free-access mineral mixture containing means, they indicate that some individuals are likely to >500 mg Cu/kg is likely to afford protection to the benefit from Cu supplementation and that cheap measures to improve Cu status would be prudent. Newborn herd. If Cu proves to be useful as an antidote to Mo calves require higher marginal ranges for liver (790–3150 mmol Cu/kg DM) and lower ranges for plasma rendering toxic thiomolybdates less available, the provision of Cu by slow rumenal release or steady dietary (3.0–4.5 mmol Cu/l). At very high dietary Mo concentrations (35 mg/kg DM), changes in the distribution of Cu in supplementation may have advantages over other the plasma and liver may complicate the assessment

forms of supplementation. The use of chelated forms of Cu status (Wang et al., 1988). With less extreme copper, such as 'proteinates' or copper lysine, as dietary though still unusual Mo concentrations (>5 mg/kg DM) supplements has given no consistent advantage over in pasture, 'teart'-like conditions may operate for simple, cheap inorganic forms and cannot be relied critical periods, with animals responding to Cu upon to 'protect' copper from molybdenum or other while normocupraemic.

Low caeruloplasmin : Cu

antagonists (Underwood & Suttle, 1999).

ratios in plasma have been claimed to indicate the antagonistic influence of Mo, but they can be low when

Toxicity (see p. 948)

the antagonist is Fe (Gengelbach et al., 1997). The relationship between caeruloplasmin and total Cu in bovine

Copper poisoning rarely occurs through the ingestion of plasma is not perturbed in molybdeniferous areas of excess Cu in weaned cattle because of poor absorption Scotland (N.F. Suttle & J. Small, unpublished data). It

and a well-developed capacity to excrete surplus Cu via remains to be seen whether supplements that achieve the bile. Tolerance of diets containing >250 mg Cu/kg normocupraemia invariably provide a sufficient DM for several months has been reported in Friesian defence against Mo-induced infertility and whether steers (Felsman et al., 1973). Much higher absorptive other parameters such as plasma Mo or plasma Fe concentrations prior to weaning mean that calves reared on milk substitutes are relatively vulnerable, but they health (Phillippo et al., 1987a, b). In establishing Cu as a can tolerate up to 500 mg Cu/kg DM for brief periods (6 limiting factor to production in a new area, a response to weeks; Jenkins & Hidiroglou, 1989). Oral drenching with a specific Cu treatment affords the best assessment, CuSO₄ solutions can cause haemolytic crises similar to though not necessarily showing Cu deprivation to be those found in the more vulnerable sheep, but slower the primary cause (see p. 254). rates of exposure via the diet may sometimes cause or

contribute to hepatic crises. Rapidly growing bull calves can die suddenly and show biochemical evidence of liver

Treatment or prevention

injury, in the form of raised AST activities, and Cu over-

The treatment of Cu deficiency is achieved readily by

load, in the form of raised kidney Cu levels, without evi-

single oral doses of Cu or parenteral injections of the

dence of haemolysis (no jaundice or increase in kidney

element. In animals close to market weight the use of

iron) and only marginally raised liver Cu (C. Low, pers.

chelates of Cu with ethylene diamine, tetra-acetic acid

comm.). Diagnosis of toxicity becomes possible when

(EDTA), glycine or methionine may result in unac-

‘liver enzyme’ values in serum are raised and values for

ceptable, ‘cold’ abscesses at the injection site, whether

indices of copper status enter the following ‘marginal’

given subcutaneously or intramuscularly. Use of

ranges: serum, 0.018–0.020 mmol Cu/l; liver 6.4–16.0,

water-soluble complexes such as the hydroxyquinoline

kidney 0.6–0.8 and diet 1.57–3.14 mmol Cu/kg DM (mul-

sulphonate and heptonate will avoid abscess formation

tiply by 63.5 to obtain mg or ppm units). False 'highs' for but increase risks of acute toxicity (e.g. Suttle, 1981). plasma Cu during acute infections will be accompanied Prevention of deficiency and disorder can be by Cu : Zn ratios >3–4. Acute Cu poisoning can be caused achieved to varying degrees of precision and duration by overdosage via the parenteral route (Mylrea & with oral and parenteral supplements. Copper oxide Byrne, 1974) and is characterized by haemolytic crises particles are more effective than serial injections, pro- and jaundice.

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Iodine disorders (see pp. 257, 586)

there is no need to weigh the gland. Problems of interpretation arise when the degree of iodine deprivation

Aetiology

and associated changes are less pronounced. In the UK, the limit of normality for thyroid weight was 0.33 g

Iodine has only one function in the body, as a constituent of the hormone tri-iodothyronine (T3), which

shown that a higher margin is necessary. Iodine deprivation regulates basal metabolic rate. The hormone is formed by the removal of iodine (deiodination) from thyroxine without causing stillbirths (McCoy et al., 1997) and (tetra-iodothyronine, T₄), the physiologically inactive iodine supplementation of beef cows significantly form synthesized in the thyroid gland. Disorders can reduced thyroid weight without reducing calf mortality arise either from a simple lack of iodine in the diet or or stillbirths (Smyth et al., 1992). Sound interpretation from the impairment of iodine metabolism by ingested requires a marginal band of 0.3–0.8 g/kg LW (Table goitrogens. Simple iodine deprivation rarely occurs in 21.1). Spurious increases in thyroid weight may occur coastal regions or on small islands because of the large during the trauma of a difficult calving as part of a gen-atmospheric inputs of iodine of marine origin. Diets eralized oedema. containing <0.1 mg I/kg DM are inadequate, those with 0.1–0.2 mg/kg DM marginal; levels are usually much higher in spring than in summer pasture (Underwood

Diagnosis

& Suttle, 1999). In areas where winter temperatures are Biochemical and histological criteria can contribute to low, iodine requirements probably increase in proportion to energy expenditure/kg LW, making the newborn commonly used criteria present the greatest problems particularly vulnerable. Selenium deprivation may of interpretation.

increase the risk of iodine disorders through its role as an activator of several deiodinase enzymes (see Thyroxine: T4 is transported in plasma bound to proteins, but concentrations in the blood are a poor and 'Selenium disorders' on p. 302).

Ingestion of inorganic, cyanogenetic goitrogens in unreliable index of functional iodine status. Proper

certain grasses (e.g. Paspalum spp., Cynodon aethiopi-assessment of iodine dysfunction requires measure-

cus), legumes (e.g. Trifolium repens, Medicago sativa), meats of T3, T3 : T4 ratios and thyroid-stimulating

brassicac (e.g. B. oleracea) and by-products (e.g. cassava hormone (TSH), the hormone secreted by the pituitary

meal) leads to the formation of thiocyanate in the
in response to iodine deprivation. The detection of
rumen which impairs iodine uptake by the thyroid.
raised TSH concentrations in the bloodstream indicates

Organic,

thiouracil-type goitrogens (e.g.

in B.

that an animal needs more T3 and is prompting the

campestris, *Leucena leucophela* and rapeseed meals) thyroid gland to increase
hormone synthesis. A recent

can impair iodine incorporation into T4 in the thyroid
experiment (McCoy et al., 1997) confirms the unrelia-
and its conversion to T3 in the tissues. Exposure to both
bility of T4 assays. Pregnant heifers were fed a diet suf-
types of goitrogen increases risk of disorder and only
ficiently low in iodine to cause a 2.5-fold increase in
the former type can be countered by providing more
thyroid weight of their newborn calves when compared
iodine.

with an iodine supplemented control group. No signifi-
cant differences in plasma T4 concentrations were found

Clinical signs

between the two groups in either mother or offspring and mean values (60–202 nmol/l) were in the normal range. Iodine deprivation is no longer synonymous with goitre. There is also a spontaneous fall in plasma T4. While goitre in the newborn remains the commonest during early lactation to values that are subnormal for manifestation, lack of iodine also has a specific effect on other stages (20–40 nmol/l).

brain development and non-specific effects on growth, fertility, milk yield and on the skin and its appendages

Serum iodine: The element has been measured in toto (Underwood & Suttle, 1999).

and in protein-precipitable and butanol-extractable forms in serum, but rarely in proven clinical or sub-clinical cases of iodine deprivation, and diagnostic

Necropsy

limits can only provisionally be set. Each parameter is. Thyroid enlargement occurs as an adaptive response to a better measure of the degree of excess than of inadequate iodine deprivation and undoubtedly provides a simple

equate iodine supply (Table 21.1).

first measure of the possibility of iodine deficiency. In clinically significant, chronic pre- or postnatal iodine

Thyroid iodine: Thyroid iodine concentrations largely deprivation, enlargement of the thyroid is gross and reflect the amount of iodine in thyroglobulin which is

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*lying in store. Since iodine stores can be depleted microflora in isolated populations can limit endogenous before cellular changes occur and cellular changes goitrogen synthesis from mimosine-containing shrubs increase the size of the thyroid, tissue iodine concen- such as *L. leucophela* (Jones & Meggarity, 1983).*

tration alone will be of limited value in determining the total amount of iodine present in the gland. Many authorities use the yardstick proposed in the 1930s that Toxicity (see pp. 261, 823)

clinical goitre is associated with thyroid I <

2

1.2 g/kg DM

Iodine is a cumulative poison which eventually sup-

and 1.2–2.0 g I/kg DM becomes a marginal range (Puls, 1994; Table 21.1).

a multiple of daily intake \times length of exposure, rather than a set dietary concentration. The marginally toler-

Milk iodine: Concentrations of iodine in bovine milk
able concentration over three to four months' exposure
are linearly related to iodine intake (Underwood &

is 25–50 mg I/kg DM (Newton et al., 1974) and is rarely Suttle, 1999) and have proven diagnostic value in the

encountered under farm conditions unless there is
ewe. Puls (1994) suggests that values <25 mg I/l indicate
access to seaweed (4–6 g I/kg DM). Iodosis is charac-
'deficiency' in cows, much lower than the corresponding
terized by poor growth and respiratory disorder in
value for ewes (<80), yet this probably overlaps the
calves (Newton et al., 1974) and by perinatal mortality
'marginal' range.

in offspring of the adult cow (Fish & Swanson, 1983).

Once lactation has begun, the cow herself becomes less

Histological criteria: A sequence of microscopic cellu-vulnerable because she is able to secrete much of any

lar changes occur in the thyroid as the organ responds to the stimulus from TSH. Firstly, stores of surplus thyroxine in the form of ethylene diamine dihydroiodide (EDDI) for treating foot rot and 'lumpy jaw' in the USA has resulted in many cases of iodosis (see page 261 and Underwood & Suttle, 1999, for further details). With stores of thyroglobulin denuded, the epithelial cells which produce thyroxine increase first in number threatening stage, rather than depletion is complete.

With stores of thyroglobulin denuded, the epithelial cells which produce thyroxine increase first in number

Selenium disorders (see p. 258)

(hyperplasia: 'hyperplastic goitre') and then in size (hypertrophy); it is the latter process which lies behind

Aetiology

visible, clinically significant goitre. Hyperplasia can

occur in calves which are normal at birth (McCoy et al., The principal function of Se in animals relates to its

1997) and may be caused by the stress of a prolonged

intracellular presence in the glutathione peroxidases, calving. Undue diagnostic significance should not be a family of antioxidant enzymes (once GSHPx, now attached to thyroid abnormalities, although these may GPXn with $n \geq 6$), which use various potentially dangerous peroxides as substrates. Selenium has been linked with iodine metabolism in ways that indicate a separate function, independent of GPX (Arthur et al., Treatment and prevention 1988). While the basic cause of Se disorders is obviously Congenital goitre must be prevented because there is a shortage of the element in the diet, which in turn no response to treatment and only the chronic forms of can be traced to soil formations also deficient in Se iodine disorders in older animals are amenable to treatment (Underwood & Suttle, 1999), other factors are probably involved in field outbreaks of Se-responsive disease. be prevented by providing inorganic iodine supplement. Calves fed experimental diets of exceedingly low Se

ments in iodized salt licks or mineral mixes containing content (0.01–0.02 mg/kg DM) indoors can be depleted

c. 0.1 g I/kg. Substantial losses can occur by volatilization of Se to the point that they have undetectable activities

tion in hot climates and the use of less volatile forms

of GPX1 in their blood and yet do not develop myo-

such as periodates is then indicated. Slow-release

pathy (Arthur, 1988; Reffett et al., 1988). In one study boluses are being developed and a long-lasting form for

(Arthur, 1988), the failure to induce myopathy was the

parenteral administration (iodized poppyseed oil) has

more surprising because the diet was also low in vitamin

long been used, although it is no longer obtainable in

E, which also contributes to antioxidant defence in the

some countries due to a failure to update product

tissues.

licences. Problems induced by organic goitrogens can

The importance of dietary sources of oxidant stress

be avoided by using cultivars selected for low glucosi-

was demonstrated by workers in Belfast (Rice &

nolate content and by-products of such crops (e.g.

McMurray, 1982), who showed that the addition of

canola rapeseed meals). Manipulation of the rumen polyunsaturated fatty acids (PUFA, progenitors of lipid

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peroxides) to diets deficient in vitamin E and Se, simulations have not always been confirmed in experi-

lating the composition of PUFA-rich spring grass,

mental studies (Reffett et al., 1988). There are several will precipitate myopathy. Other stressors may yet be

reports of inverse relationships between blood Se or

required to induce myopathy because Arthur (1988)

glutathione peroxidase (GPX) activity with somatic

has shown that feeding of PUFA-rich grass indoors to

cell counts in bovine milk (e.g. Erskine et al., 1989) and calves deficient in Se and vitamin E did not precipitate

experimental evidence that addition of Se to diets of

the disorder, whereas grazing the same grass produced

marginal Se concentration decreases the duration of

acute myopathy. He suggested that exercise or some

clinical mastitis (Smith et al., 1984). Similar responses other component of the environmental change at

can be obtained with supplements of vitamin E (Weiss

turnout contributed to the development of muscle

et al., 1997), making interpretation of responses to Se damage. The excitable behaviour of the calf will of

combined with vitamin E (the commonest approach)

course result in increased activity of muscles involved

either difficult or impossible. Improvements in calf

in locomotion, circulation and respiration: muscular

survival in herds on diets of marginal Se concentra-

exercise is known to induce oxidant stress and exacer-

tion (0.03–0.05 kg Se/kg DM) have been reported, but

bate the effects of vitamin E deficiency (Jackson, 1987).

vitamin E as well as Se was given and the role of Se

It is highly likely that the intense and sometimes pro-

alone is again unclear (Spears et al., 1986).

longed muscular activity of the uterus at parturition

makes it particularly vulnerable to Se (and vitamin E)

Diagnosis

deficiency. Adverse weather conditions have been

implicated in outbreaks of acute myopathy and the

The differential diagnosis of Se-responsive disorders is

cause of Se-responsive disorders is thus multifactorial

complicated by the non-specific clinical signs of disease:

(Underwood & Suttle, 1999).

most can also be attributed to vitamin E deficiency while others, such as retained placenta and growth retardation, have many possible causes of both nutri-

Clinical signs

tional and non-nutritional origin. The tolerance of low Selenium deprivation in cattle can impair development plasma and blood Se (or GPX1) concentrations in the throughout life. The suckling calf can develop chronic absence of exacerbative factors confirms that measures skeletal and cardiac myopathy if the dam's diet is low of Se (or GPX) alone cannot confirm a selenium dis-

in Se (Hidiroglou et al., 1985), while the calf on pastures order. Neither is there likely to be a simple relation-deficient in Se can suffer growth retardation before ship between dietary Se concentration and incidence of (Morris et al., 1984) and after weaning (Gleed et al., disease.

1983). In spring, calves can develop acute myopathy

In monitoring Se status, the assay of GPX once with myoglobinuria when turned out to graze. Selenium largely replaced that of whole blood Se because it was supplementation has improved conception rate in cows

easier to assay, yet highly correlated with blood Se con-

(McClure et al., 1986) and heifers (MacPherson et al., concentration. Enzyme activities were converted to blood

1987b). At parturition, cows of low Se status are more

Se equivalents for comparative purposes. Interpreta-

likely to retain the placenta than Se-supplemented cows

tion of blood GPX activities is, however, complicated

and they are the more susceptible to metritis and cystic

by many factors. The measurement of GPX activity

ovaries (Harrison et al., 1984). Selenium deprivation

is subject to wider interlaboratory variation than most

also affects the circulatory system: the growing calf

analyses in the clinical context: Blanchflower et al.

can develop a Heinz-body anaemia (Morris et al., 1984) (1986) identified some important variables. The use of

and when calving coincides with turnout, the cow

an assay kit helped to standardize results, but dif-

which is both Cu and Se deficient may be vulnerable

ferences can still arise and each laboratory should

to haemolysis and haemoglobinuria (Black, 1981).

determine and quote its own Se equivalence for GPX

Despite the variety of clinical signs caused by a lack

activity. Results are variously reported per ml blood, of Se, it is rare for more than one sign to appear in a per g haemoglobin and per ml cells: while these will be single herd. Problems of muscular dystrophy have not highly correlated with each other within laboratories, been reported in the many studies of retained placenta, the use of common conversion factors for comparative which is probably the commonest Se-responsive con- purposes may introduce errors. The slow turnover of dition in cattle. Similarly, growth retardation and mus- erythrocytes ensures that blood GPX activity reflects cular dystrophy have not been reported in the same past rather than present Se supply. The data of outbreak of disorder. Selenium deficiency in cattle has Hidiroglou et al. (1985) indicate that it may take four been associated with impaired phagocyte function in months for blood GPX (and Se) fully to reflect an vitro, prompting speculation that Se deficiency would improved Se supply and just as long to reflect a waning lead to decreased resistance to disease, but these sug- supply. It should be noted that the GPX in blood is now

regarded as a dispensable, storage form of the element oxidative stress but has yet to be tested in ruminants and indicative of depletion rather than dysfunction. and would not be a specific test for Se (Suttle, 2000). As Plasma Se responds immediately to changes in dietary with other trace elements, the surest diagnosis is often supply and is a useful adjunct to blood GPX in assessment provided by responsiveness to effective Se supplementing Se status concentrations of 100–120 nmol (8–10 mg/l) ments. By plotting a measure of final performance being ‘marginal’ and suggesting possible benefits from against performance prior to supplementation (e.g. Se supplementation (Underwood & Suttle, 1999). LWG), it is sometimes possible to identify a small Very small differences in Se status may determine minority of a population which would benefit from vulnerability. In the study of Hidiroglou et al. (1985) for additional Se, while mean performance remains example, myopathy occurred in calves in one year when unchanged (Suttle, 2000).

blood GPX1 declined from 29 to 19 iu/g haemoglobin

(Hb) and plasma Se from 11 to 6 mg/l, but not in the next

Treatment or prevention

when the respective falls were 51 to 28 iu/g Hb

and 16 to 13 mg/l. Pasture Se was consistently low at

The many different methods for administering Se orally

0.02–0.04 mg/kg DM. In a survey of the Se status of

and parenterally have been reviewed (Allen, 1983;

cattle in north-east Scotland (Arthur et al., 1979), 85 per MacPherson, 1983)
and three were compared by

cent of herds had a blood Se status 50 mg/l, a threshold

MacPherson and Chalmers (1984). Long-term supple-

below which a risk of clinical disease was once sug-

mentation can be achieved by both routes. By adminis-

gested, but only 10 per cent had a recent history of

tering heavy metal (iron oxide) (e.g. Hidioglou et al., myopathy; the winter diets
mostly contained <0.05 mg

1985; McClure et al., 1986) or soluble glass boluses

Se/kg DM. In New South Wales, herds of growing cat-

(Judson et al., 1985) orally and by injecting a relatively tle with blood Se
concentrations usually <20 mg

insoluble Se salt, barium selenate, in an oily base

(0.25 mmol)/l rarely benefited from Se supplementation (MacPherson & Chalmers, 1984; MacPherson et al., 1987b), blood Se concentrations have been increased (Langlands et al., 1981). Studies in New Zealand with dairy cows indicated a critical blood Se concentration as low as 12 mg/l below which milk fat yield fell (Fraser et al., 1987). The latter studies (Whelan et al., 1994) or the winter diet (Stowe et al., 1988) with Se can also provide long-term protection.

vitamin E.

Stowe et al. (1988) concluded that a supplement of 2 mg The clinically significant thresholds for dietary and

Se/day upon an estimated basal intake of 0.51 mg/day blood Se concentrations probably vary with the Se- was not sufficient to raise plasma Se in the periparturient cow to 'acceptable levels' (>60 mg/l); nevertheless E. Incidence of retained placenta can be reduced in

retained placenta and metritis was only half as prevalent in cows receiving diets of higher Se content (0.04–0.05 mg/kg DM) than those giving freedom from myopathy. Oral supplementation prior to calving (13–45.5 mg/day for 15 days) can raise the Se status of offspring substantially and for concentrations of 50 mg/l may sometimes be needed for several months (Enjalbert et al., 1999). Use of organic forms of selenium such as yeasts or selenomethionine (McClure et al., 1986; Underwood & Suttle, 1999), but studies with grazing heifers in New Zealand show possible tolerance of much lower values or adult cattle and increase the transfer of selenium to milk when compared to inorganic sources but is used (Table 21.1). Marginal bands must therefore be milk when compared to inorganic sources but is unlikely have any therapeutic, nutritional or economic advantage in the treated animal (Underwood & Suttle, 1999). The greatest limitation of blood GPX for monitoring Se deficiency is that the target tissue in Se deficiency is often

1999).

muscle not blood. Furthermore, there are wide variations between muscles in their GPX content and in the lipid composition of their membranes (Rice & Kennedy, 1988). Measurement of blood GPX alone is usually short-term problems. The Se-responsive diseases associated with parturition and turnout are acute and unlikely to reflect the risk of lipid peroxidation and conditions that can be effectively treated or prevented hence myopathy in crucial muscles. Guidelines for the by single injections of Se as selenite or selenate (e.g. interpretation of selected indices of Se status are given Eger et al., 1985; McMurray & McEldowney, 1977). in Table 21.1. The assay of a peroxidation product in There is considerable variation in dosage practice. For accessible membranes (e.g. malondialdehyde in erythrocytes) may provide an integrated measure of net doses have varied from 2.3 to 50 mg: the highest dose

rate, equivalent to 0.1 mg/kg liveweight, is the most species from pastures, treatment of soils with fertilizers commonly used.

which lower selenium uptake (ammonium and calcium sulphates) by plants, rotational grazing with less susceptible species such as sheep and improved soil drainage. Oral administration of sulphate-rich mixtures limited studies in which only one nutrient was given may prevent or alleviate signs (Arora et al., 1975) to some animals show that the benefit of providing vitamin E with Se varies from farm to farm. The need for dual supplementation may also depend on the

Manganese and zinc disorders

nature of the clinical problem. A combination of oral (see p. 260)

vitamin E with parenteral Se reduced the incidence of retained placenta in one instance in which the separate

Cattle deprived of elements such as Mn and Zn under treatments were ineffective, but Se alone reduced the experimental conditions develop diverse clinical symptoms, but the diets used contained far less Mn and Zn et al., 1984). In the treatment of acute myopathy at than the levels provided by the vast majority of natural turnout, McMurray and McEldowney (1977) found that diets (Underwood & Suttle, 1999). Field cases of Mn vitamin E (2.8 mg/kg liveweight) was less effective than and Zn deprivation would require either an abundance Se (0.0625 mg/kg liveweight) and added nothing when of dietary antagonists which impair the utilization of given with Se. In this situation cattle of low vitamin E the element or other factors which massively increase status were turned onto grass of naturally high vitamin animal needs for Mn and Zn. There is a possibility that E concentration. The variable responses to vitamin E 'protection' of protein sources from rumen degradation given with Se will be due largely to the wide variations might allow phytate to escape and lower absorption of

found in the vitamin E status of housed cattle.

both elements and that fine grinding of cereals might have a similar end result (Suttle, 2000). In the field, soil Selenium toxicity (see p. 943)

ingestion provides a rich adventitious source of manganese while access to galvanized feed and drinking Disorders occur naturally either when pastures contain troughs provides additional Zn, further reducing the plant species (e.g. Astragalus) which accumulate selenium risk of disorders. Little attention has therefore been given to reliable assessment of Mn and Zn status in the when a combination of selenium-rich soil and high pH animal. Serum Mn and Zn concentrations are probably allows high levels of selenium to accumulate in normal reasonable indices of deficiency (Table 21.1), but marginal plant species ('Alkali disease') (O'Toole & Raisbeck, 1995). Accidental toxicity can be caused by errors in the former; liver values are poor indicators of status for adding the small amounts of selenium needed to sup-

both Mn and Zn (Underwood & Suttle, 1999). Assessment of hypozincaemia is complicated by the fall in sources and multiple doses (e.g. Se-supplemented values which accompanies the acute phase of microbial anthelmintics) and failure to scale oral drenches or parenteral injections carefully to liveweight. Because selecting results for samples with raised haptoglobin or iron is well absorbed, the oral route poses almost as big a threat as the parenteral route. Selenium is a cumulative threat with Mn and Zn supplements is rarely necessary and use of expensive chelated sources is unlikely concentrations decrease as duration of exposure into improve animal status or performance any more creases. Lesions occur principally in the hoof, which than inorganic Mn or Zn sources (Olson et al., 1999; Underwood & Suttle, 1999). Toxicities of Mn or Zn

severe, debilitating lameness. The disorder can be dis-
rarely occur.

tinguished from laminitis (see p. 420) by the fact that
the lesions are epidermal rather than dermal (O'Toole
& Raisbeck, 1995). The marginal ranges for indices of

References

selenium status within which toxicosis becomes a
possible diagnosis are as follows: diet, 50–75; liver,
Allen, W.M. (1983) Parenteral methods of trace element
30–50; hoof, 25–63 and hair 63–126 mmol Se/kg DM
supplementation. British Society of Animal Production
(Underwood & Suttle, 1999; multiply by 79 to obtain
Occasional Publication No. 7, pp. 87–92.

values in mg): the corresponding range for blood is

Allen, W.M., Drake, C.F. & Tripp, M. (1985) Use of controlled 25–30 mmol Se/l
(McLaughlin et al., 1989). Preventive

release systems for supplementation during trace element

measures include the removal of 'Se-accumulator'

deficiency – the administration of boluses of controlled

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release glass to cattle and sheep. In Proceedings of Fifth on performance and
certain blood constituents of calves.

International Symposium on Trace Element Metabolism

Journal of Animal Science, **36**, 157–60.

in Man and Animals (ed. by C.F. Mills, I. Bremner & Fish, R.E. & Swanson, E.W. (1983) *Effects of excessive iodide* J.K. Chesters), pp. 719–22.
Commonwealth Agricultural

administered in the dry period on thyroid function and

Bureaux, Farnham Royal.

health of dairy cows and their calves in the peri-parturient

Arora, S.P., Parvinder, K., Khirwar, S.S., Copra, R.C. & Ludri, period. *Journal of Animal Science*, **56**, 162–72.

R.S. (1975) *Selenium levels in fodders and its relationship*

Fraser, A.J., Ryan, T.J., Sproule, R., Clark, R.G., Anderson, D.

with Degnala disease. Indian Journal of Dairy Science, **28**,

& Pederson, E.O. (1987) *The effect of selenium on milk pro-249.*

duction in dairy cattle. Proceedings of the New Zealand

Arthington, J.D., Corah, L.R. & Blecha, F. (1996) *The effect of Society of Animal Production*, **47**, 61–4.

molybdenum induced copper deficiency on acute phase

Gengelbach, G.P., Ward, J.D., Spears, J.W. & Brown, T.T. (1997) *protein concentrations, superoxide dismutase activity,*

Effects of copper deficiency coupled with high dietary iron

leucocyte numbers and lymphocyte proliferation in beef

or molybdenum on phagocytic function and response of

heifers inoculated with bovine herpesvirus-1. *Journal of*
calves to a respiratory disease challenge. Journal of Animal Animal Science, **74**,
 211–17.

Science, **75**, 1112–18.

Arthur, J.R. (1988) *Effects of selenium and vitamin E status*

Givens, D.I., Zervas, G., Simpson, V.R. & Telfer, S.B. (1988) *on plasma creatine
 kinase activity in calves. Journal of*

Use of soluble glass rumen boluses to provide a supplement

Nutrition, **118**, 747–55.

of copper for suckled calves. Journal of Agricultural Science, Arthur, J.R.,
 Morrice, P.C. & Beckett, G.J. (1988) *Thyroid Cambridge*, **110**, 119–204.

hormone concentrations in selenium-deficient and selenium

Gleed, P.T., Allen, W.M., Mallinson, C.B., Rowlands, G.J.,

sufficient cattle. Research in Veterinary Science, **45**, 122–3.

Sansom, B.F., Vagg, M.J. & Caswell, R.D. (1983) *Effects of Arthur, J.R., Price, J.
 & Mills, C.F. (1979) Observations on selenium and copper supplementation on
 the growth of*

the selenium status of cattle in the north-east of Scotland.

beef steers. Veterinary Record, **113**, 388–92.

Veterinary Record, **104**, 340–1.

Harrison, J.H., Hancock, D.D. & Conrad, H.R. (1984) *Vitamin Black, H. (1981)
 Post-parturient haemoglobinuria in NorthE and selenium for reproduction in the
 dairy cow. Journal*

land. Proceedings of the Sheep and Beef Cattle Society

of Dairy Science, **67**, 123–32.

of New Zealand Veterinary Association's 11th Seminar,

Hidioglou, M., Proulx, J. & Jolette, J. (1985) Intraruminal Massey University, Palmerston North, pp. 11–14.

selenium for control of nutritional muscular dystrophy in

Blanchflower, W.J., Rice, D.A. & Davidson, W.B. (1986) Blood cattle. *Journal of Dairy Science*, **68**, 57–66.

glutathione peroxidase: a method for measurement and

Jackson, M. (1987) Muscle damage during exercise; possible

the influence of storage, cyanide and Drabkin's reagent

role of free radicals and the protective effect of vitamin E.

on enzyme activity. Biological Trace Element Research, **11**, Proceedings of the Nutrition Society, **46**, 77–80.

89–100.

Jarvis, S.C. & Austin, A.R. (1983) Soil and plant factors limit-Bremner, I., Humphries, W.R., Phillippo, M., Walker, M.J. & ing the availability of copper to a beef suckler herd. *Journal Morrice*, P.C. (1987) Iron-induced copper deficiency in

of Agricultural Science, **101**, 39–46.

calves: dose-response relationships and interactions with

Jenkins, K.J. & Hidioglou, M. (1989) Tolerance of the calf for molybdenum and sulphur. *Animal Production*, **45**, 403–14.

excess copper in milk replacer. Journal of Dairy Science, **72**, Duncan, I.F., Greentree, P.L. & Ellis, K.J. (1986) Cobalt defi-150–6.

ciency in cattle. Australian Veterinary Journal, **3**, 127–8.

Jones, R.J. & Meggarity, R.G. (1983) Comparative responses Eger, S., Drori, D., Kadoori, I, Miller, N. & Schindler, H. (1985) of goats fed on *Leucena leucephala* in Australia and Hawaii.

Effects of selenium and vitamin E on incidence of retained

Australian Journal of Agricultural Research, **34**, 781–90.

placenta. Journal of Dairy Science, **68**, 2119–22.

Judson, G.L., Koh, T.-S., McFarlane, J.D., Turnbull, R.K. & Enjalbert, F., Lebreton, P., Salat, O. & Schelcher, F. (1999) Kempe, B.R. (1985) Copper and selenium supplements for

Effects of pre- or post-partum selenium supplementation on

cattle: evaluation of the selenium bullet, copper oxide and

selenium status in beef cows and their calves. Journal of

the soluble glass bullet. In Proceedings of the Fifth Interna-Animal Science, **77**, 223–9.

tional Symposium on Trace Element Metabolism in Man and

Erskine, R.J. Eberhart, R.J. & Hutchinson, L.J. (1989) *Blood Animals* (ed. by C.F. Mills, I. Bremner & J.K. Chesters), pp.

selenium concentrations and glutathione peroxidase activ-

725–8. Commonwealth Agricultural Bureaux, Farnham

ities in dairy herds with high and low somatic cell counts.

Royal.

Journal of American Veterinary Medical Association, **190**, Judson, J.G., McFarlane, J.D., Baumgurtel, K.L., Mitsioulis, A., 1417–21.

Nicolson, R.E. & Zviedrans, P. (1997a) *Indicators of vitamin* Fell, B.F. (1987) *The pathology of copper deficiency in animals.*

B12 status in cattle. In Trace Elements in Man and Animals In Copper in Animals and Man (ed. by J. McC. Howell &

– 9: Proceedings of the Ninth International Symposium (ed.

J.M. Gawthorne), pp. 1–28. CRC Press Ltd, Boca Raton,

by P.W.F. Fischer, M.R. Abbe, K.A. Cockell & R.S. Gibson), Florida.

pp. 310–11. NRC Research Press, Ottawa, Canada,

Fell, B.F., Farmer, L.J., Farquharson, C., Bremner, I. & Graca, Judson, J.G., McFarlane, J.D., Mitsioulis, A. & Zviedrans, P.

D.S. (1985) Observations on the pancreas of cattle deficient

(1997b) Vitamin B12 responses to cobalt pellets in beef cows.

in copper. Journal of Comparative Pathology, 95, 573–90.

Australian Veterinary Journal, 75, 660–2.

Felsman, R.J., Wise, M.B., Harvey, R.W. & Barrick, E.R. (1973) Judson, G.J., McFarlane, J.D., Riley, M.J., Milne, M.L. & Horne, Effect of graded levels of copper sulphate and antibiotic

A.C. (1981) Treatment of cobalt deficiency in calves. In Pro-

Trace Element Disorders • 307

ceedings of the Fourth International Symposium on Trace

calving performance of a trace element deficient dairy herd.

Element Metabolism in Man and Animals (ed. by J. McC.

Veterinary Record, 137, 508–12.

Howell, J.M. Gawthorne & C.L. White), pp. 191–4. Aus-

Millar, K.R., Alby, A.T. & Bond, G.C. (1984) Measurement

tralian Academy of Science, Canberra.

of vitamin B12 in the livers and sera of sheep and cattle and

Judson, G.L., McFarlane, J.D., Riley, M.J., Milne, M.L. &

an investigation of factors influencing serum vitamin B12

Horne, A.C. (1982) Vitamin B12 and copper supplementa-

levels in sheep. New Zealand Veterinary Journal, 32, 65–70.

tion in beef calves. Australian Veterinary Journal, 58, 249–52.

*Mitsioulis, A., Bansemer, P.C. & Koh, T.-S. (1995) Relationship Kennedy, D.G.,
Young, P.B., McCaughey, W.J., Kennedy, S. & between vitamin B12 and cobalt
concentrations in bovine*

Blanchflower, W.J. (1991) Rumen succinate production may

liver. Australian Veterinary Journal, 72, 70.

ameliorate the effects of cobalt-vitamin B12 deficiency on

Morris, J.G., Chapman, H.L., Walker, D.F., Armstrong, J.B.,

*methylmalonyl CoA mutase in sheep. Journal of Nutrition, Alexander, J.D.,
Miranda, R., Sanchez, A., Sanchez, B., Blair-121, 1236–42.*

West, J.R. & Denton, D.A. (1984) Selenium deficiency in

*Langlands, J.P., Wilkins, R.F., Bowles, J.E., Smith, A.J. & Webb, cattle
associated with Heinz body anaemia. Science, 223, R.F. (1981) Selenium
concentration in the blood of rumi-291–3.*

nants grazing in northern New South Wales. I. Analysis

*Mylrea, P.J. & Byrne, D.T. (1974) An outbreak of acute copper of samples
collected in the National Brucellosis Scheme.*

poisoning in cattle. Australian Veterinary Journal, 50, Australian Journal of

Agricultural Research, **32**, 511–21.

169–72.

McClure, T.J., Eamens, G.J. & Healy, P.J. (1986) Improved fer-Newton, G.L.,
Barrick, E.R., Harvey, R.W. & Wise, M.B. (1974) tility in dairy cows after
treatment with selenium pellets.

Iodine toxicity, physiological effects of elevated dietary

Australian Veterinary Journal, **63**, 144–6.

iodine on calves. Journal of Animal Science, **38**, 449–55.

McCoy, M.A., Smyth, J.A., Ellis, W.A., Arthur, J.R. & Kennedy, Olson, P.A.,
Brink, D.R., Hickok, D.T., Carlson, M.P., Schneider, D.G. (1997) *Experimental
reproduction of iodine deficiency*

N.R., Deutscher, G.H., Adams, D.C., Colburn, D.J. &

in cattle. Veterinary Record, **141**, 544–7.

Johnson, A.B. (1999) *Effects of supplementation of organic*

McCoy, M.A., Smyth, J.A., Ellis, W.A. & Kennedy, D.G. (1995) *and inorganic
combinations of copper, cobalt, manganese*

Parenteral iodine and selenium supplementation in still-

and zinc above nutrient requirement levels on post partum

birth perinatal/weak calf syndrome. Veterinary Record, **136**, two-year-old cows.
Journal of Animal Science, **77**, 522–32.

124–6.

O'Toole, D. & Raisbeck, M.F. (1995) *Pathology of experi-*

McDonald, P. & Suttle, N.F. (1986) *Abnormal fermentations*

mentally induced chronic selenosis (alkali disease) in year-

in continuous culture of rumen microorganisms given

ling cattle. Journal of Veterinary Diagnostic Investigation, 7, cobalt-deficient hay or barley as food substrates. British 364–73.

Journal of Nutrition, 56, 369–78.

Phillippo, M. (1983) The role of dose–response trials in pre-

McFarlane, J.D., Judson, J.D. & Gouzos, J. (1990) Copper

dicting trace element deficiency disorders. British Society of deficiency in ruminants in the South East of Australia.

Animal Production Occasional Publication No. 7, pp. 51–60.

Australian Journal of Experimental Agriculture, 30, 187–

Phillippo, M., Humphries, W.R., Atkinson, T., Henderson, G.D.

93.

& Garthwaite, P.H. (1987a) The effect of dietary molyb-

McLaughlin, J.G., Cullen, J. & Forristal, T. (1989) Blood

denum and iron on copper status, puberty, fertility and

selenium concentrations in cattle on seleniferous pastures.

oestrous cycles in cattle. Journal of Agricultural Science, Veterinary Record, 124, 426–7.

Cambridge, 109, 321–36.

McMurray, C.H. & McEldowney, P.K. (1977) A possible pro-

Phillippo, M., Humphries, W.R. & Garthwaite, P.H. (1987b)

phylaxis and model for nutritional degenerative myopathy

The effect of dietary molybdenum and iron on copper

in young cattle. British Veterinary Journal, 133, 535–42.

status and growth in cattle. Journal of Agricultural Science, MacPherson, A. (1983) Oral treatment of trace element deficiency-Cambridge, 109, 315–20.

deficiencies in ruminant livestock. British Society of Animal

Production Occasional Publication No. 7, pp. 93–106.

Phillippo, M., Humphries, W.R., Lawrence, C.B. & Price, J.

(1982) Investigation of the effects of copper status and

MacPherson, A. & Chalmers, J.S. (1984) Methods of selenium therapy on fertility in beef suckler herds. Journal of

supplementation of ruminants. Veterinary Record, 115, Agricultural Science, Cambridge, 99, 359–64.

544–6.

Puls, R. (1994) Mineral Levels in Animal Health, 2nd edn, p.

MacPherson, A., Gray, D., Mitchell, D.B.B. & Taylor, C.N.

120. Diagnostic data, Sherpa International, Clearbrook BC.

(1987a) Ostertagia infection and neutrophil function in Quirk, M.F. & Norton, B.W. (1988) Detection of cobalt

cobalt-deficient and cobalt-supplemented cattle. British

deficiency in lactating heifers and their calves. Journal

Veterinary Journal, 143, 348–53.

of Agricultural Science, Cambridge, 110, 465–70.

MacPherson, A., Kelly, E.F., Chalmers, J.S. & Roberts, D.J.

Reffett, J.K., Spears, J.W. & Brown, T.T. (1988) Effect of (1987b) The effect of selenium deficiency on fertility in

dietary selenium on the primary and secondary immune

heifers. In Proceedings of 21st Annual Conference on Trace response in calves challenged with infectious bovine rhino-Substances in Environmental Health

(ed.

by D.D.

tracheitis virus. *Journal of Nutrition*, **118**, 229.

Hemphill), pp. 551–5. University of Missouri.

Reid, T.C. & McQueen, T.P. (1985) Cobalt supplementation

Mee, J.F. (1991) Coat colour and copper deficiency in cattle.

for beef cattle. In Proceedings of Fifth International Sym-Veterinary Record, **129**, 536.

posium on Trace Element Metabolism in Man and Animals

Mee, J.F., Rogers, P.A.M. & O'Farrell, K.J. (1995) Effect of (ed. by C.F. Mills, I. Bremner & J.K. Chesters), pp. 739–41.

feeding a mineral–vitamin supplement before calving on the

Commonwealth Agricultural Bureaux, Farnham Royal.

308 • Chapter 21

Rice, D.A. & Kennedy, S. (1988) Vitamin E: functions

cattle to long-term and short-term supplementation with

and effects of deficiency. *British Veterinary Journal*, **144**, oral selenium and vitamin E. *Journal of Dairy Science*, **71**, 482–96.

1830–9.

Rice, D.A., O'Harte, F.P.M., Blanchflower, W.J. & Kennedy, Suttle, N.F. (1981) Comparison between parenterally administered D.G. (1989) Methylmalonic acid in the rumen of cobalt-

stered copper complexes of their ability to alleviate

deficient sheep and its effects on plasma methylmalonic

hypocupraemia in sheep and cattle. *Veterinary Record*, **109**, acid. *Proceedings of Nutrition Society*, **48**, 141A.

304–307.

Rice, D.A., McLoughlin, M., Blanchflower, W.J., Goodall, E.A.

Suttle, N.F. (1983a) Assessment of the mineral and trace

& McMurray, C.H. (1987) Methylmalonic acid as an indi-

element status of feeds. In *Proceedings of the Second Sym-*

cator of vitamin B12 deficiency in grazing sheep. Veterinary posium of the International Network of Feed Information

Record, **121**, 472–3.

Centres (ed. by G.E. Robards & R.G. Packham), p. 211.

Rice, D.A. & McMurray, C.H. (1982) Recent information on

Commonwealth Agricultural Bureaux, Farnham Royal.

vitamin E and selenium problems in ruminants. *Roche Sym-*

Suttle, N.F. (1983b) Effect of molybdenum concentration in

posium, Basle, Hoffman La Roche, pp. 1–19.

fresh herbage, hay and semi-purified diets on the copper

Richards, R.B. & Edwards, J.R. (1986) A progressive spinal metabolism of sheep. *Journal of Agricultural Science*,

myelinopathy in beef cattle. Veterinary Pathology, **23**, 35–41.

Cambridge, **100**, 651–6.

Rogers, P.A.M. & Poole, D.B.R. (1988) Copper oxide needles Suttle, N.F. (1988) *The role of comparative pathology in the*

for cattle: a comparison with parenteral treatment. Veteri-studies of copper and cobalt deficiencies in ruminants.

nary Record, **123**, 147–51.

Journal of Comparative Pathology, **99**, 242–57.

Sanson, R.L. (1990) Selenium supplementation of sheep by

Suttle, N.F. (1995) Relationship between vitamin B12 and

topdressing pastures under high rainfall conditions. New

cobalt concentrations in bovine liver. Australian Veterinary Zealand Veterinary Journal, **38**, 1–3.

Journal, **72**, 278.

Schulz, W. (1987a) Unsaturated vitamin B12 binding capacity

Suttle, N.F. (2000) Minerals in livestock production. In *Proin human and ruminant serum – a comparison of techniques*

ceedings of the Ninth Congress of The Asian–Australasian

including a new technique by high performance liquid chro-

Association of Animal Production Societies (ed by G. Stone) matography. Veterinary Clinical Pathology, **16**, 67–72.

Suppl. C, pp. 1–9.

Schulz, W. (1987b) A comparison of commercial kit methods

Suttle, N.F. (In press) Differential diagnosis of micronutri-

for assay of vitamin B12 in ruminant blood. *Veterinary*

ent-responsive disorders in beef suckler herds. *Cattle*

Clinical Pathology, **16**, 102–106.

Practice.

Smart, M.E., Cohen, R., Christensen, D.A. & Williams, C.M.

Suttle, N.F. & Sinclair, K. (2000) Suckler cow nutrition 2. Min-

(1986) The effects of sulphate removal from the drinking

erals and vitamins. *Cattle Practice*, **8**, 193–9.

water on liver copper and zinc concentrations of beef cows

Underwood, E.J. & Suttle, N.F. (1999) *The Mineral Nutrition and their calves*.

Canadian Journal of Animal Science, **66**, of *Livestock*, 3rd edn. CABI

International, Wallingford.

669–80.

Wang, Z.Y., Poole, D.B.R. & Mason, J. (1988) The effects of Smith, K.L.,

Harrison, J.H. & Hancock, D.D. (1984) Effect of supplementation of the diet of
young steers with Mo and S

vitamin E and selenium supplementation on incidence of

on the intracellular distribution of copper in liver and on

clinical mastitis and duration of symptoms. *Journal of Dairy copper fractions in*
blood. British Veterinary Journal, **114**, *Science*, **67**, 1293–300.

543–51.

Smyth, J.A., McNamee, P.T., Kennedy, D.G., McCullough, S.J.,

Weiss, W.P., Hogan, J.S., Todhunter, D.A. & Smith, K.L. (1997) Logan, E.F. & Ellis, W.A. (1992) Stillbirth/perinatal weak Effect of vitamin E supplementation in diets with a low

calf syndrome: preliminary pathological, microbiological

concentration of selenium on mammary gland health of

and biochemical findings. *Veterinary Record*, **130**, 237–40.

dairy cows. *Journal of Dairy Science*, **80**, 1728–37.

Spears, J.W., Harvey, R.W. & Segerson, E.C. (1986) Effects of Whelan, B.B., Barrow, N.J. & Peter, D.W. (1994) Selenium

marginal selenium deficiency and winter protein supple-

fertilizers for pastures grazed by sheep. I. Selenium con-

mentation on growth, reproduction and selenium status of

centrations in whole blood and plasma. *Australian Journal*

beef cattle. *Journal of Animal Science*, **63**, 586–94.

of *Agricultural Research*, **45**, 863–75.

Stangl, G.I., Schwarz, F.J., Jahn, B. & Kirchgessner, M. (2000) Wichtel, J.J., Craigie, A.L., Freeman, D.A., Varela-Alvarez, H.

Cobalt-deficiency-induced hyperhomocysteinaemia and

& Williamson, N.B. (1996) The effect of selenium and iodine oxidative status of cattle. *British Journal of Nutrition*, **83**, supplementation on growth rate and on thyroid and soma-3–6.

totropic function in dairy calves at pasture. *Journal of Dairy Stowe*, H.D., Thomas, J.W., Johnson, T., Martenuik, J.V.,

Science, **79**, 1865–72.

Morrow, D.A. & Ullrey, D.E. (1988) Responses of dairy

Adult Cattle

Mastitis and Teat Conditions

Chapter 22

***Anatomy, Physiology and Immunology
of the Udder***

K.G. Hibbitt, N. Craven and E.H. Batten

Anatomy of the udder

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Early development of the udder

Introduction

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Early development of the udder

311

The first trace of mammary development appears in

The adult bovine mammary gland

313

bovine embryos of 1.5 cm length as two short lines in the

The physiology of lactation

316

ectoderm running from the umbilicus caudally into the

Introduction

316

groin. Each line is several cells thick, but intense prolif-

Mammogenesis

316

eration of cells in the basal layer at focal points produces

Lactogenesis

317

an ingrowth, the mammary hillock (Fig. 22.1a). This

Milk synthesis and secretion

318

soon enlarges into an ovoid mammary bud (Fig. 22.1b)

Milk ejection

319

invested by a condensation of inductive mesenchymal

Galactopoiesis

320

cells. As the rudiment of the duct system each mammary

Manipulation of lactation

321

bud determines the site where a gland will form. By the

Immunology of the udder and teat

321

Non-specific immunity

321

seventh week of gestation in fetuses 9 cm long, four

The teat canal as a mechanical barrier

322

primary mammary buds are usually present, two on

Antimicrobial substances within the teat canal

322

each side, defining the future quarters of the udder.

Antimicrobial substances in mammary secretions

322

Extra buds occasionally form and develop into supernu-

Specific immunity: lymphocytes

323

merary teats. Active proliferation of mesenchymal cells

Immunoglobulins in mammary secretions

323

around the mammary bud lifts the epidermis into a rudi-

Phagocytic cell mobilization in the mammary gland
mentary teat by the 8-cm stage. Simultaneously, vigorous
and phagocytosis

324

proliferation of cells near the inner end changes the
mammary bud into a solid cellular column, which elon-
gates vertically into the mesenchyme (Fig. 22.1c) as the
rudiment of the duct system, the future single galac-
tophore. Meanwhile, division among the epidermal cells

Anatomy of the udder

below the original bud produces an epidermal cone at
the base of the duct primordium.

Introduction

By the 19-cm stage the growing duct primordium is
longer than the teat and slightly swollen at the inner
The mammary gland is essentially a skin gland. It is
end. Here a cavity or lumen appears and soon spreads
believed to have evolved by modification of a
proximally towards the teat apex. On reaching the epi-
sweat gland and retains two common features: devel-
dermal cone the split remains narrow as the lumen of

opment by ingrowth of ectoderm; and a bilayered the future teat canal. Later this will open on the teat epithelium of inner secretory cells and outer myoep- apex and both teat canal and superficial epidermis will ithelial cells, which by contracting promote flow of milk be lined by a common, thick, stratified squamous, kera- from the peripheral alveoli into the major ducts. tinizing epithelium.

Assuming the new function of catering for the immune By the 35-cm stage (Fig. 22.2) the growing duct rudi- welfare and nutrition of the neonate, the mammary ment has differentiated into three distinct regions: gland has evolved into a highly branched compound structure with enormous numbers of dilated alveoli.

(1)

An upper spheroid chamber distended with fluid This pattern allows for both synthesis and storage of represents the future gland sinus or milk reservoir. milk on a large scale. Yet neither function is possible

(2)

A slightly longer mid-portion forms the more

until the cow becomes pregnant, when the rudimentary slender teat sinus.

and inactive gland undergoes massive growth to

(3)

A narrow teat canal within the epidermal cone,

definitive structure and only then begins to synthesize

but still closed from the exterior by a plug of horny secretion.

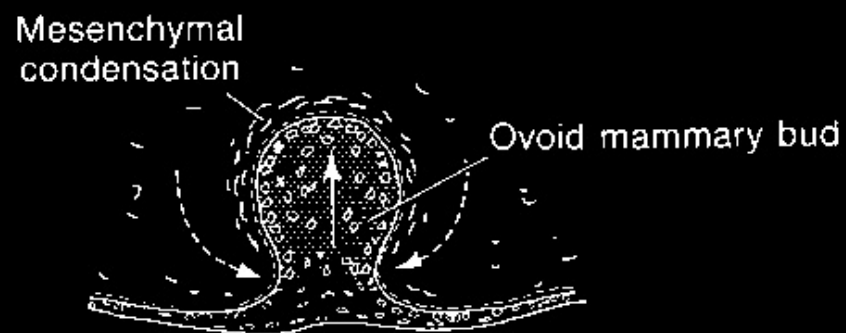
cells.

311

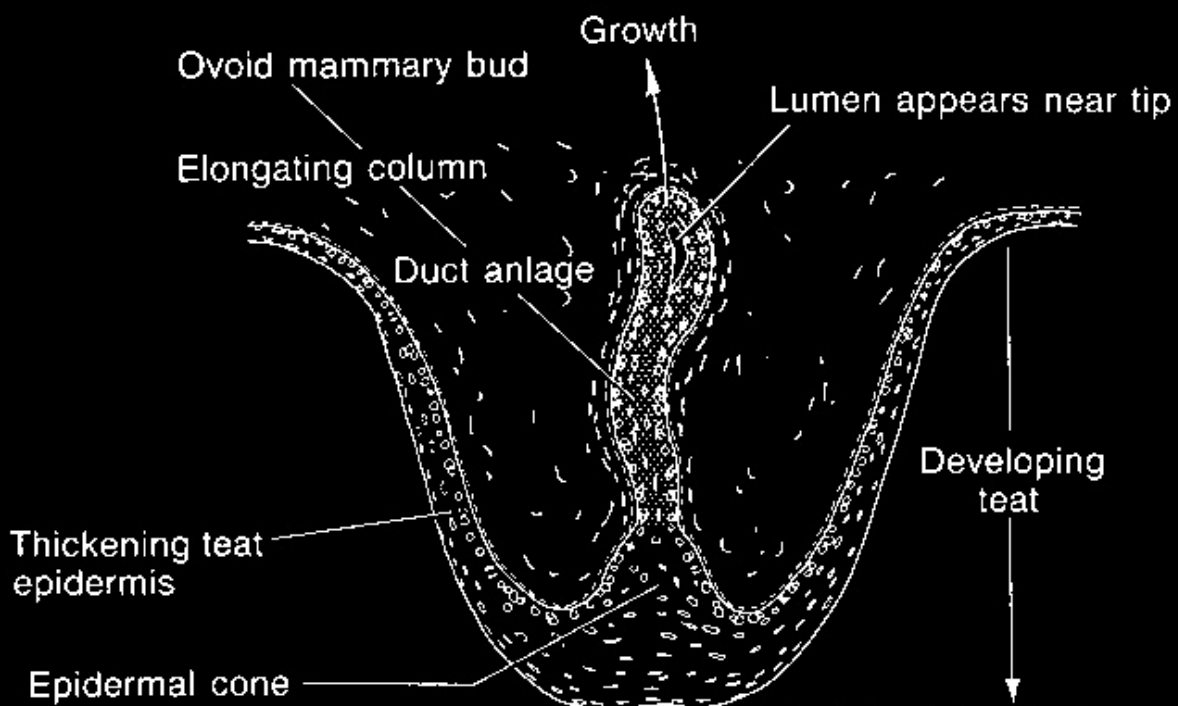
(a) Mammary hillock: 2.5 cm fetus



(b) Mammary bud: 5 cm fetus



(c) Duct anlage and epidermal cone: 19 cm fetus



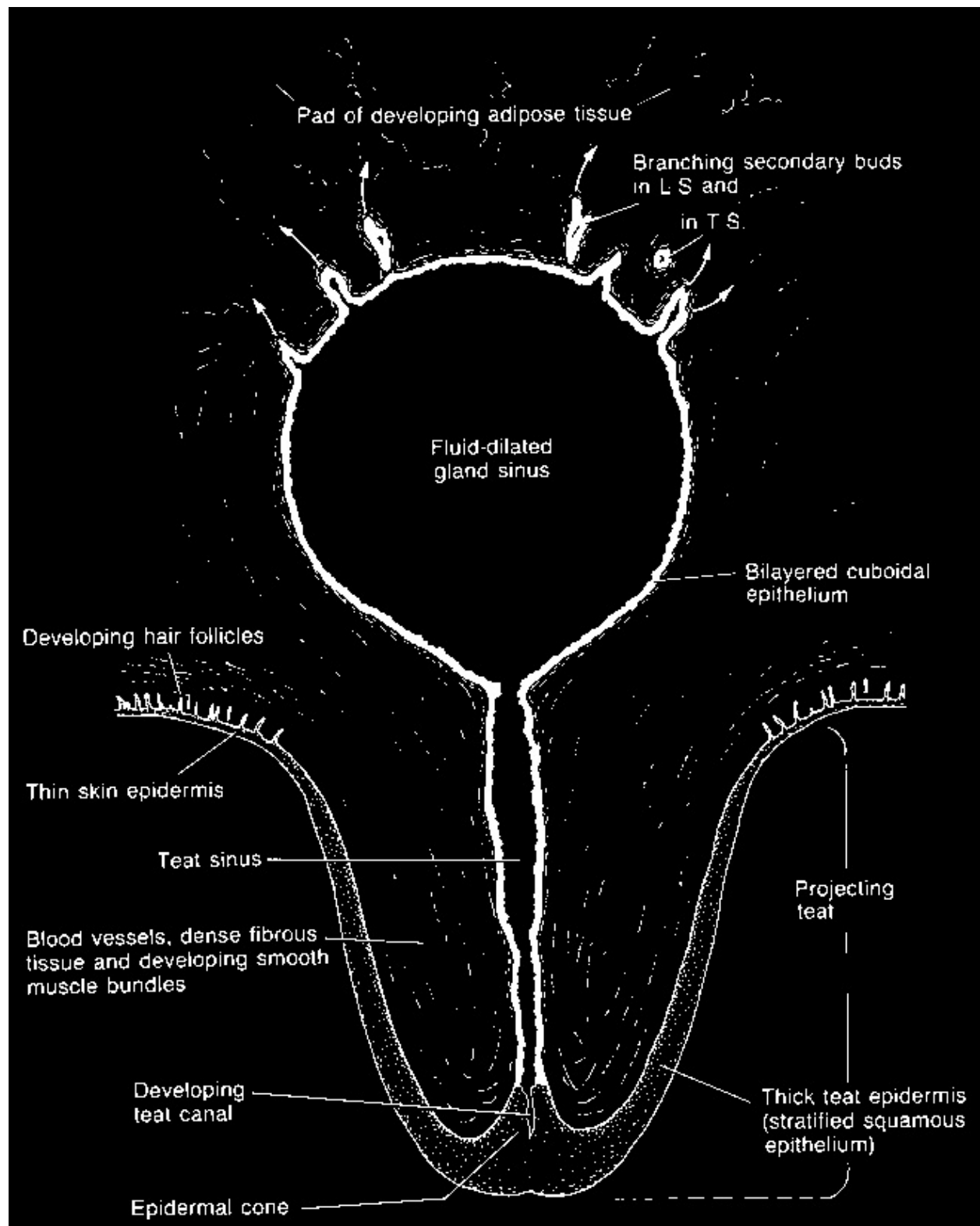


Fig. 22.2

Diagrammatic vertical section of the teat and mammary rudiment in a 35-cm fetus. Stratified epithelium is shown dotted. It appears distinctly thicker in the epidermal cone, Fig. 22.1

(a) Transverse section of a mammary line at the level

where the teat canal lumen is forming, and over the surface of of a localized cellular proliferation, which forms a mammary

the teat rather than above it where hairy skin is differentiating.

hillock and determines where a gland will develop. (b) Later

Derivatives of the original mammary bud are lined by double

stage showing an ovoid mammary bud with cells more basophilic

cuboidal epithelium (black) and comprise the narrow teat sinus (stippled) than in the epidermis. A condensation of mesenchyme-leading from the fluid-distended gland sinus. Up to 10 short sec-mal cells surrounds the bud. Curved broken arrows indicate

ondary buds project from the dome of the sinus and later will

growth of mesenchyme, which elevates a rudimentary teat. (c)

branch to form major ducts as they grow into the overlying

Sustained proliferation (upper arrow) near the tip converts the cushion of fat.

LS, longitudinal section; TS, transverse section mammary bud into a columnar vertical ingrowth, the precursor

(from Turner, 1952).

of the axial duct and storage sinuses. Meanwhile, division among the subjacent epidermal cells produces a cone, which later splits to form the lumen of the teat canal (from Turner, 1952).

stratified squamous epithelium continuous with and identical to the glabrous epidermis of the teat. The From the domed roof of the gland sinus several short latter is distinctly thicker, even in the fetus, than the episolid epithelial cords, the secondary sprouts, project dermis above the teat where hair follicles differentiate dorsally into the overlying pad of differentiating tiate from solid cellular ingrowths. The mesenchyme adipose tissue. When canalized these secondary sprouts around and between the rudimentary epithelial represent the bases of the 10 or more major lactiferous ducts differentiates to provide blood vessels, lymphatics, which in the adult deliver milk into the gland ics, small amounts of smooth muscle and an extensive sinus for storage. The teat sinus, gland sinus and duct fibroelastic stroma. Tracts of denser white fibrous and bases are lined by a bilayered cuboidal epithelium. As elastic tissue form the suspensory ligaments and doralso in the terminal alveoli, which differentiate later sally numerous lobules form in the pad of adipose in pregnancy, the inner cells are potentially secretory,

tissue.

while the outer differentiate into myoepithelial elements. Just before birth tertiary sprouts develop as short side branches from the secondary ducts, but thereafter the axial lumen, forming the teat canal, is lined by thick, the gland remains in an arrested state of development

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until puberty. Some extension to the ducts occurs

Innervation

during oestrous cycles, but full structural differentiation

The principal nerve supply derives from branches of the mammary gland is completed only during pregnancy the third and fourth lumbar nerves, which traverse the inguinal canal. Contributions from the first and second corpus luteum and other hormones. During the first half lumbar nerves supply the cranial, and from the perineal of pregnancy, intense cell proliferation at the blind ends nerves the caudal regions respectively. These are mainly elongates the ducts, which branch repeatedly estab-

sensory nerves, but they carry from the caudal mesenteric plexus sympathetic fibres, which modulate blood flow by direct action on the arterioles. Whereas the skin prospective alveoli then differentiate. In the second half of pregnancy, protein secretion and lipid droplets slowly accumulate, dilating the alveoli into saccular chambers chiefly, if not entirely, vasomotor: the secretory alveoli 120 mm across and filled with stored colostrum awaiting release in the first sucking after birth.

deeper mammary tissue may be incised without apparent sensation.

The adult bovine mammary gland

The cow's udder comprises four quarters, each an individual gland drained by a teat. The four secretory

Mammary gland: histological organization

glands are structurally separate and function independ-

Histologically classified, the lactating udder is a large, entirely, without flow of milk between them. Receiving a lobulated, compound exocrine gland with dilated large flow of blood and laden with stored milk, the lacteal alveoli storing milk. Each alveolus is a single or bifid lactating udder often weights 50–60 kg. Support for this sac, slightly longer than wide and distended to an inter-massive weight is provided by dense fibrous suspensory ligaments that insert into the pelvis and tendons of the abdominal wall. The ligaments spread laterally and ventrally over the udder, then converge inwards to join medially empty gland to stretched squamous (3 mm) in full distension state. Synthesis and release of milk constituents (fluid, casein, lactose and lipid) is continuous, the right. Septa of interlobular connective tissue span until temporarily arrested by negative feedback. The

between the lateral and medial ligaments and support outer contractile myoepithelial cells are indistinct in the heavy lobules of parenchyma. As the medial ligaments contain relatively more elastin than the predominantly collagenous lateral ligaments the full udder reveals their spider shape, with branching processes embracing the curved contour of the alveolar wall (Fig. 22.4). Towards term and during lactation the large drops in the midline and the teats become splayed outwards.

alveoli have a rich capillary supply and are packed closely together into polyhedral lobules about 2 mm across. The alveoli drain into intralobular ducts which, Blood supply

unlike those in salivary or lacrimal glands, are indistinct, since they resemble the alveoli in size, milk content and During the production of 20 kg of milk each day secretory lining. The heavy lobules are enclosed and 9000 kg of blood circulate through the udder of the cow. bound together by thin septa of supportive connective

*Most of this rich supply arrives through the inguinal tissue. This also carries distributing arteries and veins, canal in the external pudendal arteries derived from lymphatics and the larger interlobular ducts, which connect the external iliac trunks. The udder also receives a sub-
verge and unite into major ducts opening into the gland
sidiary supply, cranially through the subcutaneous
cistern.*

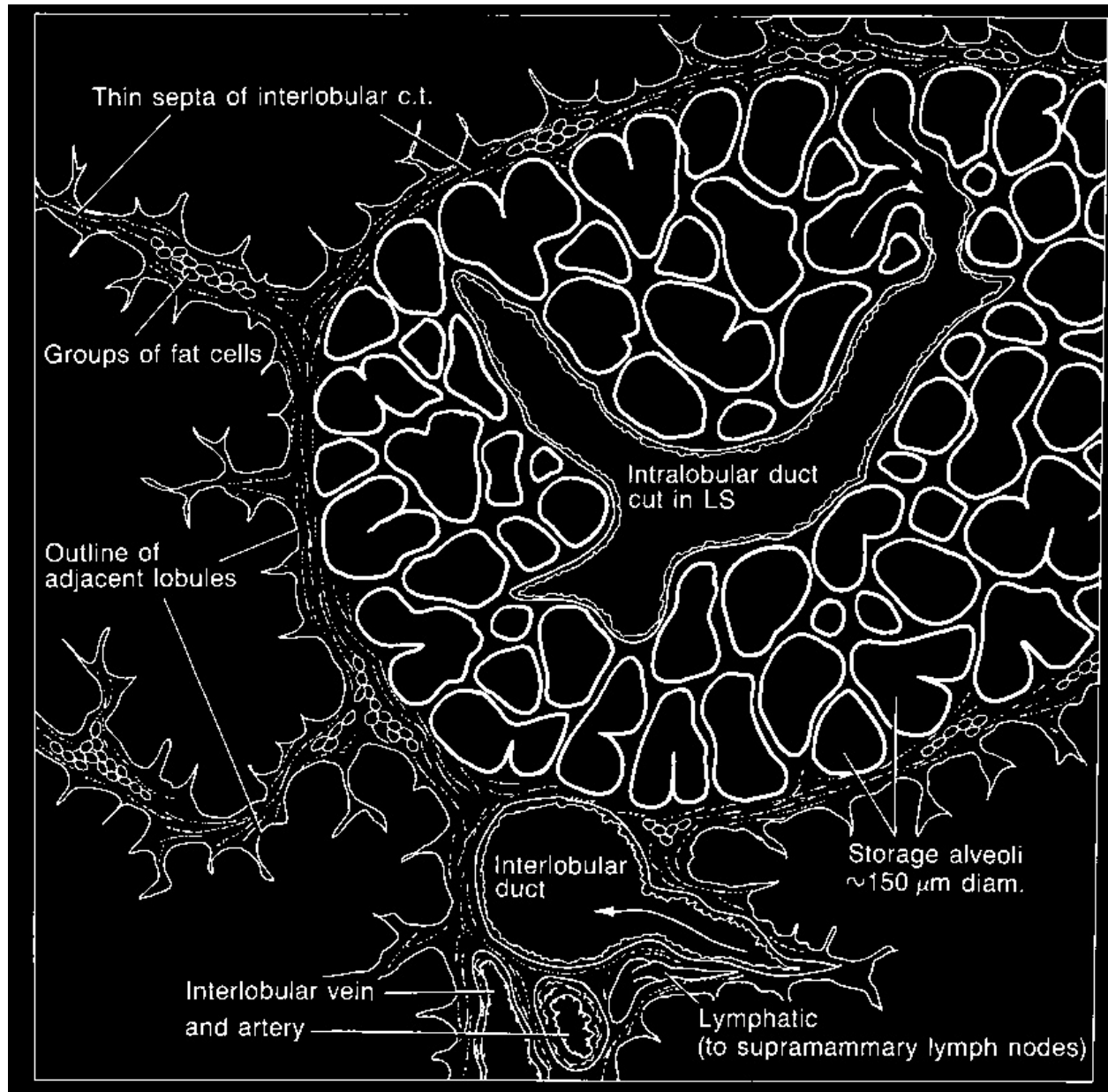
*abdominal artery and caudally via the perineal artery.
Numerous small veins leaving the parenchyma anasto-
mose and converge around the base of the udder into*

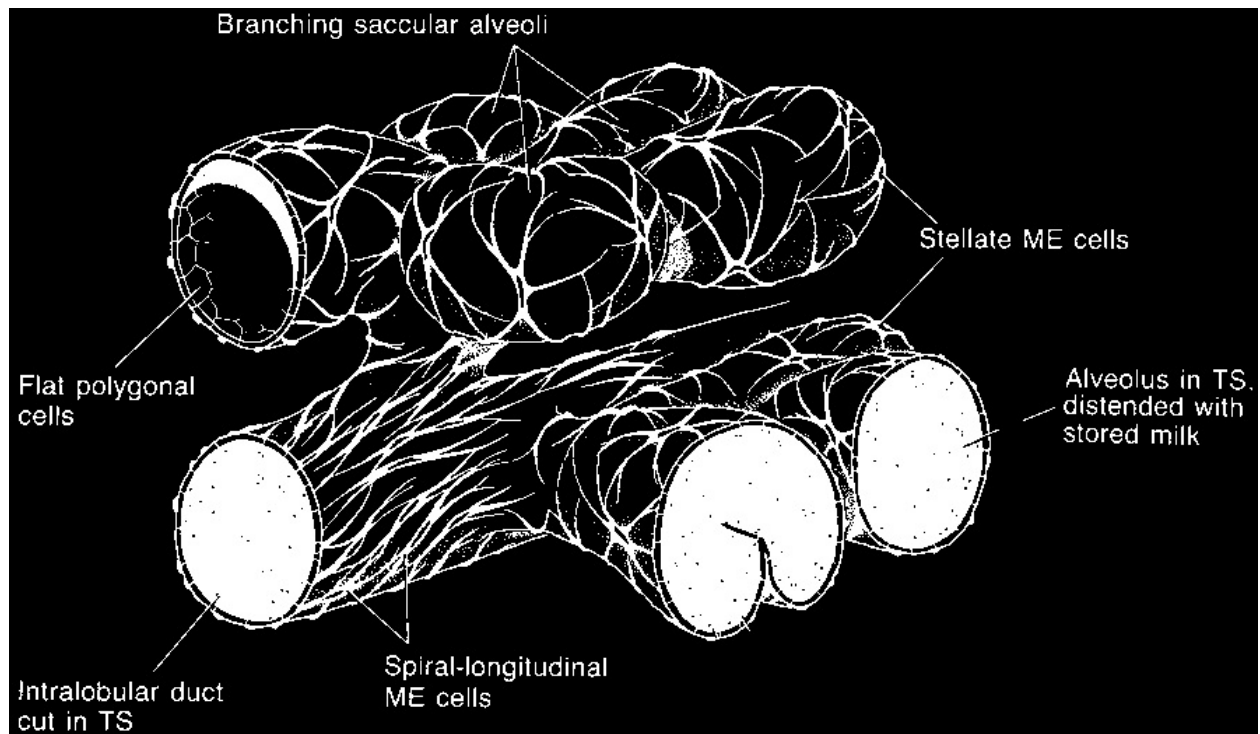
The teat

*a circular vessel that is drained by three trunks: the
large subcutaneous abdominal vein, which passes cra-
Each teat has a single narrow teat canal, which dorsally
nially and penetrates the abdominal wall near the
opens into a wider teat cistern lined by bilayered
xiphoid cartilage; the external pudendal vein, which
epithelium. Normally, the teat canal is kept closed by
departs through the inguinal canal; and the perineal
sphincter action of the surrounding smooth muscle and*

vein.

elastic tissues. Thus in section the lumen is a narrow





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Fig. 22.3

Histological features of the lactating udder, showing large polyhedral lobules supported by thin septa of connective tissue carrying the distributing service vessels: interlobular ducts emerging (at lower right) from lobules, arteries supplying and veins draining the dense networks of perialveolar capillaries. Nearby collecting lymphatics carry a considerable flow of afferent lymph rich in lymphocytes, neutrophils and macrophages and during infections, e.g.

mastitis, carrying antigens that induce immune responses in the supramammary lymph nodes.

In each lobule the alveoli tend to be similar in size, but are generally smaller, averaging 60 μ m across, with a tall cobbled epithelium in lobules that released milk at the previous lactation.

When fully distended with milk, as shown, alveoli approach 150 μ m in diameter and have a thin stretched lining. Smaller profiles represent alveoli slices in oblique to tangential planes.

Intralobular ducts drain milk from alveoli (curved arrows), but unless fortuitously sliced in longitudinal section are almost indistinguishable from the alveoli, being comparable in width and lined by identical secretory epithelium. c.t., connective tissue; LS, longitudinal section (diagram by E.H. Batten).

Fig. 22.4

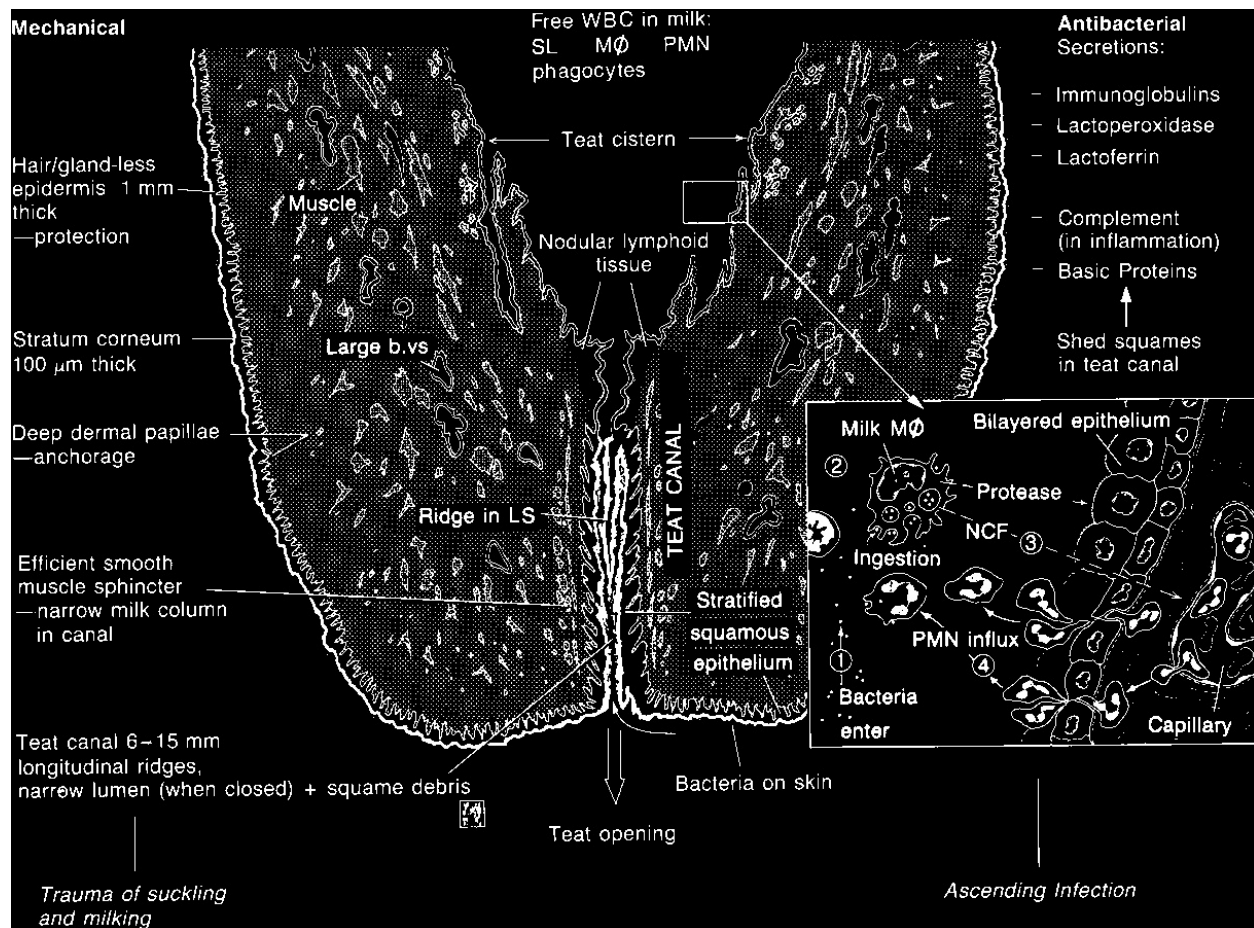
Diagram showing several branching milk-distended alveoli of the lactating udder draining into a relatively

wide intralobular duct. In the sectioned profiles fat droplets in the milk are shown white against a black background. Contraction of the network of stellate myoepithelial cells (cytoplasm black, nuclei white) expels milk from the alveoli into the intralobular ducts. In turn these become shortened and compressed by the contraction of the spirally aligned myoepithelial cells, which propel milk towards the larger interlobular ducts. ME, myoepithelial cells; TS, transverse section (after Linzell, 1961).

stellate crevice, with the lining of thick, keratinizing, lium of the teat canal. The teat is covered by thick, stratified squamous epithelium thrown into several ified squamous, keratinizing epidermis with neither hair longitudinal folds that almost meet centrally (Fig. 22.5). follicles nor sweat and sebaceous glands. Whereas the The teat is robustly constructed and well adapted to thinner skin over the udder is relatively loosely

tolerate the shear stresses generated by a suckling calf attached and can freely be moved over the underlying or milking machine.

glandular lobules, the teat skin is immobile and tightly Structurally the teat wall comprises five distinct tissue anchored to the deeper fibromuscular core. This firm layers: superficial epidermis, then dermis, intermediate surface is well suited to withstand mechanical shear layer, fibrous lamina propria and internally the epithelial forces set up by suckling, hand or machine milking. In



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Fig. 22.5

Diagram of a vertical section of a cow's teat, showing on the left factors that protect against mechanical trauma during suckling or milking, and on the right defences against ascending infection by bacteria from the skin surface (curved arrow) entering the teat canal.

Milk normally contains shed epithelial cells, from both alveoli and ducts, squames from the teat canal, small lymphocytes (SL), macrophages (MØ) and neutrophils (PMN). As phagocytes the last two cell types often contain small droplets of fat ingested from the milk. Subepithelial plasma cells occur near the ring of nodular lymphoid tissue that surrounds the rosette or inner end of the teat canal, where the stratified, keratinizing lining gives way to a bilayered cuboidal epithelium of the teat cistern. Insert right summarizes the early response to mastitis-forming bacteria experimentally introduced into the teat

canal. Bacteria (1) are ingested by milk macrophages (2), which release enzymes that damage and loosen the inner layer of the epithelium and neutrophil chemotactic factor (NCF), which induces (3) within 4 hours the emigration of large numbers of PMN from the subepithelial capillaries and venules. After insinuating through the cisternal epithelium the PMN (4) join with the macrophages in the ingestion and killing of bacteria. WBC, white blood cells; b.vs, blood vessels; LS, longitudinal section (diagram by E.H. Batten).

the lactating cow the teat epidermis is remarkable for

Over this interface the area of adhesive basement

three features.

membrane is fivefold greater than in the flat underside of hairy epidermis. This interlocking pattern binds epi-

(1)

In thickness about 1 mm, it is comparable with

dermis securely and inseparably into the dermis and

muzzle epidermis and some 12 times deeper than

dissipates shear stresses from the surface through the

epidermis of hair skin (75 mm in frozen sections,

deeper tissues. The dermal fibrous mat continues into

which preserve the 30 layers of horny squames

the dense fibromuscular layer, without the intervening

only 1 mm thick).

loose superficial fascia typical of thin skin.

(2)

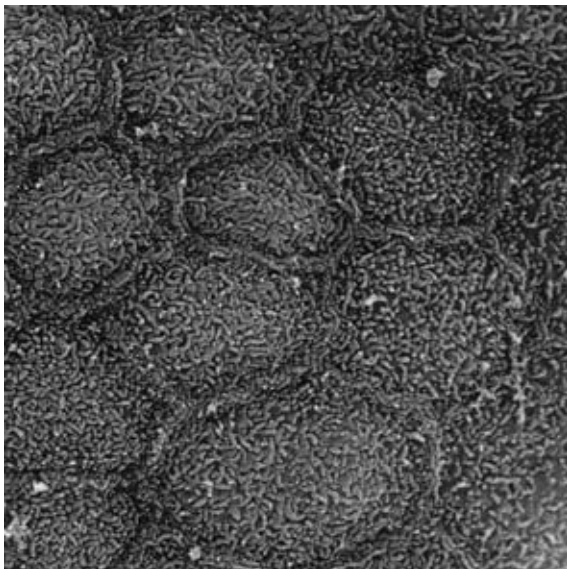
The protective stratum corneum is a compact

Beneath the epidermis the dermis carries a rich capillary network and numerous fine bundles of sensory lapping layers of dead horny squames.

fibres derived from lumbar nerves 2–4. Despite its (3)

The underside is deeply papillated: epidermal thickness as a protection against wear, teat epidermis pegs interdigitate with narrow, deep intrusions of is highly sensitive, since the penetrating dermal papill- dermal connective tissue.

lae carry sensory nerves and endings to within 200 mm



or less of the surface. This rich innervation receives around the inner opening of the teat canal (earlier tactile stimuli and relays to the central nervous system termed Furstenberg's rosette from the mucosal creases) impulses that lead to the release of oxytocin from the the surface cells are more rounded and protruding, with pars nervosa into the blood. In turn, circulating oxy-sparser microvilli. In this region just beyond the barrier tocin induces contraction of myoepithelial cells, pro-epithelium of the teat canal the lining may be a lymphating flow from the terminal alveoli and along the phoepithelium important in the uptake or penetration ducts during milk let-down. The deeper regions of of invading antigens. Intraepithelial lymphocytes are the dermis contain dense collagenous tissue surprofuse and both diffuse and nodular lymphoid tissue rounding bundles of smooth muscle arranged mainly are present in the lamina propria. The accumulations longitudinally.

of plasma cells and germinal centres (Fig. 22.7) often

The third or intermediate layer contributes much of

*present there provide evidence of local humoral
the strength of the teat wall, as it contains numerous
immune responses.*

*bundles of smooth muscle set in coarse fibroelastic
tissue. These muscle bundles are aligned in longitudi-
nal, circular and oblique planes. Major blood vessels
are present, including distributing arteries, a complex*

The physiology of lactation

*plexus of anastomosing veins and numerous collecting
lymphatics, which drain to the supramammary nodes.*

Introduction

*The lamina propria resembles the dermis in its fib-
The secretion of milk by specialized mammary glands
roelastic components, but carries more microvessels
in the female for the nourishment of the newborn is
catering for the nutrition of the adjoining epithelium of
the essential characteristic that distinguishes mammals
the teat canal. During ascending infections with patho-
from other animals. This feature is epitomized in the
genic bacteria, increased emigration from the local
dairy cow which, as a result of intensive selection, has*

venules creates leucocytic infiltrations beneath regions a disproportionately high output of energy in milk in where epithelial cells have been damaged, as explained relation to body size. Continuing improvements in milk in Fig. 22.5.

yield and feed efficiency are the result of refinement of Both the teat cistern and the gland cistern dorsal to the genetic make-up of stock and improvements in it are lined by a common bilayered cuboidal epithelium. management and nutritional practices. This focusing of Under scanning electron microscopy the superficial effort on increasing milk production has prompted the cells fit closely in hexagonal profiles densely covered facetious comment that the modern dairy cow should with microvilli (Fig. 22.6). In the junctional region perhaps be regarded as an appendage of the udder rather than vice versa! Indeed, the readjustment of physiological processes that occurs in order to meet the extra metabolic demands of lactation involves not just differentiation and activation of mammary tissue but extends to changes throughout the body. Not the least

of these is the hormonal regulation of nutrient utilization and partitioning between the mammary gland and other organs.

Milk is a complex secretion and its production is under complex control. By understanding the underlying physiology, the relevance of correct nutritional and husbandry practices in maintaining health and optimizing performance may be better appreciated.

Mammogenesis

Mammogenesis may be defined as the growth and differentiation of the mammary gland to the stage prior to active secretion. Since milk yield is ultimately dependent on the number of secretory cells and their activity, factors that influence the former during mammore-

Fig. 22.6

Characteristic outline of teat epithelial cells covered with irregular microvilli (¥9000). From Collins et al. (1986). production.



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Fig. 22.7

A lymphoid nodule with germinal centre beneath the double cuboidal epithelium lining Furstenberg's rosette (HE × 32).

From Collins et al., 1986.

From birth to puberty mammary growth is isometric, during this critical phase of mammary development i.e. in balance with the growth rate of the whole body, may in fact be caused by a decrease in endogenous but just prior to the onset of ovarian activity mammary levels of somatotropin, as has been observed during growth becomes allometric (i.e. exceeds that of the high plane feeding. Thus the interplay between nutri-

body surface). Only during the first pregnancy does tional management and hormonal balance during marked branching of the duct system and lobulo-mammogenesis may have important consequences for alveolar development occur, with expanding subsequent production.

parenchyma displacing adipose tissue within the Mammary development is partly reversed during mammary fat pad. Lobulo-alveolar proliferation accelerating lactation when gradual involution occurs. erates as pregnancy advances with division of secretory These effects become more pronounced during the dry cells continuing at least until the onset of lactation. Bal-period. A period of non-lactation between successive anced mammary development exhibits a crucial lactations is an essential prerequisite for maximal milk dependence on hormonal stimulation. The hormones production. Milk yields in the next lactation are definitely impaired if cows are dry for less than six weeks.

progesterone and adrenal corticoids) and protein hor-

On the other hand there is little or no advantage to be

mones (prolactin, somatotropin and placental lacto-

gained in terms of an increase in subsequent lactation

gen). Thyroid hormones are possibly also involved.

yield by extending the dry period to more than eight

Many of these hormones interact synergistically to

weeks.

promote the different stages of mammogenesis. Oestro-

gens and somatotropin are responsible for ductal devel-

Lactogenesis

opment whereas progesterone and prolactin appear to

regulate lobulo-alveolar proliferation. The additional

The initiation of milk secretion (lactogenesis) in all

presence of adrenal corticoids maximizes this growth.

mammals is closely coordinated with parturition. Par-

Placental lactogen may, in some species, also stimulate

turition itself involves a complex interplay of endocrine

alveolar formation but in the cow relatively little enters

controls with marked differences between species.

the maternal circulation.

There are two general concepts as to what constitutes

During the period of allometric growth just prior

the main lactogenic trigger, the positive stimulus of

to puberty the administration of exogenous soma-

lactation-promoting hormones and the release from the

totropin to heifers appears to promote an increase in

inhibitory effects of progesterone. A peak in blood

mammary parenchyma. It is known that excess energy

prolactin levels coincides with, but is not essential for,

consumption during the period of allometric growth in

parturition. Suppression of prolactin release in cows

heifers offered a high plane of nutrition results in lower

inhibits the final stages of secretory cell differentiation

mammary secretory tissue weight and is also associated

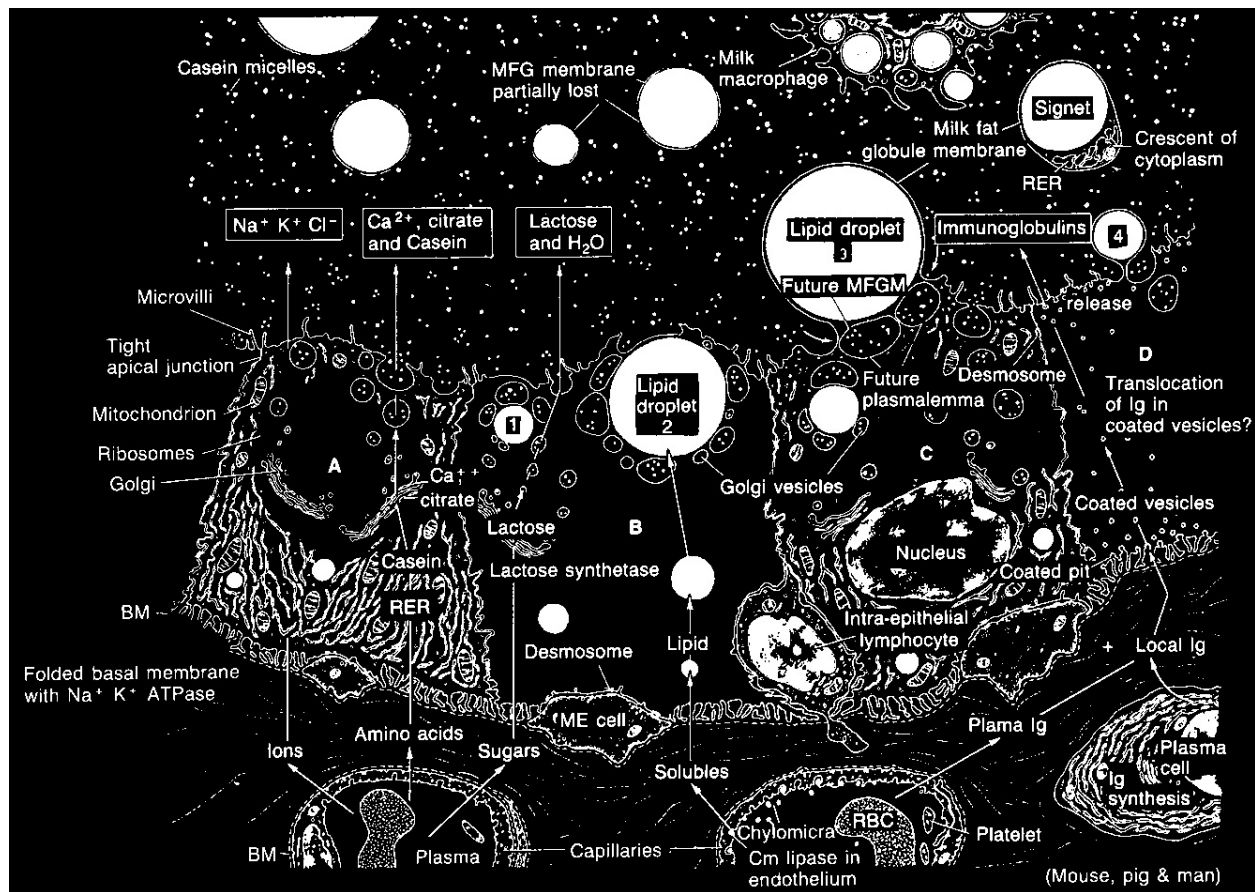
and results in reduced milk yield. Thus elevation of

with low milk production during subsequent lactation.

prolactin (together with adequate amounts of adrenal

It has been suggested that these effects of overfeeding

corticoids) and the permissive effect of progesterone



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withdrawal, appear to provide the main lactogenic stimulus in the cow. An additional feature may be the consistent of any constituent due largely to its influence removal near term of a locally produced inhibitory factor (possibly prostaglandin F2a).

the main precursor of lactose but small amounts may

Lactogenesis requires the preferential supply and

also be formed from amino acids, glycerol and acetate.

uptake of nutrients by mammary tissue. Nutrient avail-

The final enzyme-catalysed step in lactose synthesis

ability is enhanced by the disconnection of the fetal

from glucose involves an enzyme, 'lactose synthetase',

supply at parturition and increases in mammary blood

which is unique to mammary tissue. This is composed

flow and local selective nutrient uptake become

of two protein components and the availability of the

evident.

second component, α -lactalbumin (a milk protein),

determines the rate of synthesis of lactose, which occurs

in the Golgi membranes of secretory cells.

Milk synthesis and secretion

Milk protein synthesis involves the assembly of dif-

The constituents of milk are synthesized mainly from

ferent amino acids in a specific order along a chain. It

small molecules absorbed from the blood, specific

takes place in the ribosomes of the rough endoplasmic

carrier systems probably assisting their entry into se-

reticulum (RER in Fig. 22.8) of secretory cells, proteins

cretory cells. The blood supply provides not only destined for secretion being released into the lumina of precursors for milk synthesis but also adequate the endoplasmic reticulum. Total amino acid nitrogen energy-yielding substrates to drive the synthetic uptake by mammary tissue entirely accounts for the processes.

output of nitrogen in milk protein. However, the pro-Lactose (milk sugar) is a disaccharide composed of portions of different amino acids absorbed by the one molecule of glucose and one of galactose. In bovine glands are at variance with their appearance in milk

Fig. 22.8

Mammary alveolus secretory pathways. RBC, red blood cell; Ig, immunoglobulin; RER, rough endoplasmic reticulum; BM, basement membrane; MFG, milk fat globule; MFGM, milk fat globule membrane (diagram by E.H. Batten).

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protein, indicating that some are metabolized while apocrine, since it does involve some loss of cell components are synthesized by mammary tissue. The major-nents – the membrane investing the lipid and beneath

ity of milk proteins are synthesized locally and are it usually a thin veil of cytoplasm, as illustrated in Fig. unique to milk (e.g. caseins). However, certain blood 22.8. About 5 per cent of droplets are signet-shaped, proteins and similar proteins synthesized by plasma with a crescentic fragment of cytoplasm containing cells adjacent to secretory epithelia are also transferred a few mitochondria or short profiles of rough ER. into lacteal secretions – in large amounts in colostrum Immune proteins, such as various immunoglobulins (see below).

arriving via the blood or derived from local plasma The fat content of bovine milk varies widely between cells, are taken into the cell base by receptor- and within breeds and is influenced by diet and environmental factors. The majority of milk fat is in the through the cytoplasm and released at the apical form of triglycerides, which are assembled in or near membrane.

the endoplasmic reticulum of secretory cells using fatty

Mature milk is characterized by a high concentration of acids and glycerol synthesized in the cytosol. Approximately half of the fatty acids in milk are derived directly from the blood – chiefly those greater than 14 carbons is 86 per cent water. The amount of water (and hence in length. De novo synthesis of fatty acids utilizes milk volume) is determined largely by the rate of volatile fatty acids arising from rumen fermentation, lactose secretion since lactose is not reabsorbed across fatty acid synthesis from glucose being negligible in epithelial cell membranes and it exerts an important osmotic ‘draw’ within the milk duct system.

Acetate and b-hydroxybutyrate are important precursors and contribute mainly to the synthesis of shorter-chain fatty acids. The glycerol required for

Milk ejection

milk triglycerides is partly derived from hydrolysis of blood lipids and partly by synthesis from glucose.

In the interval between milkings, milk continues to be

Esterification of fatty acids with glycerol is followed by synthesized and secreted at a more or less constant rate aggregation of triglycerides to form the characteristic into the alveolar lumina and thence to the large col-fat droplets of secretory cells.

lecting ducts and sinuses. The resistance of the teat The mechanisms for cellular release of secretion sphincter retains the milk in the sinuses but much of the differ for the various components of milk. Instead of milk remains within alveoli and will not flow out pas-combining into a large exocrine granule typical of sively even when the teat sphincter is patent. Forcible zymogenic and serous cells, milk protein remains sepa-expulsion is, therefore, required to remove this alveolar rate as multiple small granules. These are visible only milk from the gland. Milk ejection is under neuroen-ultrastructurally and present a large surface area, docrine control.

which facilitates rapid digestion. Non-immune protein, The milk ejection reflex involves stimulation of nerve such as casein, is delivered from the rough endoplasmic

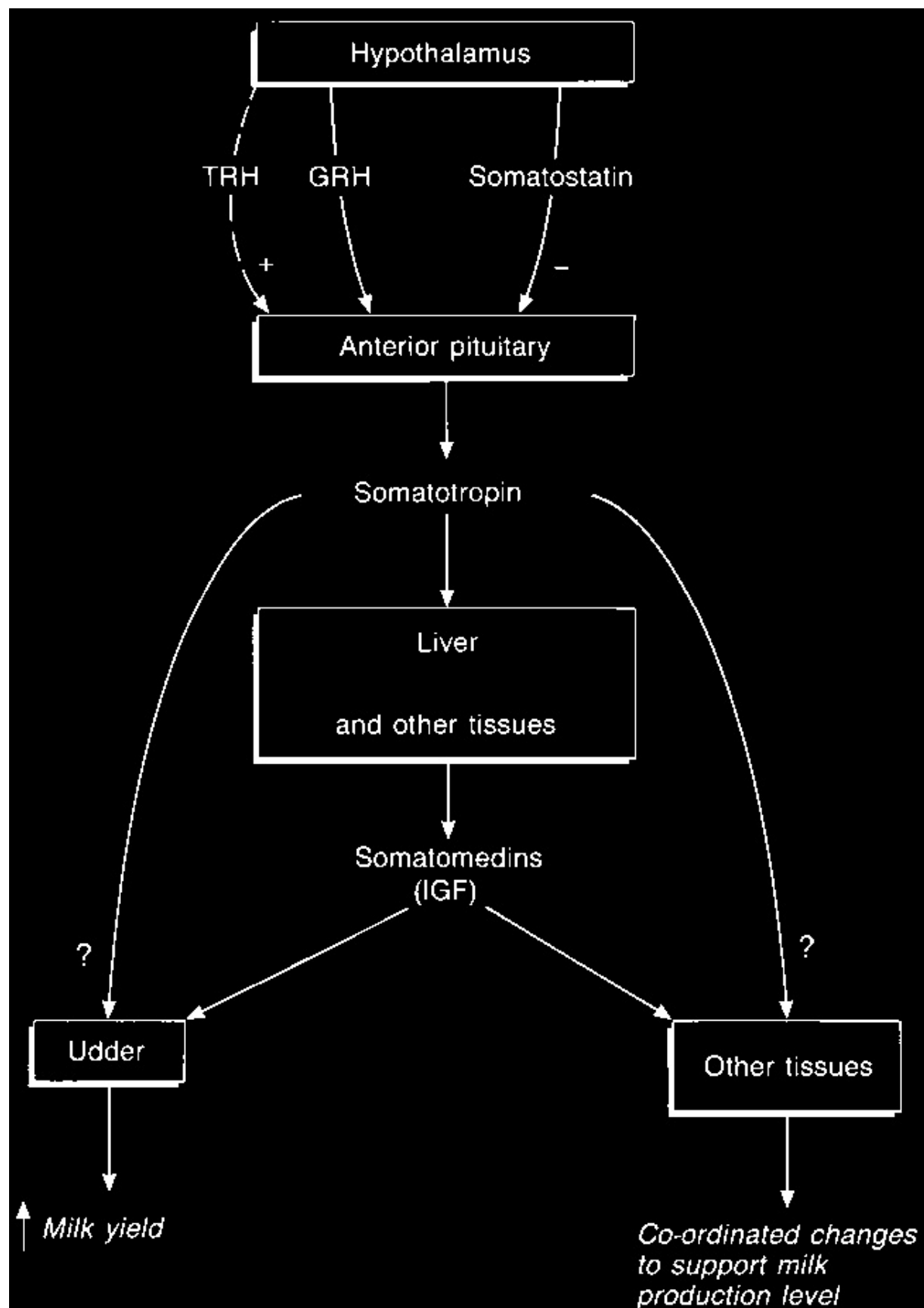
receptors in the skin of the teat. Mechanical stimulation triggers an impulse in afferent mammary nerves. This impulse is transmitted via the spinal cord and ultimately causes the vesicles leaving the Golgi to swell osmotically. As they rise through the supranuclear cytoplasm, the vesicles become tightly clustered around the sides of the more superficial vesicles fuse with the apical plasma membrane and their content of protein, lactose, calcium and fluid is emptied into the alveolar secretory granules are released into blood capillaries

lumen by conventional exocytosis. As the vesicles successively discharge, their inner walls remain over the surface of the protruding lipid as the milk fat droplet

Thus following mechanical stimulation of the teats of cows, oxytocin increases rapidly in the blood, reaching a peak within 2 minutes.

branes between the lowest vesicles, the milk fat droplet is liberated into the lumen without damage to the form a basketwork surrounding the alveoli. Oxytocin in cell and in a manner reminiscent of the shedding from mammary blood binds with high affinity to receptor sites on myoepithelial cells (Fig. 22.4), resulting in contraction of the cells and expulsion of the milk from the alveoli.

The process of milk fat release is best termed micro-



Although sucking and milking are the most potent and natural stimuli to elicit the milk ejection reflex, udder washing and even visual and auditory cues associated with milking routines can substitute, as the reflex becomes conditioned by experience. Stressful stimuli which involve the release of catecholamines and activity of the sympathetic nervous system can have negative influences on oxytocin release, mammary blood flow and oxytocin binding to myoepithelial cells, thereby inhibiting milk ejection. Myoepithelial cells also contract to an extent in response to direct mechanical stimuli, independently of oxytocin release. Palpation of the udder (or butting by calves) may trigger this local so-called ‘tap reflex’.

Galactopoiesis

Frequent milk let-down and its removal from alveolar lumina assists in the maintenance of milk production (galactopoiesis) by preventing ‘end product inhibition’. Nevertheless, the continued secretion of milk also

depends on a continued hormonal drive, which induces and maintains the synthetic enzyme complement of secretory cells and also ensures an adequate supply of substrates. The chief hormones implicated in the maintenance of lactation are different from those responsible for lactogenesis.

The essential role of the pituitary gland in the maintenance of lactation in all mammals has long been rec-

Fig. 22.9

*Regulation of somatotropin release and its effects on
ognized. The roles of the various pituitary hormones
milk production. TRH, thyroid hormone releasing hormone; GRH,
have been revealed by studies on hypophysectomized
growth hormone releasing hormone; IGF = insulin-like growth
animals with hormonal supplementation or by the use
factor.*

*of specific antagonists. In most species (i.e. rabbits, pigs,
dogs, humans) prolactin has been identified as the key
galactopoietic hormone and suppression of prolactin
action of the specific releasing hormone. A direct effect
secretion by bromocriptine has a rapid effect in inhibit-*

of increased levels of somatotropin is the induction in
ing milk secretion. In ruminants, however, suppression
the liver and other organs of the synthesis of other pep-
of prolactin secretion has only a partial (sheep) or no
tides known as somatomedins or insulin-like growth
inhibitory effect (cows, goats) on lactation. In these
factors (IGFs). IGFs in turn appear responsible for
species, somatotropin (pituitary growth hormone) pro-
mediating many of the effects attributed to soma-
vides the main galactopoietic stimulus. The dose-
totropin (Fig. 22.9).

dependent stimulation of milk yield in dairy cows that
It seems likely that somatotropin does not exert its
follows administration of somatotropin has been known
galactopoietic effect by direct stimulation of the udder.
for many years. With the advent of recombinant DNA
Rather it acts by partitioning available energy away
technology, commercial exploitation of this effect is
from body tissues and towards milk production but the
now practicable and our knowledge of the role of soma-
increased yield is not merely a passive response to an

totropin has increased as a result of the considerable increased nutrient supply. There is also an increased recent research on this topic.

synthetic activity per secretory cell. Administration The release of somatotropin into circulation from the of somatotropin also elevates cardiac output and anterior pituitary is under the influence of various mammary blood flow. It is considered that this increase hypothalamic peptides that arrive in the hypophyseal in blood flow is, at least in part, a consequence rather portal blood supply; these include a specific somat- than a cause of the increased activity of mammary totropin releasing hormone and an inhibitory peptide, tissue.

somatostatin. Thyroid hormone releasing hormone also The metabolic effects of administering supplemen- enhances somatotropin release and synergizes with the tary somatotropin to dairy cows are well documented.

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In early short-term studies, acute responses of hyper- management during this period is clearly of practical

*glycaemia ('diabetogenesis') and elevation of cir-
relevance in influencing mammogenesis and sub-
culating free fatty acids ('lipolysis') were reported. In
sequent yield.*

contrast, there is now substantial evidence from studies

*The hormonal manipulation of galactopoiesis has
where somatotropin has been administered for long
considerable commercial potential, both in a free
periods which indicates that the significant increases in
market for milk products and for efficient quota
milk yield are perfectly balanced by increased volun-
management under a milk quota system. Biotechnol-
tary feed intakes and alterations in metabolism of body
ogy-derived somatotropin in prolonged-release formu-
tissues such that steady-state concentrations of blood
lations is already available for commercial use in some
metabolites are maintained. These coordinated changes
countries (see p. 1073). Endogenous somatotropin pro-
are consistent with the role of somatotropin as a home-
duction may also be enhanced by administering releas-
orhetic control, allowing a substantial shift in the parti-*

ing factors or by immunization of animals against their
tioning of nutrients to the mammary gland.

natural inhibitory peptide, somatostatin. Research into
The key role of somatotropin in milk production is
such approaches is under way.

underscored by the evidence that cows selected for high
Developments in direct genetic transfers between
genetic merit for yield show higher endogenous levels
mammalian cells make possible the insertion of extra
(basal and after stimulation) of somatotropin than
somatotropin genes which, if expressed, will enable
low yielders. Furthermore, somatotropin levels decline
cows to produce high endogenous levels of soma-
with advancing lactation in line with milk production.
totropin. The genes might be triggered by an external
Somatotropin levels are also influenced by nutritional
stimulus such as a dietary factor. Such transgenic 'super
status independently of these genetic effects. Underfed
cows' could be propagated rapidly using modern repro-
cows have generally higher somatotropin levels than
ductive techniques. Transgenic techniques might also be

those that are well fed, although this may be offset by employed to modify the characteristics of the milk containing a lower somatotropin responsiveness of the underfed constituents produced.

animals.

There are also opportunities for optimizing milking

Other hormones with a suggested role in galactogenesis procedures to exploit physiological controls of lactation topogenesis include adrenal corticoids and thyroid more fully. Further automation of the milking process hormones. In most species adrenal corticoids are essential for lactation but there is little evidence that several times a day and, with some sophistication, plasma levels are a limiting factor. Thyroxine and triiodothyronine may have a stimulatory effect on milk milking.

yield via effects on metabolic rate.

Immediate increases in milk yield can be obtained by commencing three times a day milking, which indicates

Immunology of the udder and teat

that the secretory capacity of mammary cells can be increased. Frequency of milking does not appear to affect endogenous somatotropin levels. The mechanism of action of these two systems (see Chapter 29). Firstly, a non-specific system that offers a first line of defence when a pathogen invades, and frequently is able to resist the invasion of a number of potentially infectious agents. Secondly, if the non-specific system fails, the tissues of the mammary gland can adapt and produce a specific defence against each infectious agent. Frequently this yields of dairy cows is the result of judicious genetic selection and improvements in management, especially nutritional management. Experts expect these trends to

Manipulation of lactation

the mammary gland can adapt and produce a specific defence against each infectious agent. Frequently this yields of dairy cows is the result of judicious genetic selection and improvements in management, especially nutritional management. Experts expect these trends to

system on a later occasion.

continue for some years to come. However, a growing understanding of the physiological processes of lacta-

Non-specific immunity

tion, together with advances in biotechnology, now make possible a variety of new ways to control and

Most infections of the mammary gland of the dairy cow manipulate lactation.

enter through the teat canal so the teat provides a very

Administration of somatotropin to prepubertal

important barrier to infection, particularly since its ex-

heifers has been shown to increase mammary paren-

terior surface is frequently exposed to a most con-

chyma but whether this translates into increased milk

taminated environment. The defences of the mammary

yields is not yet clear. Nevertheless, optimal nutritional

gland may be considered under several headings.

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The teat canal as a mechanical barrier

bind electrostatically to the mastitis pathogens which,

at the same pH, carry a negative charge. The inhibiting

The length of the teat canal has no effect on the susceptibility of the various concentrations of these proteins to mastitis but it has been reported that it has been demonstrated in vitro on the growth of staphylococci and streptococci. This antimicrobial activity of the teat ducts from quarters susceptible to infection had a larger diameter than those from quarters where these proteins was not seen when micro-organisms were incubated with whole keratin under in vitro conditions. Differences in teat duct patency might be expected to correlate positively with ease of milking but a relationship between milking characteristics and the incidence of intramammary infection is not universally accepted. Teat ducts with a temporary occlusion of these cationic proteins would overcome such binding. Despite the antimicrobial environment milk is maintained for some time after milking, may

*within the teat canal, local colonization by Gram-
offer a means of entry for micro-organisms and a way
positive cocci has been reported, but the growth is very
in which they may be transported upwards through the
feeble with a low percentage viability.*

*duct. In capillary columns of milk at 37°C, micro-
organisms are capable of rising rapidly in association
with ascending milk fat globules. Any mechanism,*

Antimicrobial substances in

therefore, that is likely to break the fat-rich milk

mammary secretions

column and prevent an upward passage of bacteria is

Non-specific antimicrobial substances in mammary

likely to increase the mammary gland defences. Various

secretions also contribute to the defences of the

suggestions have been made on how the teat duct

mammary gland. Lactoferrin is an iron-chelating

closes, e.g. it may close in a spiral fashion ‘wringing out’

protein which, in the presence of bicarbonate, inhibits

the last drops of milk, but as the streak canal has ridges

the growth of micro-organisms having a high iron

arranged longitudinally a spiral wringing would not be requirement. The activity of this protein is inhibited by effective. Teat canal keratin is a white wax-like material the presence of citrate. Lactoferrin is thought to exert derived from the surface cells of the stratified squamous its most protective effects in the mammary secretions epithelium and which forms a network in the lumen of during the dry period when its levels are high compared the canal, thereby trapping invading bacteria and dis- with those in full lactation. These higher concentrations rupting the milk column. Another simple mechanical of lactoferrin may also influence the defences of the dry method of preventing organisms from penetrating the gland through the modulation of leucocyte functions. teat canal is the flushing action during milking. Organ- Another antibacterial system present in milk in- isms not adhering to the teat canal wall or the keratin volves the enzyme lactoperoxidase. This enzyme, in mesh at the beginning of milking are soon flushed out. the presence of thiocyanate and hydrogen peroxide, can The higher infection rate of quarters at drying off has

produce a short-lived highly oxidative system that is been attributed to the cessation of milking and the con-bacteriostatic for Gram-positive and bacteriocidal for sequent failure to flush out pathogens. Later in the Gram-negative organisms. Thiocyanate is present in dry period, there is an increase in the antimicrobial milk, particularly in animals fed on diets containing properties of the canal, which may restrict bacterial legumes or brassicas. Doubt has been expressed in the multiplication.

past over the source of hydrogen peroxide (H_2O_2).

Phagocytosing neutrophils are likely to be one source of H

Antimicrobial substances

$2O_2$ and some can be produced by the metabolism of catalase-negative organisms such as streptococci.

within the teat canal

Another enzyme present in milk, xanthine oxidase, may Antimicrobial substances in the teat canal may con-also contribute by generating H_2O_2 . Recent research tribute to the defence of the mammary gland. The

has demonstrated that xanthine oxidase is present in antimicrobial role of esterified and non-esterified fatty the teat duct and secretory tissue of the bovine acids, particularly myristic acid, palmitoleic acid and mammary gland. The action of xanthine oxidase on its linoleic acid has been described. These fatty acids are substrate leads to a release of hydrogen peroxide, which present in the teat canal keratin and it was suggested is then available to the lactoperoxidase system.

that the unsaturated fatty acids were the most

In addition to the above, antimicrobial systems that inhibitory.

operate locally within the teat canal are likely also to

Some proteins isolated from the teat canal also play be operative in the milk itself and in deeper parts of the an important role in the defence of the mammary gland.

mammary gland. The role of lysozyme in bovine body

At physiological pH they carry a positive charge and fluids, including milk, is equivocal. Some authors doubt

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its existence and, if present, the concentration would be

Differences in the functional activity of T lymphocytes are extremely low, but possibly increasing in mastitis. As yet cytes in blood and those in mammary secretions are no defensive role has been ascribed to lysozyme in evident as differing responses to specific antigens. bovine tissues.

Much of this work has been carried out using human Antimicrobial cationic proteins, as found in the teat mammary secretions and supports the view that lymphocytes in the mammary secretion form a distinct sub-population. They belong to a mucosal immune system the cationic proteins in the milk cells and are thought to in which lymphocytes migrate from secretory surfaces have left the cells by a process of reverse pinocytosis. of the body, e.g. the gut, to the mammary gland, but this Earlier methods used by many workers attempting to is not the case in ruminants.

demonstrate haemolytic complement were too insensitive. A similar migration occurs with B cells, but in the cow

tive, but more recently a sensitive microassay revealed it has been suggested that following intestinal immunization there is a migration of antigen-stimulated IgG in milk throughout lactation, albeit usually masked by lymphoblasts and perhaps of antigen to the spleen and the anticomplement activity of the milk. Complement peripheral lymph nodes. This would be consistent with levels are highest in colostrum, in inflamed glands and the appearance of serum-derived IgG antibodies in the in late lactation. A high correlation between the levels colostrum and milk. When antigen is infused directly of serum albumin and the third component of complement into the udder all classes of antibody, but particularly during an inflammatory response suggests that IgA and IgG1, are produced locally from antibody-complement components are passively transferred into forming cells in the glandular stroma and in the regional the milk but this relationship is not seen in normal milk. supramammary lymph nodes.

Although the defensive role of the classical comple-

*Tissues in the terminal inner portion of the teat
ment pathway remains in doubt it has been established
contain a high proportion of plasma cells, lymphocytes
that complement-sensitive organisms, such as some
and macrophages, which may be involved in local
strains of Escherichia coli, are killed by the alternative immunity, and antibody-
containing cells have been
complement pathway. In vitro tests, however, do not
demonstrated in germinal centres in the distal
support this since serum-sensitive strains of E. coli
Furstenberg's rosettes of the teat cistern (Fig. 22.7). Of
can be grown in milk drawn from the udder. It is possi-
the plasma cells in this region 88 per cent synthesize
ble, however, that in vivo a very mild inflammatory
IgG1, 10 per cent IgM while only a few synthesize IgA.
response is needed to elevate slightly the serum-derived
The distribution of these cells does not change in rela-
complement in milk.
tion to the mammary secretory activity.
Although the homing of lymphocytes to the mam-
mary gland appears to be well established in some*

Specific immunity: lymphocytes (see

species, not all migrate into the secretions; in the rat

Chapter 29)

mammary gland some remain in the alveolar and ductal

Lymphocytes are present in cows' milk at all stages of epithelium. These intraepithelial lymphocytes are likely

lactation and constitute 1–2 per cent of the milk cell

to be the predominating cell types infiltrating bovine

population, which is low compared with that of human

mammary tissues and remain in association with the

milk. Lymphocytes consist of T and B cells together

epithelia after a Staphylococcus aureus infection. Infor-with a population of lymphoid cells, which do not con-

mation from other species may reveal the precise iden-

sistently carry markers of either T or B cells, the so-

tity and function of these cells. In humans a substantial

called null cells. The ratio of these cells (T : B : null) is

proportion are T cells. Intraepithelial lymphocytes may

similar to that in peripheral blood, but the percentage

form an important defence against infection at mucosal

of B cells is slightly increased in dry gland secretions.

surfaces. Currently, there is a paucity of information on Lymphocytes in mammary secretions are functionally the role or even existence of natural killer (NK) and competent, responding to mitogens and antigens.

killer (K) cells in the bovine mammary gland; never-

However, some responses of the T cells from mammary theless, there is some evidence to suggest that lympho-secretions to mitogens are lower than those of T cells cyte activity in bovine mammary tissues may differ from from the systemic circulation. The precise reason for that of other species.

this difference in responsiveness is not clear, although it is known that mammary secretions contain soluble

Immunoglobulins in mammary secretions

immunosuppressive factors that may be responsible. In (see p. 380)

addition, the T cell population in the mammary secretion may contain a greater proportion of suppressor

Milk contains most immunoglobulin classes in differing cells which would inhibit responses.

amounts that vary during lactation. IgG1 is present in

Table 22.1

*Immunoglobulin (mg/100 ml) to the blood and
plement in milk. There have been some reports that
mammary secretions of the cow (from Lascelles, 1979).*

*IgA antibodies assist the binding of bacteria to fat-
globule membranes. This would hinder the mammary*

IgG1

IgG2

IgA

IgM

*defence system by making the bacteria more difficult to
phagocytose.*

Blood serum

1400

1300

39

380

Colostrum whey

1000–9000

250

470

540

Milk whey

40

6

11

9

***Phagocytic cell mobilization in the
mammary gland and phagocytosis***

The most effective system of udder defence against the largest concentrations and it passes from the blood invading pathogens is the phagocytic activity of neutrophils. Normally, these cells are present in milk in very low numbers, the predominating leucocytes in the milk transferred from the blood. Some immunoglobulins of healthy cows being macrophages and lymphocytes. such as IgM and IgA are locally produced and they Neutrophil numbers, however, rapidly increase in the arise from cells of the lymphoblast-plasma cell series

very earliest stages of infection. The value of neutrophils that are located close to glandular epithelium in various parts of the mammary gland. The distribution of immunoglobulin classes in blood, colostrum and milk is shown in Table 22.1.

anti-bovine leucocyte serum.

It will be noted from Table 22.1 that colostrum has a markedly higher concentration of IgG1 than serum. This is likely to be triggered by the presence of bacteria and bacterial products, which stimulate the formation of receptor sites for this immunoglobulin on the membranes of bovine mammary epithelial cells. There are suggestions in the literature that the levels of IgA in cause a relaxation of endothelial cell junctions and

milk and colostrum have probably been underestimated because much of the IgA is associated with milk surrounding subepithelial connective tissues. The neutrophil membranes, and some IgM may also be in turn may release other inflammatory agents, globule bound. The precise origin of IgG2 and IgA in further accelerating the process of neutrophil mobilization. The identity of the inflammatory mediators, the mammary gland of the cow remains in doubt. A local synthesis of IgG2 has been reported in the cow but which has been studied by a number of workers, in this was not confirmed in the ewe. There appears to be includes prostaglandins produced by the cyclo-oxygenase less disagreement concerning the origin of IgM and pathway, and leukotrienes produced by lipo-oxygenase-IgA, which are thought to be largely derived from local and interleukins. Prostaglandins have been measured in bovine milk and shown to increase in the ewe the synthesis of these immunoglobulins appears

concentration from 4 hours after the administration of to be confined to the period of mammary involution intramammary endotoxin. Prostaglandins are synthesized whereas in early and mid-lactation large amounts are sized in bovine mammary tissue and may also be generated from the blood.

erated by neutrophils.

Inflammation of the mammary gland leads to a rapid Pathogens entering the mammary gland and causing increase in levels of immunoglobulin and other serum mastitis are rarely invasive. Therefore, if an antimicrobial environment is to be produced in the mammary hours. In this situation IgM and IgG1, together with gland neutrophils must be present in the teat and lactiferous sinus regions, the ducts and in the alveolar serum-sensitive coliform organisms. Phagocytosis by lumen. Milk collects in the teat and lactiferous sinus neutrophils is more efficient if they are coated with regions and it is thought that neutrophils are attracted

specific IgG2 antibodies.

as far as the luminal surface of the two-cell-thick epithe-

The precise role of IgA in the mammary gland of the

lium by a concentration gradient of chemotactic factors

cow remains in doubt. Some workers believe that it may

originating from the lumen of the gland. Under these

block IgG- or IgM-mediated complement fixation, in

conditions, the neutrophils pass through the basement

which case it would have detrimental effects on the pro-

membrane and between the epithelial cells (Fig. 22.5).

tection of the mammary gland in early infection. On the

In severe staphylococcal infections the surface cells

other hand, it may activate complement, but this may

suffer toxic damage and in some cases this leads to large

not be of great importance due to the low levels of com-

areas of epithelial erosion. Infections with E. coli

Anatomy, Physiology and Immunology of the Udder • 325

frequently lead to more localized toxin-induced epithe-

opsonin for neutrophil phagocytosis of mammary gland

lial lesions and it is from these regions that the neu-

pathogens using de complemented non-immune bovine

trophils migrate into the lumen.

serum is IgM.

A rapid neutrophil migration is essential for the efficient defence of the mammary gland following E. coli

References

invasion, but particularly if the response occurs before the bacterial count in the milk exceeds 10⁴/ml. Some

Collins, R.A., Parsons, K.R. & Bland, A.P. (1986) Antibody cows, however, particularly if recently calved, fail to

containing cells and specialised epithelial cells in the bovine recruit neutrophils into their milk, and this leads to an

teat. Research in Veterinary Science, 41, 50–5.

unrestricted bacterial growth and severe damage within

Lascelles, A.K. (1979) The immune system of the ruminant

the mammary gland tissues. This situation may not be

mammary gland and its role in the control of mastitis.

observed clinically since there may be a complete lack

Journal of Dairy Science, 62, 156–60.

of inflammatory reaction. Nevertheless, these animals

Linzell, J.L. (1961) Recent advances in the physiology of the

soon become severely ill and death may follow. Fre-

udder. In Veterinary Annual (ed. W.A. Pool). John Wright quarterly, cows respond

with a neutrophil recruitment

and Sons, Bristol, 44–53.

but there is often a delay such that bacterial numbers

Turner, C.W. (1952) The Mammary Gland. In The Anatomy of

may increase to a high level. Bacteria and neutrophils

the Udder of Cattle and Domestic Animals. Lucas Brothers may then coexist in the mammary gland resulting in a

Publishers, Columbia, 176–83, 203–7.

protracted mastitis.

A rapid neutrophil response is also beneficial for

Further reading

controlling the growth of Staphylococcus aureus in

mammary tissues, thereby preventing severe disease. A

Butler, J.E. (1974) Immunoglobulins of the mammary secre-

complete elimination of these organisms, however,

tions. In Lactation: a Comprehensive Treatise, Vol. III (ed.

rarely occurs and frequently a chronic subclinical

by B.L. Larson & V.R. Smith), p. 217. Academic Press, New

disease remains in which small numbers of organisms

York.

are present in the milk, which has a permanently ele-

Cowie, A.T. (1977) *Anatomy and physiology of the udder*. In *Inflamed cell count. These chronic infections may not be* *Milking Machine* (ed. by C.C. Thiel & F.H. Dodd), pp. 156–78. National Institute for Research in Dairying, *low grade inflammatory response in the tissues*.

Reading.

Craven, N. & Williams, M.R. (1985) *Defences of the bovine* *In any consideration of the immunology of the* *mammary gland against infection and prospects for their* *bovine mammary gland, availability of opsonins to per-* *enhancement. Veterinary Immunology and Immunopathol-* *mit the efficient phagocytosis of invading pathogens* *ogy*, **10**, 71–127.

must be considered. The effector mechanisms of the

Hibbitt, K.G., Cole, C.B. & Reiter, B. (1969) *Antimicrobial phagocytes in the* *mammary gland may be activated by*

proteins isolated from the teat canal of the cow. Journal of the Fc region of one *or more classes of immune globu-General Microbiology*, **56**, 365–71.

lin and/or by fragments of the complement system

Larson, B.L. (ed.) (1985) *Lactation. Iowa State University bound to particles.* *Phagocytic cells have receptors for*

Press, Ames, Iowa.

opsonins, where the opsonin is recognized and binds.

Mephram, T.B. (ed.) (1983) Biochemistry of Lactation. Elsevier, This is linked with an effector unit that triggers the cell

Amsterdam, New York.

function. Different species of animals have different

Peel, C.J. & Bauman, D.E. (1987) Somatotropin and lactation.

Journal of Dairy Science, 70, 474–86.

specificities for their receptors for different classes

Poutrel, B. (1982) Susceptibility to mastitis: a review of factors of immune globulin. Non-ruminant neutrophils have

related to the cow. Annales de Recherches Veterinaires, 13, not been shown to bind to cytophilic antibody but

85–99.

cytophilic IgG2 is active for ovine neutrophils and

Weber, A.F., Kitchell, R.L. & Sautter, J.H. (1955) Mammary increases the bacteriocidal activity against Staphylo-gland studies. 1. The identity and characterisation of

coccus aureus. IgG2 from immune bovine serum or milk

the smallest lobule unit in the udder of the dairy cow.

whey will opsonize E. coli. Furthermore, the major

American Journal of Veterinary Science, 16, 255–63.

Chapter 23

Mastitis

P.W. Edmondson and A.J. Bramley

Introduction

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cell counts in affected animals. This can result in a

Dynamics of herd infection

326

raised somatic herd cell count. It is essential to main-

Effects of lactation age and stage

327

tain a low cell count in order to comply with statutory

Teat canal

327

milk quality requirements, e.g. the EU requires all milk

Transmission, sources and control of udder pathogens

328

to be under 400 000 cells/ml, and to ensure that there is

Diagnosis of clinical mastitis

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no penalty to milk price. In the UK, many farmers with

Diagnosis of subclinical mastitis

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cell counts over 150 000/ml are penalized.

Cytological examination

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Bacteriological examination

329

The subclinical stage may be prolonged or proceed

Biochemical and other tests

330

rapidly to clinical mastitis in which external signs of

Use of individual cow or quarter sampling in herd

disease such as swelling and hardness of the affected

investigations

330

gland(s) or clots or discoloration of the secretion are

Cattle housing and mastitis

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present. Nevertheless, the subclinically infected quarter

Machine milking and mastitis

331

has a lowered milk yield, altered milk composition

Milking hygiene

332

and excretes the infecting organism. Subclinical masti-

Aetiology of mastitis

332

tis is a major element in the worldwide economic

Staphylococcus aureus

332

loss associated with mastitis, which exceeds £1/2 billion

Streptococcus agalactiae

333

annually.

Streptococcus dysgalactiae

333

Streptococcus uberis and other aesculin-hydrolysing

streptococci

333

Coliforms

334

Arcanobacterium (Actinomyces, Corynebacterium)

Dynamics of herd infection

pyogenes

334

Mycoplasmas

334

Figure 23.1 illustrates the dynamics of udder disease

Leptospira infection

335

within the herd. The mammary glands can be placed

Coagulase-negative staphylococci and

in one of three categories: uninfected, subclinically

Corynebacterium bovis

335

infected or clinically infected. The relative proportions

Recommendations for control of mastitis

335

of animals in these categories vary between herds.

Pathogen type also influences the dynamics. For

example, coliform infections tend to become rapidly

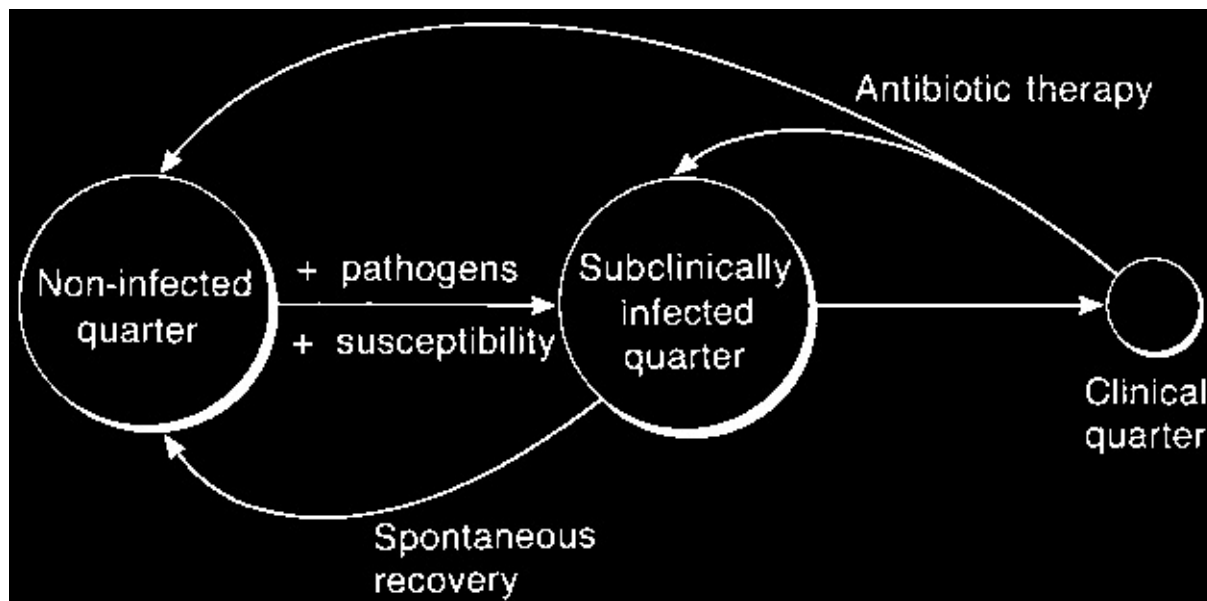
Introduction

clinical while Staphylococcus aureus infections often persist as subclinical infections for weeks or months.

Mastitis is the most prevalent infectious disease of adult dairy cattle. Several species of bacteria, fungi, mycoplasmas and algae have been isolated from the mammary gland. Clinical mastitis will usually be treated with antibiotic therapy and the clinical cure rates are variable and will depend on the pathogen. Bacteriological cure rates vary from 90 per cent for most coliform infections to as low as 25 per cent with *Staphylococcus aureus* in older cows. Table 23.1. Furthermore, many clinical cases of mastitis have been shown to reproduce it naturally. The predominant species are listed in Table 23.1. The mammary gland has a range of defence mechanisms that are due to infection are negative on bacteriological culture of the mammary gland secretion (Dairy Federation 1987a). Spontaneous elimination of infection by these mechanisms will occur. The rates of spontaneous undetectable without the use of diagnostic tests applied

elimination are low for staphylococcal infections (<20 per cent), high for *Escherichia coli* (>70 per cent) and mastitis. Subclinical mastitis results in raised somatic intermediate for streptococcal infections.

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Mastitis • 327

Table 23.1

Species of micro-organism frequently isolated from

Table 23.2

Distribution of clinical coliform mastitis cases with clinical bovine mastitis.

stage of lactation when first found.

Staphylococcus aureus

Escherichia coli

Lactation stage

Number of

Percentage

Streptococcus agalactiae

Klebsiella pneumoniae

when found (weeks)

coliform cases

Streptococcus dysgalactiae

Enterobacter sp.

Streptococcus uberis

Proteus sp.

0–1a

86

20.8

Arcanobacterium

Pseudomonas aeruginosa

2–5b

94

22.9

(*Actinomyces*; *Citrobacter*

sp.

6–10

48

11.6

Corynebacterium)

11–20

58

14.1

pyogenes

21–30

51

12.4

Bacillus cereus

31–40

33

8.0

Mycoplasma bovis

Prototheca

>40

41

10.0

Mycoplasma californicum

Aspergillus sp.

Mycoplasma canadense

Fungi

a Uninfected at drying-off.

b Uninfected at calving.

ronment. The bovine teat canal varies in length from 4 to 18 mm, with a median of 12 mm. It has a heavily keratinized surface and the keratin lining is crucial to the maintenance of its barrier function (Plate 23.1). Keratin traps bacteria as they try to penetrate the teat canal.

Excess keratin is removed by shear forces during the milking process and this is a normal event. If there are poor shear forces, excess keratin and bacteria are not

Fig. 23.1

Pattern of intramammary infection and clinical mastitis removed and may grow up and invade the udder. The mastitis in a dairy herd.

barrier function has both physical and chemical elements. Antibacterial lipids and basic proteins have been identified within the keratin lining of the teat duct

Effects of lactation age and stage

(Williams & Mein, 1985).

The diameter of the canal and the depth of the Udder infection may develop when the cow is lactating keratin layer have been shown to be positively and negatively correlated with infection respectively. Certain although these infections may not persist or develop defects in machine milking operation can adversely into clinical mastitis until the next lactation. Clinical affect the defensive properties of the teat duct, increasing mastitis is most common at calving and in the first three months of lactation. The incidence of clinical disease p. 331 and Chapter 26). Any damage to the teat end or and new infection may increase with lactation number. teat canal is likely to increase the likelihood of new An example for coliform mastitis incidence by stage of infections. This will occur due to a weakened physical lactation is shown in Table 23.2. barrier and also the increased bacterial colonization on

The precise reasons for these effects of age and stage the damaged teat canal.

are not known. The high incidence of mastitis around

Colonization or infection of the streak canal, partic-

calving is largely a consequence of high new infection

ularly adjacent to the teat orifice, is common with

rate in the dry period and a periparturient suppression

pathogens such as Staph. aureus or Streptococcus dys-of host defences.

Increasing disease with age is proba-

galactiae. Such colonizations may persist for long

bly not due to increased intramammary susceptibility

periods in the absence of intramammary infection but

but to increasing ease of penetration of the teat duct by

are largely prevented or eliminated by postmilking teat

pathogens and accumulated previous infections.

disinfection. Other organisms are often present within

the teat duct including Corynebacterium bovis and

coagulase-negative staphylococci and there are reports

Teat canal

of the isolation of anaerobes. The environmental organ-

isms, such as coliforms and Strep. uberis rarely colonize The teat canal is the primary defence mechanism

*the teat duct and this difference is important in the
between the semisterile udder and the outside envi-
pathogenesis of infection.*

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Transmission, sources and control

cial effects on coliform mastitis prevention but does

of udder pathogens

*reduce the rate of new dry-period infection with Strep.
uberis.*

Environmental mastitis is controlled through good

Broadly, the micro-organisms causing bovine mastitis

environmental management, a good milking routine,

can be partly classified into two groups: contagious and

an efficient milking machine, vaccination against col-

environmental.

iform mastitis and premilking teat disinfection.

Contagious mastitis: The udder and teats are the reservoir of infection.

Transmission occurs during the

Diagnosis of clinical mastitis

milking process or udder preparation by contaminated

hands, udder cloths and liners. Infection establishes on

The detection of clinical mastitis depends upon the examination of the surface of the teat and teat canal. Bacteria may then penetrate the mammary gland and its secretion.

The affected gland may show swelling, heat, pain and/or subclinical and result in raised cell counts. Control

measures include postmilking teat disinfection, dryness. The secretion may be clotted, serous or, occasionally, bloodstained (Plate 23.2a,b). In acute cases sys-

temic signs of disease may also be present including

Staphylococcus aureus, coagulase negative Staphs, pyrexia, anorexia and occasionally recumbency in toxic

Streptococcus agalactiae and Strep. dysgalactiae. If a E. coli and Staph. aureus infections. Examples are herd somatic cell count is over 200 000/ml then this indicates that there is a problem with contagious mastitis.

Clinical disease is most commonly seen in lactating

Careful application of control measures based upon animals, particularly in early lactation, but may develop teat disinfection and dry-cow antibiotic therapy will in the non-lactating gland also. Because the dry animal

eradicate Strep. agalactiae over the course of one to is rarely closely scrutinized some of these cases remain

two years provided infected animals are not introduced

undetected until calving. 'Summer mastitis' is an acute

into the herd. Strep. agalactiae can also be eliminated disease of the non-lactating mammary gland and may

through the use of 'blitz' intramammary treatment

affect cows, heifers or young calves. Characteristically,

during lactation and these bacteria are very sensitive to

it is caused by mixed infection with Arcanobacterium

any of the penicillin antibiotics. Levels of Staph. aureus pyogenes and anaerobic bacteria, commonly Peptococ-and Strep. dysgalactiae will also fall to low levels. In cus indolicus.

some herds, poor elimination rates of staphylococcal

It is important (and in many countries statutory) that

infection by therapy will require the heavy culling

dairy cows are examined before each milking for signs

of chronically infected animals to achieve rapid

of clinical mastitis. This allows abnormal milk to be dis-progress.

carded and the affected animal to be given therapy as

rapidly as possible, thereby reducing tissue damage and

Environmental mastitis: The environment is a reservoir giving higher cure rates,

and minimizing the risks of infection. Infections are transmitted onto teats infection transfer. Discarding mastitic milk also helps to between milkings or during udder preparation. Organisms are forced up through the teat canal during the or TBC. An alternative to examination of the foremilk milking process or after milking if cows are allowed to for abnormalities during preparation for milking is to lie down immediately following milking. Most infections cause clinical mastitis. Sub-clinical infections are use in-line filters, installed in the long milk tube to screen the total milk produced. Disadvantages of this less common with *E coli*, but frequently occur with approach are that (i) not all cases of clinical mastitis *Strep uberis*. Environmental mastitis is controlled by result in the formation of clots that are trapped in the provision of a clean environment, adequate accommodation for cows, milking through correctly functioning filters; (ii) the affected quarter(s) have to be identified by an additional examination of the strippings milk; (iii)

machine, good udder preparation and pre-milking teat disinfection. Environmental organisms include *E coli*, *Streptococcus uberis* (straw bedding), *Klebsiella* spp. milking systems and (iv) many milkers fail to check the filters. Palpation of the udder after milking also aids there is potential for interquarter transfer at milking clinical mastitis detection but the increase in cows time it appears not to be the predominant infection milked per person hour and the provision of automatic mechanism. Postmilking teat disinfection does not cluster removers have precluded this process in many prevent infection. Antibiotic therapy has some benefit-parlour operations. Some milking equipment contains Mastitis • 329 sensors to aid the detection of abnormal milk and this using a variety of tests such as the CMT or Whiteside is likely to become more common. test. These tests are based upon a gelling reaction

between the nucleic acid of the cells and a reagent, either a detergent (CMT) or sodium hydroxide (White-

Diagnosis of subclinical mastitis

side). The reactions are categorized from 0 to 4. Reactions of 3 and 4 have a high probability of infection

A range of tests can be applied to milk to detect sub-being present. The advantage of the count is that the clinical mastitis (Schultze, 1985). These tests generally results are available immediately, are repeatable, cheap measure the somatic cell count or detect the presence and give an individual quarter result. Many countries of infecting organisms. These changes are both cytolog-employ cell counting of herd bulk milk as an estimate ical and biochemical. Most tests are laboratory based of herd mastitis levels. Individual cow milk cell count although the California Mastitis Test (CMT) and elec-services are available in many countries as an intermit-trical conductivity measurements can be used at the tent or regular service (e.g. National Milk Records in cow side. Tests can be placed in three categories and England and Wales and the Dairy Herd Improvement

applied either alone or in combination.

Association in the USA). A consistently high bulk milk cell count is characteristic of a high level of subclinical infection in the herd. Herds with counts <200 000

Cytological examination (see p. 323;

cells/ml generally have little economic loss associated Chapter 25)

with subclinical disease. Such herds may however still

Milk from a healthy, uninfected bovine mammary

suffer significant costs due to a high incidence of clini-

gland contains somatic cells comprising macrophages,

cal mastitis, associated with short-duration infections

neutrophils and lymphocytes. The number of these is

such as coliforms.

usually <50 000 cells/ml milk. When mastitis is present

the cell count increases, primarily as a consequence of

Bacteriological examination

neutrophil infiltration. Subclinically infected quarters

have cell counts in excess of 200 00 cells/ml but, because

The secretion from a normal mammary gland is sterile

of the dynamic nature of the disease, this value can vary

although it may acquire bacteria from a colonized teat considerably and will sometimes fall below this threshold during collection. Therefore, the detection of a

old (Table 23.3).

pathogen in an aseptically collected milk sample is

During clinical episodes the cell count will be indicative of infection. The number of organisms excreted per millilitre of milk from an infected gland monplace. The cell count is detectable by a microscopic fluctuates widely; hence the recovery of even low count or by electronic means. Detailed descriptions of numbers of bacteria can be regarded as meaningful if the methods employed are available (Dairy Federation, 1984). An indirect estimate of cell counts can be made not contaminated. Bacteriological examinations have the advantage of being positive or negative, whereas most other tests require the imposition of a threshold

Table 23.3

Variation in the cell count of a quarter infected with

value which varies between animals and herds (Dairy

Staph. aureus compared to an uninfected quarter in a Friesian Federation, 1987a).

heifer.

A suitable sample is required for bacteriological examination obtained by an aseptic technique. Samples

Milking No.

Somatic cell count C \leq 1000 cells/ml

can be frozen (-20°C) prior to examination although this reduces pathogen numbers (particularly Gram-

Control quarter

Infected quarter

negatives) and precludes cytological and some bio-

1

13

424

chemical examinations. The bacteria most commonly

2

80

3943

causing bovine mastitis will grow aerobically on blood

3

64

528

agar. The more unusual causes of mastitis such as anaer-

4

84

4046

obes, Mycoplasma spp. and Leptospira spp. require 5

44

797

more specialized techniques not appropriate to the

6

91

4540

practice laboratory. The inclusion of 0.1 per cent aes-

7

107

1471

culin in the blood agar is useful in the differentiation of

8

61

1446

Strep. uberis from other streptococci. A volume of milk 9

64

528

is spread over the surface of the agar plate, which

10

84

4046

should be incubated for 24–48 hours at 37°C. An

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advantage of a bacteriological examination is that the

Table 23.4

*Changes in milk composition associated with mas-
causative organism can be identified and provides a
titis (examples of ‘typical values’).*

*possibility for antimicrobial sensitivity testing. Some
organisms such as Staph. aureus may be shed intermit-*

Somatic cells (1000)

tently and in small numbers in milk. With these infec-

Normal

Mastitic

tions, a negative result may indicate that the bacteria

< 20 to 75

100 to > 10 000

have not been isolated due to a dilution effect. Repeat sampling may be necessary in order to confirm a nega-

Sodium (mg/100 ml)

57

104

tive result. Disadvantages include cost, complexity and

Chloride (mg/100 ml)

100

200

time delay. Detailed methods for the collection and bac-

Potassium (mg/100 ml)

170

150

teriological examination of milk samples for infection

Conductivity (mM NaCl)

<50

>56

are available (Dairy Federation, 1981).

Lactose (mg/ml)

48

44

Catalase

increased 20-fold

NAG

increased 6-fold

Biochemical and other tests

BSA (mg/ml)

0.25

>0.6

Inflammation of the mammary gland leads to a variety

NAG, N-acetyl glucosaminidase; BSA, bovine serum albumin.

of compositional changes in milk either because of local

effects or because of an increase of serum components

entering the milk. Some of these changes can be used

to screen for subclinical mastitis.

tests and their application the reader is referred to

Significant changes in ionic composition occur with

Schultze (1985).

both Na⁺ and Cl⁻ levels increasing, K⁺ decreasing. The

individual ions can be directly determined but more commonly the net change in ionic composition is used,

Use of individual cow or quarter

measured as an increase in electrical conductivity. Both

sampling in herd investigations

laboratory and farm-based meters exist and milking

equipment incorporating electrodes to measure milk

Mastitis control relies upon the application of effective

conductivity during milking is marketed in some coun-

control measures to the herd rather than identification

tries. There are a variety of other factors which may

or special treatment of individual animals. It is essen-

ential to identify the organisms responsible for the

ity of electrical conductivity meters work on pooled

problem and in high cell count herds, the proportion of

milk from all quarters and are unlikely to be very accu-

cows infected. This requires bacteriology and individual

rate due to a dilution effect. However, as for the major-

cow cell count data which will help to identify the

ity of the milk parameters, the conductivity values of

nature of a herd problem or individual animals for segregation and infected quarters overlap and conductivity regulation or culling. As payment of milk on the basis of values vary between herds. These facts make the application of cell count becomes more common than the emphasis of threshold values for diagnosis inaccurate. A placed on these systems tends to increase.

fall in lactose concentration is also usual and has been If the aetiology of clinical mastitis is to be determined employed diagnostically but has poor discrimination. then a microbiological analysis is needed. Such Enzymatic changes also occur. Some of the enzyme analyses provide information on the patterns of the changes are associated with the increase in cell count disease within a herd since there will be delay between (e.g. catalase), others with secretory cell damage (e.g. sampling and diagnosis during which therapy will part of N-acetylglucosaminidase (NAG) increase) or usually have begun. Often a broad-spectrum antibiotic with increased permeability (plasmin). The use of NAG is employed to cover the range of possibilities.

has been widely tested and found to correlate well with
However, the pattern of disease may be helpful in
cell count and infection and offers promise as a cow-
determining the attention needed to remedy the herd
side test.

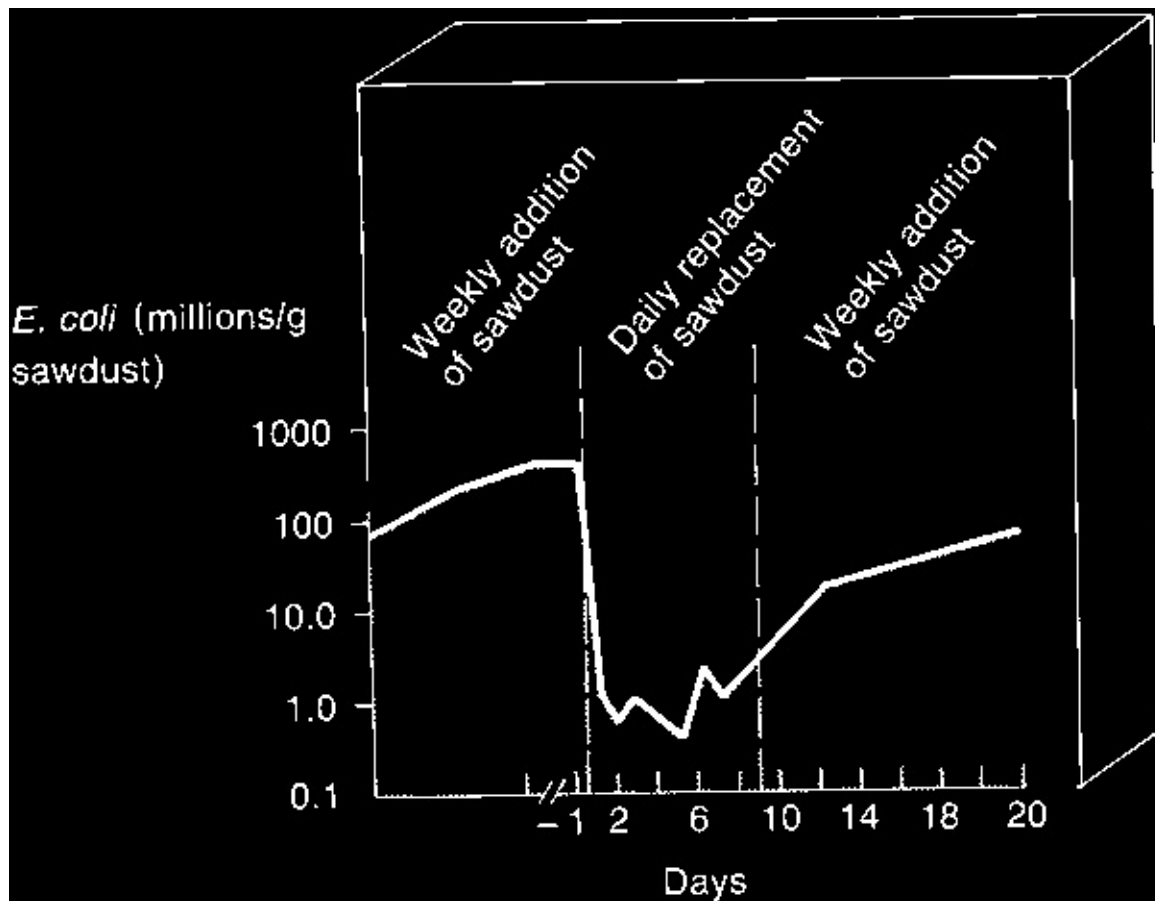
problem. The isolation of *Strep. agalactiae* indicates As serum components leak
into the mastitic gland

either that teat disinfection and dry-cow therapy are
they can be employed as indicators of inflammation.
not being employed effectively or infected animals have

The most studied have been antitrypsin and bovine
been introduced. A pattern of high herd cell count,
serum albumin (BSA). Automated methods for the
repeated clinical cases and the frequent isolation of
detection of antitrypsin have been developed and the

Staph. aureus would be typical of poor elimination of test is more sensitive than
BSA. Table 23.4 summarizes

infection by lactation or dry-period therapy. This might
the major compositional changes in milk due to udder
prompt the use of bacteriological analysis of samples
infection and inflammation. For more detail of these
taken after treatment or at drying-off and calving.



Mastitis • 331

Individual cow cell count or other diagnostic tests can be used to identify chronically infected animals for culling, treatment during lactation and other options, although such animals might be identifiable via an examination of clinical records. In the majority of herds the records are unfortunately inadequate for such a purpose. When using individual cow cell counts or other indirect tests the variation in inflammation should be remembered. Confident diagnosis on a single sample

is not possible. The changes in these measures due to lactation stage and yield are also relevant. Cell counts in colostrum and in dry-cow secretion are naturally elevated and diagnostically unreliable. As lactation declines and yield falls cell count and electrical conductivity increase slightly. Milk composition and cell count may remain permanently altered or elevated in

Fig. 23.2

Controlling coliform contamination of sawdust in glands that have suffered severe secretory tissue cubicles by daily removal and replacement.

damage, even if the causative organism has been successfully eliminated (see Chapter 25).

and replacement of bedding offers the soundest basis for reducing it (Fig. 23.2; Dodd et al., 1984). Herds which house cattle in straw yards should clean these out

Cattle housing and mastitis

every 4 to 6 weeks. In mild weather, this may need to be reduced to every 3 to 4 weeks.

In many countries climatic conditions require that

The climate within cattle housing is also important

cattle are housed for at least part of the year with con-
since high humidity favours the survival and multipli-
sequences for mastitis. In some countries cows are
cation of micro-organisms. It has been found that cattle
housed all year round. Housing increases the risk of
litter generally appearing to be clean and dry can
mastitis because the confinement of the animals and the
harbour high numbers of micro-organisms and this can
multiplication of micro-organisms in various litters
pose significant problems when giving advice over herd
elevate teat challenge, and consequently mastitis.
mastitis problems.

Prominent among these bacteria are the coliforms,
The design and size of yards and cubicles influence
Strep. uberis and faecal streptococci.

cow cleanliness and thus milk bacteriological quality and
The relationship between housing and mastitis is
mastitis. Cubicles that are too short will leave cows'
most clearly established for coliform mastitis. Coliform
udders lying in the alleyway while those which are too
bacteria have been shown to multiply readily in organic

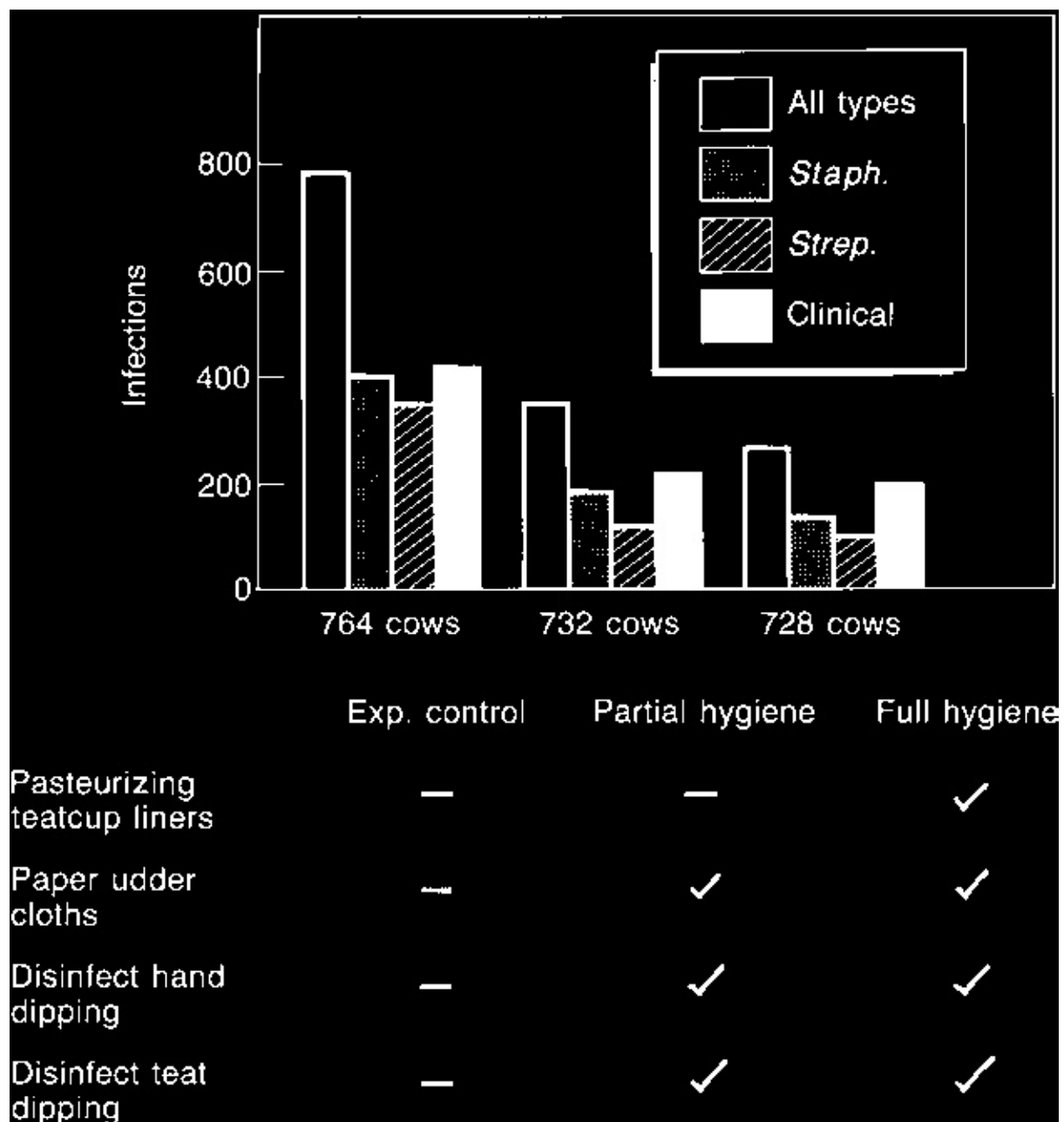
long will lead to dunging on the cubicle surface itself. litters, particularly sawdust, reaching counts of >100 million/g. Obviously, the correct dimensions vary depending upon breed and age. There should be 5 to 10 per cent more bovine faeces and impose a risk of infection. Sawdust cubicles than cows. In loose housing, a lying area of 6 m² has been shown to act as a source of *Klebsiella pneumoniae*, particularly if not kiln-dried, and can lead to loose yards will lead to an increase in environmental severe mastitis problems. Some outbreaks have been infections, in particular *Strep. uberis*. Improper place-controlled by switching from sawdust to sand, which ment of access gates and water troughs in yards will lead tends to harbour lower numbers of coliforms and to excessively dirty lying areas. Use of dung channels and reduces the risk of disease. grids can increase the incidence of damaged teats, etc. *Streptococcus uberis* colonizes the bovine gut and is These and other ways in which the environment influ- excreted in faeces and consequently contaminates

ences mastitis are the subject of a review by the Inter-cattle litter. In straw, multiplication of the organism national Dairy Federation (Dairy Federation, 1987b). often occurs leading to high levels of teat challenge and Detailed information on suitable dimensions and increased infection rates (Bramley, 1982). Since Strep. designs for cattle housing are also available (pp. 42–4). uberis is particularly infectious for the non-lactating or parturient udder this can be a particular problem if dry-cow or calving accommodation is not adequately

Machine milking and mastitis

cleaned and bedded.

Since multiplication in the litter is a crucial factor in There is unequivocal evidence that the events occurring increasing challenge to the teat surface, regular removal at milking time influence the incidence of mastitis.



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These influences may be via the hygiene practised at

milking time or because of effects of the milking

machine per se. For details on how the milking machine can affect mastitis see Chapter 26.

Milking hygiene

The milk secreted by infected cows contains varying numbers of pathogenic micro-organisms. Herd milking provides opportunities for the transmission of these bacteria between udder quarters and cows via the milking machine itself, the milker's hands or cloths. Various techniques can be employed at milking time to reduce transmission including:

(1)

Using disinfected water for teat washing.

(2)

Employing individual paper towels or cloths for teat drying.

(3)

Wearing clean rubber gloves.

(4)

Applying a suitable disinfectant to the teat surface after milking.

(5)

Disinfecting or heat treating the cluster between

Fig. 23.3

*Effect of milking time hygiene on new udder infections.
milking cows.*

(6)

Premilking teat disinfection.

*In addition, segregation of infected animals from the
herd or TBC. There will be an increased risk of liner
healthy herd can be used although it is impractical in
slip and impact forces at the teat end. These factors will
most herds and requires the use of individual cow cell
increase the risk of environmental infections. Ideally,
counts or a similar test. The use of measures such as
a suitable concentration of an approved disinfectant
those described above can reduce infection rates by
(usually iodine or chlorine) should be included in the
about 50 per cent, effects being greatest against Staph.
wash water supply. There have been examples of col-
aureus, Strep. agalactiae and Strep. dysgalactiae (Fig.
iform and Pseudomonas mastitis problems associated
23.3). The measures are less effective against coliforms
with contaminated udder wash water supplies. These
and Strep. uberis infection because of their wider dis-are unlikely where mains*

water is in use although

tribution in the environment.

uncovered water storage tanks may become contami-

Of the various measures to prevent new intramam-

nated with faeces or carcasses of small animals and

many infections among milking cows, postmilking teat

provide a source of infection. For a review of this

disinfection is the most valuable. Various disinfectants

subject see Bramley & McKinnon (1990)

are used, most commonly iodophors, chlorhexidine and

hypochlorite. Emollients are often added to promote

good skin condition and rapid healing of lesions. These

Aetiology of mastitis

disinfectants will rapidly destroy bacteria reaching the

teat surface during milking and will prevent coloniza-

It has already been stated that mastitis is a consequence

tion of the teat duct and the infection of teat lesions.

of intramammary infection with one or more microor-

The speed of kill of the postmilking teat disinfectant is

ganisms of many different genera. The following section

not important provided that it is effective. This is con-

provides information on the most common or important to the action of premilking disinfectants which of the different mammary infections.

must have a rapid speed of kill. This fast action is required to avoid slowing down the milking procedure.

Staphylococcus aureus (see p. 385)

The importance of premilking hygiene in the production of milk of high bacteriological quality should In many countries this organism is the predominant be emphasized. Data show that effective washing of the cause of subclinical mastitis but is also isolated from teat followed by drying significantly reduces the bacterial clinical disease. The important sources of infection are bacterial content of bulk milk. Washing without drying will infected udders, teat ducts or teat lesions. Extramammary result in bacteria being put in suspension. This solution many sources do exist, notably the vagina and tonsils will be sucked in with the milk and increase the bacterial but do not appear important in the pathogenesis of Mastitis • 333

infection. It has proved possible to eradicate Staph.

tion. The duration of infection is shorter than staphylo-
aureus mastitis from individual herds by the application cocci, primarily because
of the better response to
of hygiene at milking time and culling. The major limi-
therapy. More rapid elimination of *Strep. agalactiae* can tation to more effective
control of *Staph. aureus* masti-be achieved by diagnosis and treatment of
infected
tis remains its poor response to antibiotic therapy. Cure
animals although the economic basis for this is doubt-
rates are highest in first and second lactation animals.
ful (for therapy see p. 396).

In older cows cure rates during lactation may be as low
as 25 per cent following intramammary antibiotic

Streptococcus dysgalactiae (see p. 387)

therapy. Even following the use of dry-cow therapy,
This organism has both contagious and environmental
cure rates of 33 per cent in older cows are common.
characteristics often associated with poor teat skin con-
There are a variety of reasons for this of which antibi-
dition. Very good control of lactation infections can be
otic resistance plays only a minor role. In vitro demon-achieved by teat
disinfection indicating that cow-to-cow

stration of sensitivity of *Staph. aureus* to an antibiotic transfer is an important

mechanism. However, infection is no guarantee of therapeutic success. The ability of the bacteria to survive inside polymorphonucleocytes particularly among animals not protected by dry-cow therapy. Outbreaks of clinical mastitis with *Strep.* from antibiotic action, may significantly contribute to dysgalactiae frequently follow a breakdown in herd hygiene practices or increases in teat lesions. The incidence of teat lesions can increase rapidly following failure of *Staph. aureus* infections renders chronically infected cows essentially incurable. Most commonly housing or climatic conditions. In such outbreaks the *Staph. aureus* udder infection is chronic, acute mastitis machine should be given a thorough examination by an expert and the teats closely inspected for damage. The

acute gangrenous *Staph. aureus* infections can arise in inclusion of a high level of a suitable emollient in the

which uncontrolled growth of the organism occurs elab-

teat dip can promote rapid healing of lesions and

orating large quantities of a toxin. Such infections are

prevent their colonization.

probably not due to strains of increased virulence but

Clinical *Strep. dysgalactiae* mastitis can be acute with rather to a failure by the host to mount an effective

anorexia and pyrexia in addition to the local signs.

defence. Workers in California showed the critical role

However, the response to therapy is usually rapid and

of the PMN in this defence since subclinically infected

elimination rates with penicillins are similarly high to

cows, made neutropenic, rapidly developed acute gan-

Strep. agalactiae. *Streptococcus dysgalactiae* is also grenous *Staph. aureus* mastitis (Schalm et al., 1976) (for encountered in mixed culture with other organisms,

therapy see p. 396)

notably *A. pyogenes* and *P. indolicus* in summer mastitis. The bacteria can be isolated from bovine tonsils and

Streptococcus agalactiae (see p. 387)

the bovine genital tract and these sources, allied to the

ability of the organism to infect and colonize lesions,

The sources of *Strep. agalactiae* are similar to those of may be important in the pathogenesis of dry-period

Staph. aureus, namely the teats and udder. Consequently, milking time hygiene is an effective means of preventing infection during lactation. The presence of

***Streptococcus uberis* and other**

Strep. agalactiae in a herd suggests that there are defects

aesculin-hydrolysing streptococci (see

in milking hygiene, postmilking teat disinfection and/or p. 387)

dry-cow therapy. Since *Strep. agalactiae* is easily eliminated by intramammary antibiotic therapy it is elimi-

These organisms are an important cause of bovine mastitis and, in some herds, are the major cause of clinical

dry-cow therapy effectively and routinely. It is possible

disease. Several species can be involved but the pre-

to include the elimination of infection by blitz therapy

dominant one (>70 per cent) is *Strep. uberis*. Others during lactation. Blitz therapy is where all quarters of

include *Strep. bovis*, *Strep. faecalis* and *Strep. faecium*.
infected cows are treated with intramammary anti-
Infection with these organisms is not controlled by
biotics. The whole herd may be treated or selected
postmilking teat disinfection although dry-cow antibi-
animals. The level of infection relates well to individual
otic therapy reduces infection rates by about 50 per
cow cell counts. It is important to confirm that *Strep.*
cent. However, *Strep. uberis* remains the commonest
agalactiae is the cause of the high cell count before cause of infection in the dry
period and susceptibility to
embarking on blitz therapy. The disease may exist as an
infection has been shown to increase following drying-
acute clinical mastitis or persist as a subclinical infec-
off (Marshall et al., 1986). In untreated dry cows the
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highest rates of infection occur in the initial two weeks
high-yielding cows, this is not effective, possibly because
following drying-off while in animals given antibiotic
the rate of neutrophil diapedesis is poor. Recent
therapy at drying-off infection tends to be periparturi-

research indicates that low levels of selenium may be ent. The high infection rates in the dry period and the implicated in the process (Erskine et al., 1989) (for failure of post milking teat disinfection to control therapy see p. 396)

disease emphasize the independence of milking and transmission. The bacteria are widely disseminated on

Arcanobacterium (Actinomyces,

the cow, are present in low numbers in cattle faeces and

Corynebacterium) pyogenes (see Chapter 24)

multiply in bedding materials, particularly straw, to

reach high levels. Deep straw yards for housing dry

This bacterium is often involved in suppurative condi-

cows seem to pose a particular risk factor. Not all

tions in cattle, including mastitis. It is most frequently

strains are virulent for the lactating gland and a capsu-

encountered as one of the mixture of pathogens respon-

lar layer is often elaborated conferring resistance to

sible for 'summer mastitis' in northern Europe. This

phagocytosis. Susceptibility of the lactating gland to

acute clinical disease of the non-lactating animal has

infection may be influenced by the lactoperoxidase

been described in the section on diagnosis (see p. 328).

system, which in turn can be affected by feeding regi-

Arcanobacterium pyogenes may also infect the lactating menses altering milk thiocyanate levels. Although *Strep.*

cow, or infection may be carried over from the dry

udder is sensitive in vitro to a range of antibiotics, period. Infection in the lactating cow is usually associated with intramammary therapy often is ineffective and chronic

associated with teat damage.

infections are common in some herds. Under these

The epidemiology of summer mastitis has been much

circumstances cow-to-cow transmission may become

studied because of its epidemic and destructive nature.

more important (for therapy see p. 396)

The disease has been associated with fly-borne trans-

mission and the sheep head fly *Hydrotaea irritans* has been shown able to carry *A. pyogenes*, *Strep. dysgalac-*

Coliforms (see pp. 383, 1016)

and the anaerobic peptococci responsible for the The coliform species most commonly implicated in

disease. With one exception transmission experiments

mastitis are *E. coli*, although other species including with infected flies have proved unsuccessful but data do

Enterobacter aerogenes, Pseudomonas aeruginosa, Ent.

reveal that the use of insecticides or barriers (surgical

oxytoca, K. pneumoniae and Citrobacter spp. are also tape, Stockholm tar, etc.) reduces the disease incidence.

encountered. Infection is more common among housed

The most effective control measure is the prophylactic

cows and occurs particularly around calving. Subclini-

use of dry-cow therapy. A review of the epidemiology,

cal infection is less common and peracute mastitis

transmission and pathogenesis has been published

occurs frequently around calving. About 50 per cent

(Thomas et al., 1987).

of these peracute infections die. Treatment involves

frequent stripping to remove endotoxin, antibiotic,

Mycoplasmas (see p. 385)

supportive therapy including hypertonic saline and

oxytocin, etc. The use of anti-inflammatory drugs, par-

In recent years several species of Mycoplasma have

ticularly non-steroidal anti-inflammatories, may also be

been recognized as important causes of bovine mastitis.

helpful. Many new E. coli infections occur during the The most common cause is Mycoplasma bovis but other

dry period but do not manifest as a clinical disease until species implicated include *M. bovigenitalium*, *M. canadense*, *M. californicum* and *M. alkalescens*. The incidence of coliform mastitis has increased despite the adoption of the National Characteristically, infection with *Mycoplasma* spp. Institute for Research in Dairying (NIRD) five-point leads to a mastitis, often involving multiple quarters, plan, improved milking machines and better hygiene. which is refractory to antibiotic treatment. The secretion Transmission between cows is unimportant in pathogenesis may remain normal at the onset although a granular or flaky deposit is recognized on standing. In the dry-cow therapy does not reduce infection rates. The later stages a purulent or thick secretion, without offensive primary source of infection is bovine faeces although sive smell is often reported. Swelling and firmness is secondary multiplication of the bacteria to high common but after a few days the mammary gland may numbers in cattle litter is often a factor (see section on

reduce in size. Milk secretion is severely reduced.

cattle housing and mastitis, p. 331).

Swelling of the supramammary lymph nodes occurs and

Infection with coliform bacteria leads to a rapid

there may be pyrexia, transient malaise and arthritis.

development of inflammation and often the influx of

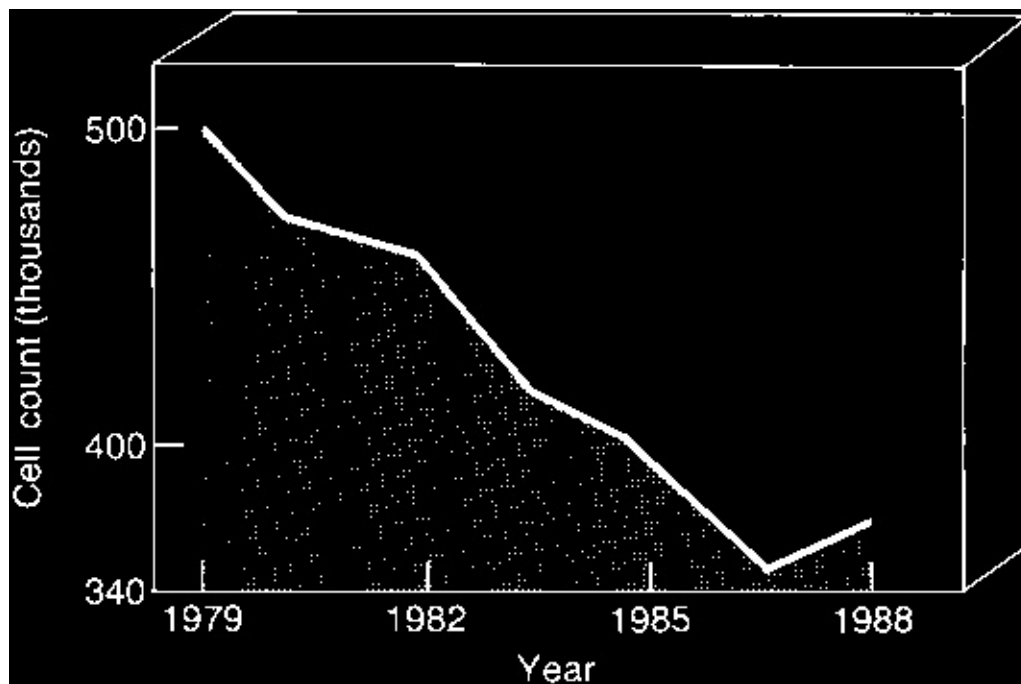
Secretion of mycoplasmas in the milk frequently lasts

neutrophils eliminates the infection (Hill et al., 1979).

for two months and often for longer. Intermittent shed-

In some animals, particularly in early lactation and in

ding is common. Clearly, such cows constitute an infec-



tion risk for the herd. Most reports relate to infection

These include *Staph. epidermidis*, *Staph. simulans* and in lactating cows but outbreaks among dry cows,

Staph. xylosus. Most of these organisms are of low vir-notably with *M. californicum*, do occur (Jasper, 1982).

ulence although some isolations are associated with

Diagnosis requires the application of specific micro-

clinical disease. These organisms generally produce a

biological and serological tests in specialist veterinary

slight elevation of somatic cell count, which may

diagnostic laboratories. These involve culture from milk

increase resistance to infection with major pathogens.

onto selective media and a range of serological tests,

Similarly, *Corynebacterium bovis* is not usually asso-

often growth inhibition, to identify the species.

ciated with clinical disease and primarily colonizes the

The most severe problems with this disease have

distal teat duct. It is associated with suboptimal teat dis-

occurred in large dairy herds in California and Florida.

infection. It is rapidly eliminated by antibiotic therapy

It should be considered as highly infectious and

and its spread is prevented by effective teat disinfect-

affected animals either removed or isolated within the
tion. In the absence of such measures it may be isolated
herd. Examination of bulk milk has proved a useful
from >70 per cent of aseptically collected milk samples.
screening method for affected herds. If a herd is sus-
Growth on blood agar may require incubation for 48
pected of having a problem with *Mycoplasma mastitis*
hours, preferably at 30°C, and stimulation by fatty acids
then specialist advice should be rapidly sought. For
supplied either by the milk or by additions to the media
further information the reader is referred to detailed
such as Tween 80.

reviews (Boughton, 1979; Jasper, 1982) (for therapy see
p. 396)

Recommendations for control

***Leptospira* infection** (see also p. 737)

of mastitis

Increasing concern has arisen over leptospirosis for a
variety of reasons. Most importantly organisms of the
In the USA, UK and Australasia and many other parts
hebdomadis subgroup are pathogenic for man. The

of the world there is a recommended mastitis control

organism most involved is Leptospira hardjo and this is scheme based upon the '5-point plan'. The elements in

responsible for the so-called 'milk drop syndrome' and

the scheme are as follows:

is also associated with abortion in affected herds. The

(1)

Apply an approved teat disinfectant after every

characteristic milk drop appears initially as a mastitis, milking.

usually with the milk yield of all four quarters falling to

(2)

Treat clinical cases of mastitis.

zero within 24 hours. Pyrexia is common and the udder

(3)

Infuse long-acting antibiotic into all quarters at

secretion is thickened or clotted and occasionally drying-off.

bloody. The udder remains flaccid and the condition

(4)

An annual milking machine test and appropriate

usually resolves within seven to fourteen days. Cases maintenance.

may be restricted to a few animals or up to 50 per cent (5)

Cull cows showing repeated cases of clinical of animals may become affected over a period of two mastitis.

to three months (Jackson & Bramley, 1983). Antibiotic therapy with tetracyclines or streptomycin may aid the These measures were the basis of controlled experimentation in many countries and have been widely although immunity develops following infection. A employed since the 1970s. Teat disinfection is to reduce vaccine is available, which requires an annual booster to ensure adequate protection. Infection of human beings may occur via infected urine droplets, particularly in herringbone milking parlours and leads to a febrile illness characterized by severe headaches. In a proportion of cases this can lead to complications including meningitis and encephalitis.

Coagulase-negative staphylococci and

Corynebacterium bovis

The incidence of coagulase-negative staphylococci has increased as herd cell counts have decreased. Several species of coagulase-negative staphylococci are com-

Fig. 23.4

*Reduction in bulk milk cell count in England and
monly isolated from aseptically collected milk samples.*

Wales between 1979 and 1988.

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rates of new infection during lactation; the dry-cow

Erskine, R.J., Eberhart, R.J., Grasso, P.J. & Scholz, R.W. (1989) therapy serves to eliminate a high proportion of sub-Induction of Escherichia coli mastitis in cows fed selenium-clinical infections present at the end of lactation and to deficient or selenium-supplemented diets.

American

prevent many new dry-period infections. Treatment of

Journal of Veterinary Research, 50, 2093–2100.

clinical mastitis assists the elimination of infection and

Hill, A.W., Shears, A.L. & Hibbitt, K. (1979) The pathogenesis of experimental Escherichia coli mastitis in newly in the resolution of clinical signs of disease and culling

calved dairy cows. Research in Veterinary Science, 26, is employed to remove

chronically affected cows.

97–101.

Finally, the milking machine maintenance is intended to

Jackson, E.R. & Bramley, A.J. (1983) Coliform mastitis.

ensure efficient milking and prevent machine-induced

Veterinary Record (In Practice Suppl., Vol. 5, No. 4) 135–

infections as described above (therapy for mastitis is

46.

dealt with in Chapter 30).

Jasper, D.E. (1982) The role of Mycoplasma in bovine masti-A review of the progress in mastitis control follow-

tis. Journal of the American Veterinary Medical Association, ing the introduction of these techniques has been

181, 158–73.

described by Booth (1988). This shows major reduc-

Marshall, V.M.E., Cole, W.M. & Bramley, A.J. (1986) Influence tions in clinical mastitis incidence and in the reduction

of the lactoperoxidase system on susceptibility of the udder

to Streptococcus uberis infection. Journal of Dairy Research, of bulk milk cell count (Fig. 23.4). The present status of

53, 507–14.

improving mammary gland immunity is described in

Schalm, O.W., Lasmanis, J. & Jain, N.C. (1976) Conversion of Chapter 29.

chronic staphylococcal mastitis to acute mastitis after neutropenia in blood and bone marrow produced by an equine anti-bovine leukocyte serum. American Journal of Veteri-

References

nary Research, 37, 885–94.

Schultze, W.D. (1985) Development in the identification of dis-Booth, J.M. (1988) 1. Control measures in England and Wales.

eased udder quarters or cows. Proceedings International

How have they influenced incidence and aetiology? British

Dairy Federation Seminar Progress in the Control of Bovine Veterinary Journal, 144, 316–22.

Mastitis. Kieler Milchwirtschaftliche Forschungsberichte, 37, Boughton, E. (1979) Mycoplasma bovis mastitis. Veterinary 319–28.

Bulletin, 49, 377–87.

Thomas, G., Over, H.J., Vecht, U. & Nansen, P. (eds) (1987) Bramley, A.J. (1982) Sources of Streptococcus uberis in the Summer Mastitis. Martinus Nijhoff, the Netherlands,

dairy herd. I. Isolation from bovine faeces and from straw 1–224.

bedding of cattle. Journal of Dairy Research, 49, 369–73.

Williams, D.M. & Mein, G.A. (1985) The role of machine

Bramley, A.J. & McKinnon, C.H. (1990) The microbiology milking in the invasion of mastitis organisms and implica-

of raw milk. In *Dairy Microbiology*, Vol. 1 (ed. by R.K. Robinson). Academic Press, New York.

national Dairy Federation Seminar Progress in the Control of Bovine Mastitis. Kieler Milchwirtschaftliche Forschungstitis work. International Dairy Federation Bulletin 132, berichte, 37, 415–25.

Brussels.

Dairy Federation (1984) Recommended methods for somatic cell counting in milk. International Dairy Federation Document 168, Brussels.

Further reading

Dairy Federation (1987a) Bovine mastitis. Definition and guidelines for diagnosis. International Dairy Federation

Blowey, R.W. & Edmondson, P.W. (1995) Mastitis Control Bulletin 211, Brussels.

in Dairy Herds. Miller Freeman, Ipswich, UK, pp. 1–

Dairy Federation (1987b) Environmental influences on mastitis. International Dairy Federation Bulletin 217, Brussels.

Bramley, A.J. & Dodd, F.H. (1984) Mastitis control—progress Dodd, F.H., Higgs, T.M. & Bramley, A.J. (1984) Cubicle man-and prospects. *Journal of Dairy*

Research, **51**, 481–512.

agement and coliform mastitis. Veterinary Record, **114**, Schalm, O.W., Carroll. E.J. & Jain, N.C. (1971) *Bovine Masti*-522–3.

tis. Lea & Febiger, Philadelphia, pp. 1–360.

Chapter 24

Summer Mastitis

J.E. Hillerton

Aetiology

337

Japan, and parts of the USA, as well as being reported

Epidemiology

337

from Greece, Australia, Zimbabwe and Brazil. The inci-

Transmission

337

dence varies greatly from country to country, as does

Pathogenesis

338

the severity of the infection. Most cases in North

Clinical disease

338

America are recognized when heifers calve with a non-

Treatment

339

functional quarter from which the causative bacteria

Control

339

can be recovered, whilst in Japan systemic illness is more common.

The incidence of summer mastitis varies locally. It is

Summer mastitis is an acute or peracute multifactorial

greater in the more intensive dairy areas but does not

infection of the non-lactating bovine mammary gland

just depend on the density of cattle. It is only in the

although clinical signs are often not obvious until par-

dairy industry that the importance of the disease is well

turition. The disease is reported from all four continents

recognized. It can be prevalent in beef cattle but loss of

where dairy cattle are herded. It is, however, most

function of one-quarter is not so important economi-

prevalent in northern Europe and particularly common

cally in the suckler cow. The Dutch, Germans and

in some European cattle such as the Friesian/Holstein Danes believe that summer mastitis is associated breeds, being rare in the Zebu.

with sandy soils, but these correlate well with the best grassland. The high humus content, open structure and free-draining of sandy soils are particularly suitable

Aetiology (see p. 334)

for foraging, soil-dwelling insect larvae.

The disease is most common in summer and early

Bacteriological analysis of summer mastitis secretion

autumn but can also occur in spring. As well as season,

shows a complex infection. Usually Arcanobacterium

it is particularly associated with calving pattern. In

(Corynebacterium) pyogenes predominates and the Ireland, with many spring-calving herds, the spring inci-severity of the infection is related to toxin production

dence is marked. The black and white dairy breeds

from synergistic growth with other bacteria that are

seem most susceptible and in the absence of preventive

anaerobic (see p. 338). Peptococcus indolicus is the

measures the incidence is greatest in older cows.

most common anaerobe but Bacteriodes melaninogeni-

cus and Fusobacterium necrophorum are often found.

An undescribed microaerophilic coccus, the Stuart–

Transmission

Schwann coccus is also found. It rarely grows in pure

subculture. Streptococcus dysgalactiae is also common. It is a long and widely held belief that flies are involved

and believed by some to be a primary agent predisposing to A. pyogenes infection.

The peak incidence of disease in northern Europe

Much of the reported variation in bacterial culture,

occurs in July, August and September when flies are

and especially isolation of anaerobes, probably results

most abundant on cattle. The sheep headfly (or planta-

from suboptimal collection and transport of samples,

tion fly), Hydrotaea irritans, is the most frequent visitor allowing anaerobes to die out.

to the teats of dry cows and heifers. Flies around pas-

tured cattle have been shown to carry the summer mas-

titis pathogens even in the absence of disease in

Epidemiology

the herd. The disease prevalence is coincident with the

geographical distribution of the fly in most of Europe.

Summer mastitis is considered to be epidemiologically

Control of flies on cattle reduces the incidence of

most severe in northern Europe. It is also prevalent in

summer mastitis. Some 60 per cent of infections occur

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in the front quarters, which, it has been suggested, flies

*the teat with *A. pyogenes* alone. There is, however, no can reach more easily.*

unequivocally accepted mechanism. Possibilities, each

*Various studies using *Hydrotaea irritans* to transmit*

with proponents, include colonization of the teat skin

summer mastitis have given incomplete results. The flies

or orifice spreading through the teat duct into the gland,

have been shown to carry the various bacteria and to

bacteria entering the gland by draining via a supra-

be able to regurgitate the bacteria on to teat skin

mammary lymph node from another site and invasion

several days after a previous meal. Contaminated teats

by haematogenous spread. All of the pathogens are

have been demonstrated to be one precursor to disease.

ubiquitous on cattle, easily recoverable from mucous
Recently a study in Sweden confirmed a much earlier,
membranes and various lesions, and so a number of
anecdotal, UK report that contaminated *H. irritans*
routes of invasion may occur naturally.

could transmit summer mastitis experimentally.

Summer mastitis also occurs frequently outside the
fly season and in areas where the sheep headfly is not

Clinical disease

found. No other obvious vectors have been suggested
in these situations. Cases of clinical mastitis, especially
Diagnosis of the later pathogenic stages of summer
in newly calved cows, involving the same complex of
mastitis is relatively easy and requires little experience.
bacteria, have been reported throughout the year.

Early stages are more difficult to distinguish from other
There are likely to be several risk factors to these infec-
infections.

tions including appropriate exposure to the bacteria

The infection is most probably ascending so the
and teat damage.

primary sites are in the teat or in lactiferous tissue near

Initial cases of summer mastitis in an outbreak prob-

the base of the teat. Initially, there is local inflammation

ably occur by chance. Increased incidence will depend

and oedema. Expression of udder secretion may reveal

on the density of animals at risk; hence there is a peak

a foul smell indicative of anaerobic bacteria. In summer

incidence in late summer-calving herds simply because

months the teat may be attractive to flies and if the

more animals are challenged when they are most sus-

ceptible. Probably the role of the fly is to increase the

Slightly later, behavioural changes in the animal related

challenge. It therefore is involved in secondary trans-

mission. The fly may also contribute by stressing

lethargic, inappetant, stop cudding and will become

animals. Fly pestering can reduce milk yields by induc-

detached from the herd. These signs are easy to recog-

ing gross behavioural changes. Susceptibility to infec-

nize at pasture. In newly calved and housed animals the

tion during late gestation when there are other first signs are often tenderness in the quarter, which can environmental and husbandry stressors, especially poor be confused with excessive intramammary pressure, forage and heat, may increase with fly challenge. and local temperature. The secretion will be discoloured, thick and creamy, rather than colostrum, perhaps very clotted in a serous fluid, and frequently

Pathogenesis

foul smelling.

In addition to the extreme mastitis, in later stages Damage to secretory tissue is caused primarily pyrexia and lethargy develop. If the infection remains by a toxin from *A. pyogenes*, with other virulence local there may be rupture of mammary abscesses to factors including a haemolysin, a coagulase and a the exterior. More commonly, the animal becomes systemically ill with progressive lameness from the hind appears to be enhanced in mixed culture with *P.* legs, swelling of joints and an elevated temperature. The

indolicus.

bacteraemia/toxaemia may be fatal in a few per cent of

The route of invasion of the udder by a mixture of

cases; similarly there may be abortion. Perinatal death

bacteria is unknown but it seems unlikely that simulta-

is substantially increased and many calves, often born

neous invasion by up to five species of bacteria occurs.

prematurely, fail to thrive.

It is possible to isolate one or more of these pathogens

The infection develops rapidly after the initial recog-

from the udder of the lactating or dry cow in the

nizable signs so confirmation of summer mastitis by

absence of overt clinical signs so it is likely that some

bacteriology is rarely quick enough. Attempts have

predisposing factor(s) is needed to cause clinical

been made to develop cow-side diagnosis of anaerobic

disease.

involvement based on specific metabolites but no prac-

Evidence for the teat route of invasion in natural

tical technique is available. The time delay for confir-

infections comes from the frequency of summer masti-

mation is impractical. Evidence shows that 80 per cent of following teat-end damage. It has been possible to of preliminary diagnoses of summer mastitis are correct produce infections following surface contamination of (Hillerton et al., 1987). The other infections would also

Summer Mastitis • 339

respond readily to the same therapy so unless drastic control the disease is made by the 40–45 per cent of action, such as teat amputation or immediate culling, is farmers who regularly experience losses. Preventive indicated misdiagnosis in favour of summer mastitis has measures are neither simple nor foolproof and usually no economic or welfare disadvantages.

are damage-limiting rather than totally preventive. The basic aims are to limit exposure to infection from the

Treatment

herd, to prevent colonization of lesions and to prevent spread of bacteria to the mammary tissue.

Assuming that summer mastitis infections will arise

A number of different approaches to treatment can be

anyway it is important to recognize these early and seg-

taken. The one selected should depend on the anticipated an infected animal to limit direct transfer of bacteria via, for example, bedding systems and indirect the clinical severity of the infection. In a small number transfer by flies. *Hydrotaea irritans* do not pester of cases the only recommendation is for immediate housed cattle, so separate housing of infected animals culling. Less drastic is to treat the infection purely as an is good practice.

abscess; to amputate the teat and drain. This is practised

The epidemiological spread of infection is related to commonly in northern Europe with non-pregnant the density of susceptible animals and the abundance of younger animals, which are often then reared for beef.

vectors. Reducing the density of late gestation cattle by

In cows and heifers near to or at parturition the spreading the calving pattern and completely avoiding prognosis depends on the speed of diagnosis. In most the fly season of July, August and September in northern cases in dry cows the infection is sufficiently well devel-

Europe are likely to be highly effective strategies.

oped that destruction of secretory function is virtually

Considerable effort is spent on fly control but this is

complete. A clinical cure can sometimes be achieved

the least effective method of disease control as persist-

but rarely a bacteriological cure. The prognosis is much

ent presence of insecticide on teats is hard to achieve.

better if the infection is diagnosed early, and therapy

The synthetic pyrethroids now in common use are

started immediately.

applied in ear-tags or as pour-ons to the back and prob-

The pathogens are susceptible to a wide range of

ably spread by diffusion in sebum. However, the teats

antibiotics including penicillin/streptomycin prepara-

lack hair and sebaceous glands are sebum deficient, so

tions. However, there is a poor cure rate when the only

limiting spread. Effective fly control on teats is only

therapy is infusion into the udder. This is because the

achieved by direct application or frequent reapplication

tissue destruction is extensive locally and so diffusion of

of insecticide, or by the use of two ear-tags per animal.

the antibiotic is limited. A greater success has been
There has been a gradual decline in the proportion
claimed for erythromycin, which diffuses more readily.
of dry cows suffering summer mastitis, and in the
Success has also been claimed for anti-anaerobe prepa-
number of herds affected, in England and Wales since
rations containing metronidazole. Chlortetracycline can
1988. This has been coincident with the control of flies
be useful in the lactating animal but should not be used
by use of ear tags and later by pour-on formulations.
in the dry gland as all secretory function will be destroyed.
Frequently, summer mastitis follows infected lesions
Claims have been made for a variety of other prepa-
on or near the teats. These can be prevented or limited
rations including proteolytic enzymes mixed with
by use of surgical tapes, ensuring that the teats are
antibiotics but controlled studies are rare. Usually, the
cleaned first and not bandaged too tightly, or by fre-
most successful outcome follows a coordinated effort:
quent application of teat disinfectant. Many farmers
frequent stripping of the quarter combined with par-

teat-dip dry cows and heifers daily. This allows frequent enteral intramuscular antibiotics and an intramammary inspection, trains the heifers to the milking parlour and infusion. This conventional approach, although labour ensures good teat condition. Prevention of teat lesions intensive, affords the best prognosis for the udder. If may reduce greatly the attractiveness for flies.

systemic involvement is established then treatment of The best tested and most effective means of reducing clinical signs to secure the health of the animal is all that the incidence of summer mastitis is the general application of prophylactic antibiotic infused into the teat at has been lost.

drying-off. Following administration of synthetic penicillins in a long-acting base, summer mastitis rarely occurs within three weeks. Longer effects are achievable with some preparations. Where the incidence of further infection.

Control

disease warrants extra investment it is advisable to repeat the application of antibiotic after three weeks.

Summer mastitis is so destructive and expensive that

The main problem is avoiding milk contamination

considerable investment in time and materials to

after an early calving. All the evidence available

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shows a significant reduction of summer mastitis fol-

Reference

lowing use of dry-cow therapy. It can also be applied

to heifers if the antibiotic is introduced gently into the

Hillerton, J.E., Bramley, J.A. & Watson, C.A. (1987) The epi-teat duct.

demiology of summer mastitis: a survey of clinical cases.

Attempts to produce a vaccine against summer

British Veterinary Journal **142**, 520–30.

mastitis have met with little success. This remains a

long-term goal and will become more likely as multi-

valent vaccines are developed.

Chapter 25

Bulk Milk Testing and

Mastitis Monitoring

D.J. O'Rourke and R.W. Blowey

Cell counts

341

infections, there are certain other factors, some of which

What is a somatic cell count?

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are physiological, that affect the cellular content of milk.

Factors affecting the somatic cell count

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Determination of the cell content of milk

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Interpretation of somatic cell counts

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Stage of lactation

Total bacterial counts

345

The somatic cell count is high during the first week of

Sources of bacteria

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lactation, then soon decreases and remains low for

Bulk tank analysis

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Mastitis monitoring

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several weeks after which a gradual increase occurs

Introduction

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until the end of lactation. As the milk volume decreases

Recording systems

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in the latter part of lactation, an apparent increase in

Analysis of information

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cell numbers occurs from mere concentration of cells in

Target figures

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a smaller volume of milk. The presence of high cell

Uses of mastitis monitoring

350

numbers in colostrum may be due to an excessive

desquamation of epithelial cells in a small volume of

milk in a gland resuming functional activity after a

Cell counts

dormant period (Schalm et al. , 1971).

What is a somatic cell count?

Lactation age

The majority of somatic cells found in milk are white

The somatic cell count increases with the lactation age

blood cells, with the remainder mainly epithelial cells

of the cow (Fig. 25.1) (Blackburn, 1966). On the basis

shed by the secretory tissue of the udder. White blood

of histopathological examination of the udders, the

cells are found throughout the body and their main

increase in the average number of polymorphonuclear

function is to protect against disease. When large

leucocytes with advancing lactation age was attributed

numbers accumulate they are visible as pus. The mea-

to an increase in the extent of subacute inflammation

surement of the number of somatic cells in milk is

of the ducts as well as an increase in the severity of

known as a somatic cell count.

lobular lesions.

Factors affecting the somatic cell count

Stress

Mastitis

A sudden upset in the cows' routine, for example a herd

Inflammation in the udder is in no way different from

blood test or coming indoors in the autumn, can cause

inflammation in other tissues. The cardinal signs of

a raised cell count for a day or two.

inflammation, namely swelling, redness, heat, pain and

loss of function may not always be of recognizable

Oestrus

intensity in mastitis. Signs of clinical mastitis are grossly

visible but subclinical mastitis may go unnoticed. The

Bulling cows tend to have increased cell counts that are

majority of mastitis cases are bacterial in origin. One of

probably stress related.

the basic host responses to a bacterial infection is the

infiltration of white blood cells from the blood into

Milking interval

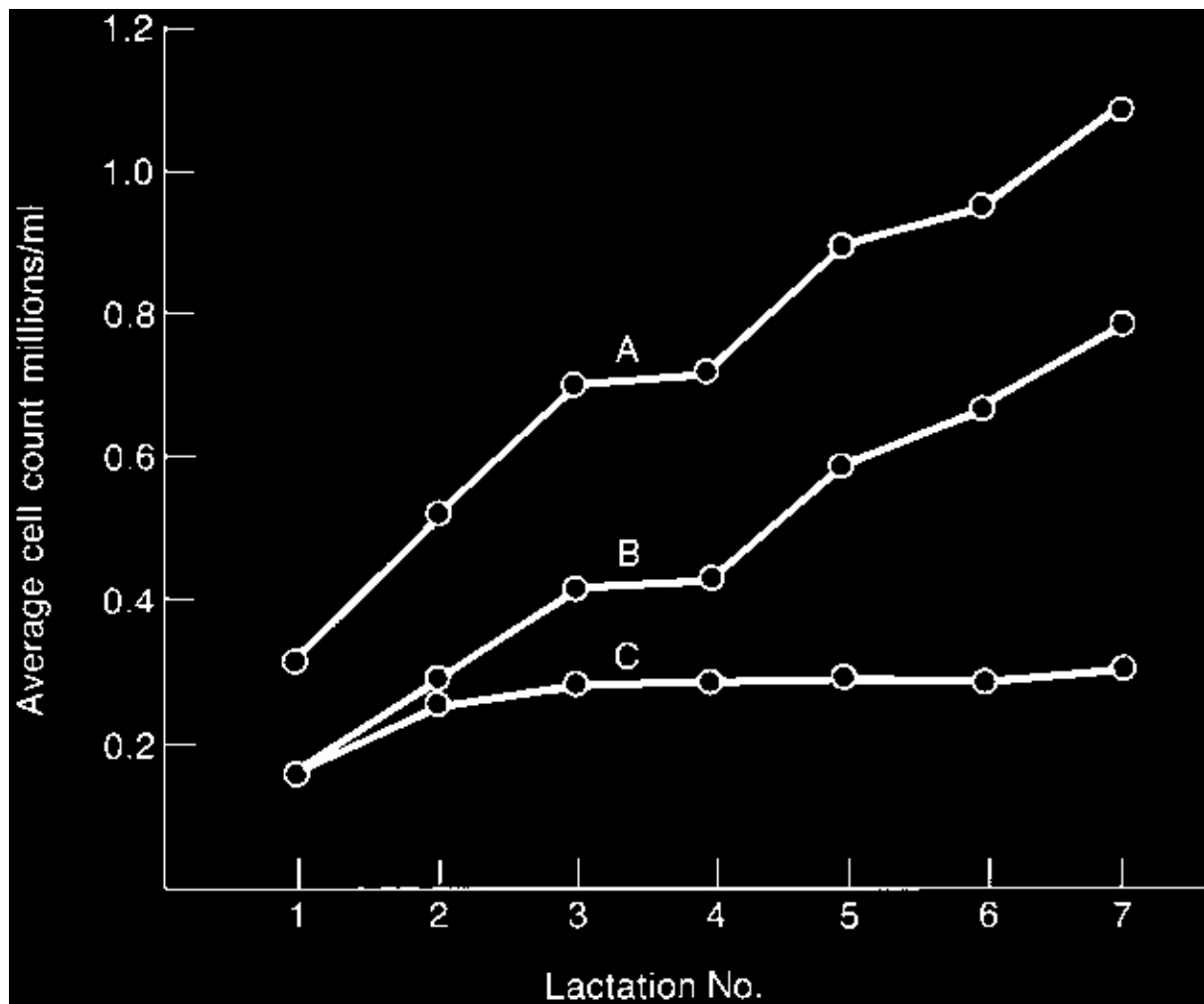
the udder. This is accomplished through the processes

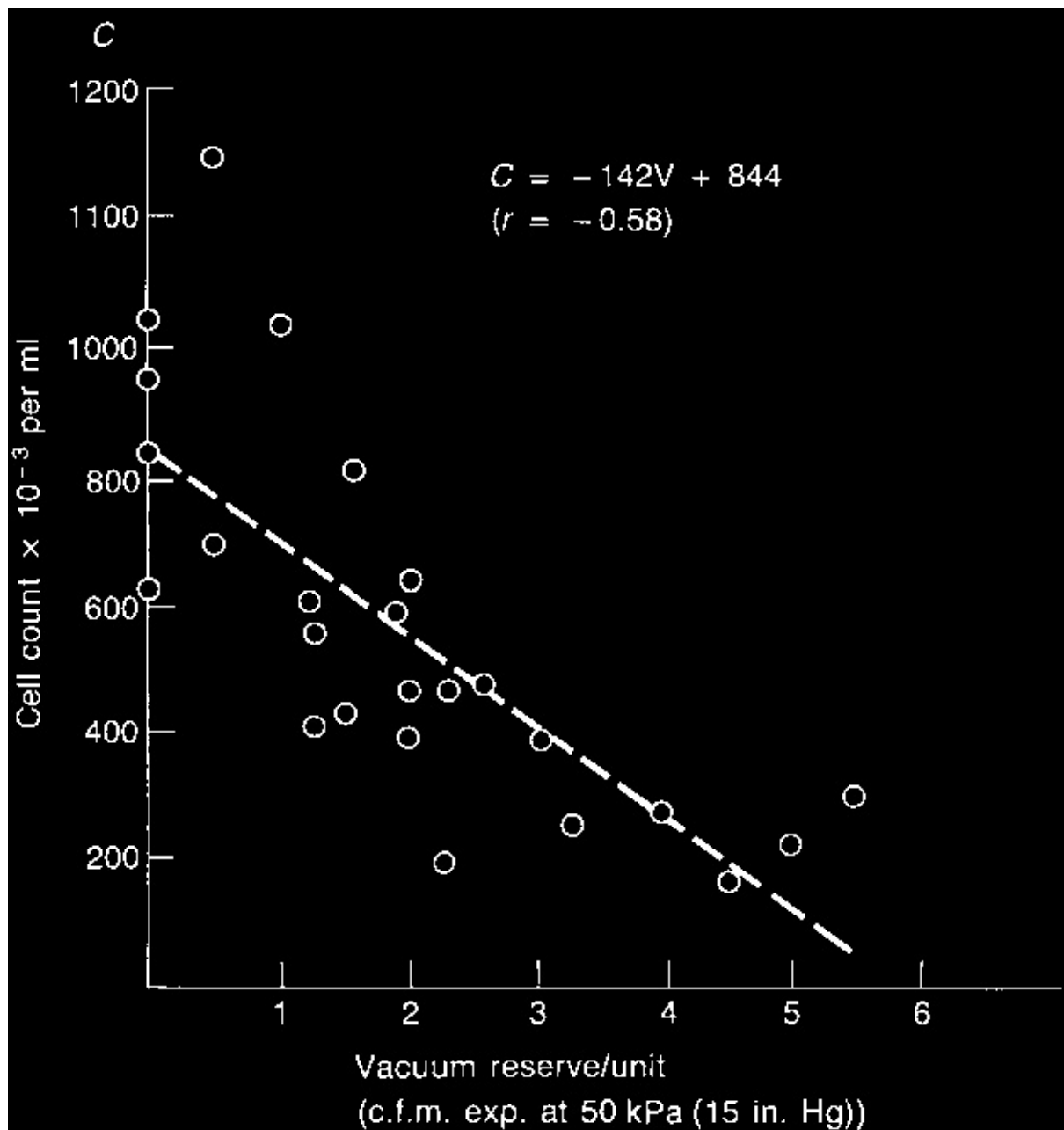
of diapedesis and chemotaxis. The degree and nature of

Irregular milking intervals will influence the somatic

the cellular response are likely to be proportional to cell counts in milk. Comparisons of somatic cell counts the severity of the infection. In addition to bacterial in milk have generally shown higher counts for shorter

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Fig. 25.1

Average cell count for each of seven lactations.

A, total cell count; B, polymorphonuclear leucocyte count; C, count of cells other than polymorphonuclear leucocytes. Source: Blackburn

(1966), reproduced with permission of Cambridge University Press.

Fig. 25.2

Relationship between mean cell count of herd bulk milk (C) and vacuum reserve per unit (V). Source: Nyhan & Cowhig (1967).

milking intervals. This variation in cell numbers is explained on the basis of total volume of milk secreted leading to a greater dilution of somatic cells during the
(1)

Distribution of cells in the smears may not be longer milking interval (Schalm et al. , 1971). homogeneous. The decision as to whether the stained structures observed are actually cells

Milking machine

may be difficult in some cases and must be made subjectively.

A significant correlation between cell counts and
(2)

To estimate the cell content every microscopic milking machine reserve air was observed by Nyhan

field examined must be multiplied by a relatively
and Cowhig (1967) in a survey of milking machine
large factor, and this can be a source of consider-
efficiency and mastitis in a random sample of 26 dairy
able error.

farms (Fig. 25.2). Six of the herds were milked with
machines that had a vacuum reserve of more than 0.11
Consequently, the primary object of microscopic count-
m³/min free air (4 ft³/min free air) and these were the
ing is the screening of other counting techniques.

only herds with a mean bulk milk cell count of <300 000

cells/ml. However, later work in Australia (Olney et al. , Electronic particle
counting

1983) concluded that in the absence of mastitis infec-
tions vacuum fluctuations, vacuum level, overmilking or
This can be carried out by the following methods.

varying pulsator rates will not cause stress or irritation
that will lead to an increase in somatic cell counts.

Coulter counter (International Dairy Federation (IDF,

1981)): With the aid of the Coulter counter it is possible to determine rapidly
and accurately the number of

Determination of the cell content of milk

particles over a certain size in a suspension. Prior to the

Reference method: microscopic counting

determination of the number of cells, the milk is treated

(Heeschen, 1975)

as follows.

The method of microscopic counting of cells in a dry

(1)

Cells are stabilized to make them resistant to

stained smear has been available for many years now

further treatments.

although it has some possible errors. The optical and

(2)

The milk to be examined is diluted with an

manual operations involved are tedious, especially in

electrolyte.

the case of serial tests, and the following sources of

(3)

The fat globules are dispersed to well below the

error must not be overlooked.

Coulter counter threshold.

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The treated milk is passed through a 100 mm aperture

Table 25.1

Distribution of cow and quarter infections shown

located between two electrodes. When a particle passes

against cell count ranges. Source: Pearson & Greer (1974).

through the aperture, a small quantity of highly con-

ductive liquid in the circuit is displaced by a particle of

Cell count ranges

No. of

Infections

($\times 10^3$ cells/ml)

herds

lower conductivity. The increased resistance raises the

In quarters

In cows

voltage, producing a voltage pulse proportional to the

(%)

(%)

volume of the particle. The number of pulses indicates

the number of particles passing through the aperture.

219–490

12

9.61

25.8

The pulses are fed into a threshold circuit so that only

535–789

12

17.76

42

those exceeding a particular threshold value (T) are

1005–1700

5

29.54

54.4

counted.

Fossamatic (IDF, 1981): This instrument is an auto-

matic microscope for counting cells in liquids. Cells are

Table 25.2

Estimated infection prevalence and losses in milk

stained with ethidium bromide and are then excited

production associated with elevated BMCC. Source: Eberhart et al. (1982), reproduced with permission of the International Asso-with a high-energy lamp, causing them to emit light at

ciation for Food Protection.

a characteristic wavelength. The emitted light energy is detected electronically, the result being displayed and

BMCC

Quarters Production

printed out for each successive sample.

(¥103 cells/ml)

infected (%)

lossa (%)

200

6

0

Accuracy of these methods

500

16

6

The precision obtained in the Fossamatic instrument

1000

32

18

is comparatively high. However, it is not essentially

1500

48

29

different from that obtained in the Coulter counter. In

a

practical operation, the Fossamatic instrument has

Production loss calculated as a percentage of production expected at 200 000 cells/ml.

proved to be superior to other methods tested. This applies to the rate of samples as well as the handling of the instrument (Heeschen, 1975).

Table 25.3

Guidelines for interpretation of bulk milk cell counts.

In an International Dairy Federation (IDF) questionnaire in 1983 on the cell counting methods 20 coun-

Bulk milk cell count

Estimate of

tries indicated they used Fossamatic, 17 used Coulter

($\times 10^3$ cells/ml)

mastitis problem

counter and two used the microscope method.

<200

Slight

200–400

Average

Interpretation of somatic cell counts

400–700

Bad

Somatic cell counts can be carried out both on herd

>700

Very bad

bulk milk and individual cow's milk.

Herd bulk milk

The prevalence of quarters infected clinically, and

Monthly cell counting has been shown to provide a fair

perhaps more importantly subclinically, with major

estimate of the subclinical mastitis status of a herd; fur-

pathogens such as Staphylococcus aureus or Streptococcus thermophilus, the annual mean can be updated on a rolling

coccus agalactiae and Strep. uberis, and to a lesser extent basis by substituting the current month's count of the

Strep. dysgalactiae, is the most important factor affect-same month of the previous year (Booth, 1985).

ing the BMCC. Fenlon et al. (1995) confirmed this in a survey of 30 dairy herds, where they found a good correlation-

ters could expect a bulk milk cell count (BMCC) of relation between the number of mastitis streptococci

<200000 thousand cells/ml. Herds with cell counts

(*Strep. agalactiae*, *Strep. dysgalactiae* and *Strep. uberis*) below this figure do exist (26 per cent of herds in

found in bulk tank milk and BMCC. Gram-negative

England & Wales; Booth, 1997) but evidence shows that

organisms such as *Escherichia coli* are usually rapidly even in these herds there is some subclinical infection

eliminated from the udder and do not tend to cause

(Tables 25.1 and 25.2). A BMCC of 200 000 cells/ml is

many subclinical infections. Unless milk from clinical

considered to be a realistic upper limit for herds with

cases reaches the bulk tank, infections of this type do

mastitis under control (Table 25.3).

not tend to influence the BMCC significantly; BMCC,

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therefore, is a poor indicator of Gram-negative infec-

Can the BMCC be too low? There is a perception that

tions (David & Jackson, 1984).

too low a cell count can lead to an increase in susceptibility to mastitis. However, BMCC is not a measure to be examined to detect trends over a period of time of resistance to mastitis. Resistance against mastitis rather than figures for a single month. Where subclinical infections with major pathogens like *Staph. aureus* of the cow to mobilize large numbers of efficient are not well controlled it is possible to see a gradual increase in neutrophils in a short period of time. Timms (1990) confirmed this when he showed that cows with a low sudden explosive increase. A sudden spectacular rise in somatic cell count (SCC) were not at greater risk the BMCC where the levels have been consistently of mastitis infections when compared to cows with a <250000 cells/ml could indicate an outbreak, with high SCC. In a survey of 125 herds with a low BMCC titic milk reaching the bulk milk supply due to faulty (<150 000 cells/ml) in the Netherlands the rate of clini-

detection.

cal mastitis was significantly related to certain variables

Fifteen countries reported a decrease in national

that increased the exposure of environmental mico-

mean cell in an IDF survey (Booth, 1995) (Table 25.4).

organisms: poor cubicle cleanliness, rubber mats in

cubicles and drinking water from sources other than

public water all increased the rate of clinical mastitis

Table 25.4

National mean cell counts in 1990 and 1993.

(Schukken et al. , 1990). Berry (1992) found that it is Source: Booth (1995).

Reproduced with permission of the Inter-possible to have a low cell count and achieve a low inci-

national Dairy Federation.

dence of mastitis in a survey of 35 herds in England and

Wales with a BMCC of less than 70 000 cells/ml.

Country

Cell count

Change

Meana

(¥ 106/ml)

(%)

Why is the BMCC important? Within the dairy indus-

1990–93

try, BMCC has become the single most important test

1990

1993

*used to measure milk quality, to regulate whether milk
can be sold and to determine the price paid for raw milk*

Austria

379

313

-66 (17)

A

(Bennett, 1993; Wells & Ott, 1995; Leslie et al. , 1996).

Belgium

307

265

-42 (14)

G

Denmark

368

309

-59 (16)

A

Reasons to include BMCC as a pricing component

Finland

282

186

-96 (34)

A

include: (i) domestic consumer demands for quality, (ii)

Germany

274

237

-37 (14)

G

processor need for high-quality raw milk for further

Hungary

419

351

-68 (16)

A

processing, (iii) to help improve udder health and (iv)

Iceland

471

408

-63 (13)

A

pressure from international markets for documented

Italy

434

426

-8 (2)

A

high-quality dairy products (Wells & Ott, 1995). EC

Japan

260

280

+20 (8)

A

Directive 92/46 states that all milk (liquid milk or milk

Netherlands

320

280

-40 (13)

A

for manufacture) must have a BMCC of less than

New Zealand

345

255

-90 (26)

W

400 000 cells/ml.

Norway

206

194

-12 (6)

A

Buyers are offering incentives for low BMCCs (Table

Sweden

230

231

+1 (0)

G

25.5), but some may question whether they, the buyers,

Switzerland

117

104

-13 (11)

A

UK

329

277

-52 (16)

G

need low cell counts in milk. Badinand (1994) indicates

that the quality of milk is reduced once the number of

a A, arithmetic; G, geometric; W, weighted.

cells is greater than 70 000 to 100 000/ml. Inflammatory

Table 25.5

*Incentives to reduce cell counts. Source: Milk Marketing Board and Milk
Marque (UK).*

Cell count (¥103)

Apr 1994

Apr 1995

Oct 1995

Apr 1996

Cell count (¥103)

Apr 1997

0–150

+0.2 p/l

<250

—

+0.2 p/l

+0.2 p/l

—

151–250

—

251–400

—

—

—

-0.2 p/l

251–400

-0.5 p/l

401–500

-0.5 p/l

-0.5 p/l

-1.0 p/l

-2.0 p/l

>400

-6.0 p/l

500–1000

-1.0 p/l

-3.0 p/l

-4.0 p/l

-6.0 p/l

>1000

-2.0 p/l

p/l, pence/litre.

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Table 25.6

Effects of mastitis on milk and milk products. Source: Munro et al. (1984), reproduced with permission of the Australian Journal of Dairy Technology.

Substrate

Alterations

Raw milk

Development of a rancid flavour; lower heat stability of whey proteins

Pasteurized milk

Reduction of flavour and quality

Recombined concentrated milk

Less stable proteins

Cheese

Reduced starter activity; changed clotting time; reduction of curd firmness; losses of fat and casein with the whey; lower yield

Butter

Impairment of flavour; less flavour; oxidative taste; longer churning time; inhibition of diacetyl production

changes in the mammary gland influence the process

Table 25.7

Interpretation of individual cow milk cell count.

of milk synthesis both quantitatively and qualitatively.

The changes in constituents of milk affect the major

Count (thousand cells/ml)

Interpretation

components (lactose, fat and proteins) as well as fatty

Less than 200

Probably uninfected

acids, protein fractions, caesins, whey proteins, anions

200 to 500

Suspicious: possibly infected

and cations, conductivity enzymes, etc. With increasing
in one quarter

numbers of somatic cells the growth of starter cultures

Over 500

Infected in at least one

in milk may be adversely influenced (Table 25.6). Ren-
quarter

netting time and heat stability of the milk can be
impaired (Heeschen & Reichmuth, 1995).

Individual cow cell counts (Table 25.7) can be of

Individual cow's milk

value to farmers and their veterinary surgeons for the

The normal somatic cell count in milk in various parts
following reasons:

of the same udder varies widely from near zero in unin-

- To estimate the proportion of cows in a herd that
fected areas to something in the order of three hundred
are infected;

million cells per litre in the worst infected areas. A cow

- To screen the herd in order to identify cows for
can have <200 000 somatic cells/ml with some small area

bacteriological examination;

of the gland badly infected.

- *To identify cows for possible culling (at least two*

Many farmers have monthly somatic cell counts

samples at monthly intervals are required);

(SCC) carried out on the milk samples taken from cows

- *As a guide to the effectiveness of dry-cow therapy;*

for milk recording purposes. These can provide a rather

- *In a herd with a high total bacterial count (TBC)*

more detailed monitor of the herd mastitis situation

where the equipment has already been checked, as

than that provided by the monthly BMCC. The cost of

the first step in identifying the cows that may be the

monthly SCC testing for one year is very low and can

cause of the high TBCs.

be recovered in mid-sized herds by preventing one or

two clinical cases of mastitis. The recovery of the cost is

Stage of lactation can have some effect; counts are

many times the out-of-pocket expense because of the

high in the first week after calving and tend to rise

many uses of SCC data and the number of cows at risk

slightly in the last few weeks of lactation. First calvers in a year's time. SCC reach the highest level in milk that have never been infected will normally have counts within the first several hours after milking and reach a of 100 000 cells/ml or less.

low point just prior to milking (Schalm et al. , 1971).

Anything less than a daily composite has reduced reliability and repeatability. Repeated samples over days

Total bacterial counts

or months add perspective to the cow's udder health history and improve the interpretation of SCC.

Although by no means a new technique, there has been

Monthly SCC data accumulated over a year for a cow

a recent increase in interest in the use of quantitative and

or herd describe more completely the mastitis history

semi-quantitative bacterial counts in bulk milk as an aid

of the herd for the previous year. Greater confidence is

in the monitoring and investigation of mastitis and milk

gained as more samples are represented.

hygiene problems. The majority of samples submitted

are for the investigation of herd problems with milk borne in mind that a considerable number of bacteria hygiene, rising cell counts and/or a high incidence of come from other sources, such as soiling on teats and clinical mastitis. With the majority of countries now the milking apparatus (Bramley et al. , 1984).

basing payment on both the cell count and bacterial count of milk, the results also have economic importance.

External contamination of cows' teats

It is vital that any sample taken is kept refrigerated during transit from the bulk milk tank to the processing Attention to udder hygiene and housing, especially laboratory, and this can produce practical difficulties. cubicle beds and passage ways, is important in reducing EC Directive 92/46 states that all milk (liquid milk or contamination from the udder skin. The handling of milk for manufacture) must have a total bacterial count cows' teats before milking must always result in the risk (TBC) of less than 100 000 bacteria/ml. Consequently, of transfer of bacteria between teats and from one cow milk supplies are subject to testing for TBC as a

to another. Herds where teats were dry wiped with measure of milk quality and bonus/penalty payments paper towels had the lowest BMCC and TBC in both are applied according to the level. The bacterial count the summer and winter period, followed by those herds obtained refers to all bacteria which includes saprophytic bacteria, faecal organisms such as *E. coli* as well paper towels (O'Rourke, 1987).

as other major udder pathogens.

Contamination from the milking equipment

Sources of bacteria

Contamination of the milking equipment is generally

Bacteria in milk comes from three main sources:

found to be the most frequent cause of high bacterial counts. Probably the two major causes are cracked and

- From within the udder

perished rubberware and inefficient cleaning of the

- External contamination of cows' teats

plant (see Blowey & Edmondson, 2000a).

- The milking plant

If there is a problem with cooling and refrigeration of milk all three sources may contribute to an increased

Bulk tank analysis

TBC.

The organisms counted and the standards currently in use by one processing laboratory (Blowey &

From within the udder

Edmondson, 2000a) are shown in Table 25.8. Other

The milk from a healthy uninfected quarter contains laboratories may use different criteria. In addition, a virtually no bacteria. Neave (1975) found counts of up to 28 million bacteria/ml in a quarter with subclinical bacteria isolated from that sample. The target values

Strep. uberis infection. However, the majority of quarters have been reduced in line with the increasingly strict

quarters with subclinical infections (Staph. aureus, Strep.

standards being imposed by the milk purchasing

dysgalactiae and Strep. uberis) had counts of around 10 000 bacteria/ml. Bacterial counts from quarters with

coliform infections were slightly lower.

TBC vs Bactoscan

Milk from cows with clinical mastitis should always be withheld. Neave (1975) found counts of up to 680 million bacteria/ml, and several quarters with counts of milk when cultured on milk agar medium at 37°C for greater than 100 million bacteria/ml (although 105 or 106/ml is more usual). Two litres of such mastitic milk

Table 25.8

Target values for different bacterial types in bulk added to 2000 litres of bulk milk may raise the TBC by milk. LPC, laboratory pasteurised count.

100 000 bacteria/ml (Cousins, 1978).

Herds with satisfactory average TBC sometimes

Organism

Target value (cfu/ml)

experience occasional wild fluctuations. This may indicate that mastitis cases are not being detected promptly

Thermoturic/LPC

<175

and mastitic milk is entering the bulk tank. It is there-

Coliforms

<20

fore essential for mastitis detection to be as thorough

Pseudomonas

<500

Total staphylococci

<200

as possible especially where milking is direct to pipeline

Staph. aureus

<10

(David & Jackson, 1984).

Strep. uberis

<2200

TBC levels can provide some information regarding

Total bacteria count

<5000

the mastitis situation in a herd. However, it must be

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48 hours. It therefore measures the number of bacteria

Staphylococcus aureus count

which grow at a specific temperature on a specific

High Staph. aureus counts are commonly found in herds culture medium. Most bacterial counts are now measured with high cell counts and indicate that additional atten-

ured automatically by Bactoscan. This counts all viable

tion to hygiene is needed during the milking process

organisms, irrespective of their favoured growth tem-

and careful postmilking teat disinfection, to prevent the

perature or cultural requirements. Bactoscan levels are

spread of infection. Spread of infection is by hands, teat

therefore higher than TBCs, by a factor of approxi-

wipes and especially the teat liners (infections). In a pro-

mately four- to five-fold. However, there is no direct

portion of herds the Staph. aureus levels in bulk milk relationship between the two, as this will depend on the

are quite high and yet cell counts are acceptable. Advice

type of bacteria present in any particular sample

given in these circumstances is that the herd is at risk

(Blowey & Edmondson, 2000b).

from a rising cell count, and that there are probably a

number of carrier cows present in the herd.

Thermotolerant count

The number of thermotolerant organisms present in the

Total staphylococcal count

sample (i.e. organisms which are not killed by heating to 64°C for 35 minutes) is taken as an indicator of poor

The total staphylococcal count was introduced because, milking plant cleaning. This may be caused by inade-

with the elimination of Staph. aureus, there has been an quite quantities of hot water (less than 18 litres per

increasing number of herds with high cell counts caused

cluster), inadequate water temperatures, a lack of

by coagulase negative staphylococci (CNS). This range

swirling and jetting during the washing routine, inade-

of organisms includes Staph. epidermidis and Staph.

quite amounts of dairy chemical used or particularly

haemolyticus.

hard water leading to inactivation of the circulation

cleaner. Many farms are now able to achieve a ther-

moduric count of 10 cfu/ml or lower, although with

Differential screen

problem herds the count may rise to above 600 cfu/ml.

The differential screen is a semi-quantitative assess-

ment of other bacteria present in the milk sample. The

Coliform count

bacteria commonly found include:

Coliform counts or, more precisely, enterobacteriaceae,

- *Streptococcus uberis*. Herds with high levels of *Strep. uberis* in bulk milk samples tend to have more contamination on teats. This could therefore indicate a clinical mastitis and may have widely fluctuating problem with poor housing, or poor premilking teat Bactoscans. *Strep. uberis* can sometimes be associated with somatic cell count problems, even when arise from cows clinically or subclinically affected by *E. staphylococcal* levels are low.

coli mastitis, with the milk entering the bulk tank. This

- *Streptococcus dysgalactiae* has been associated with could occur, for example, where foremilk is not poor teat skin condition, particularly in heifers.

tised and where mastitis detection is poor. Fluctuating

High levels of total staphylococci or Staph. aureus cell count and Bactoscan values are often associated

may also be present, as both organisms are associated with increased levels of clinical mastitis, especially E.

ated with poor teat skin condition.

coli, Strep. agalactiae and Strep. uberis.

- *Streptococcus agalactiae is sometimes identified from herds that did not even know that the cows were infected. Strep. agalactiae can be the cause of*

Pseudomonas count

increased bulk milk tank somatic cell counts and

Pseudomonads are non-enteric coliforms, sometimes high and fluctuating Bactoscans.

referred to as non-lactose-fermenting coliforms or

- *Corynebacterium bovis has been associated with NLFs. An increasing number of NLFs were found in suboptimal postmilking teat disinfection. If staphy-milk samples from clinical samples and high cell count*

lococcal levels are rising and C. bovis is identified cows (Blowey et al. , 2000), and subsequent testing

in significant numbers in bulk milk samples, then it showed that these were primarily Pseudomonas species.

would suggest that additional attention needs to be

The pseudomonad count is taken as an indicator of paid to postmilking teat disinfection.

non-enteric, non-faecal environmental contamination,

- *Other organisms include Strep. faecalis, Bacillus*

and raised levels are likely to be found in herds with species, yeasts and moulds. The presence of a wide poor premilking teat preparation and/or poor environmental range of these environmental organisms is often mental hygiene.

associated with high coliform and Pseudomonas

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counts and with an increased incidence of clinical Holstein/Friesian herds in England (average herd size environmental mastitis. Poor housing and/or poor 132 cows) over three years, recorded an incidence of premilking teat preparation could be involved.

43.4 quarter cases per 100 cows per year. The disease affected 25.9 per cent of the cows in the herd each year, with 1.6 quarter cases per affected cow, giving an overall

Mastitis monitoring

recurrence rate of 19.4 per cent. This would suggest that some cows are more susceptible to clinical mastitis

Introduction

than others. Although there has been little improvement over the past 10 to 15 years, these figures represent a marked reduction in incidence compared with the 1960s, when Kingwell et al. (1970) recorded an incidence of 120 cases per 100 cows per year. The dairy herds in all countries and many of the factors leading to a high incidence are associated with hygiene and husbandry, i.e. they are under the direct control of the farmer or stockman. It is therefore vital that there is a constant monitoring to ensure that all parties involved are made aware immediately a problem starts to develop.

that has been associated with increased yields has led
One of the largest British surveys, starting with 45 000
to a 12-fold increase in susceptibility to new infections
cows in 378 herds, was completed in 1983 (Wilesmith
(Grindal & Hillerton, 1991).

et al. , 1986). Defining a 'case' of mastitis as one quarter affected and with a
separate case commencing if seven

days had elapsed since the disappearance of clinical

Recording systems

signs, they found an incidence of 54.6 cases per 100 cows

The precise system used by the farmer to keep mastitis
in 1980, declining to 41.2 cases per 100 cows by 1982.

records is not particularly important, provided:

This is in broad agreement with the data of Blowey
(1986), reporting a much smaller survey of 22 herds

- It is used accurately and kept up to date;
monitored by a veterinary practice, where the incidence
- All information is recorded, namely date, cow
of mastitis fell from 51.0 cases in 1979 to 31.8 cases
identity, quarter affected, treatment used, with-
per 100 cows in 1985 (Table 25.9). More recently

drawal period and results of bacteriological samples

Kossaibati et al. (1998), in a study of 144 well-recorded (if taken);

Table 25.9

The average performance of the 22 herds monitored (Blowey, 1986).

Period of

Number

Herd

Rolling

Annual

Percentage

Mastitis

Intramammary

Intramammary

Percentage

records

of herds

size

mean

milk

cows

cases/

tubes used

tubes used

cases

being

herd

sales

affected

100

per cow

per case

which

recorded

milk

(l)

cows

treated

recurred

cell

count

(¥103/ml)

Oct. 1979–

Sept. 1980

22

91.7

346

6011

26.5

51.0

2.6

5.1

25.0

Sept. 1980–

Aug. 1981

24

105

302

5820

25.4

49.6

2.7

5.8

16.3

March 1981–

Feb. 1982

22

101

310

5921

25.8

459

2.4

5.6

14.9

April 1982–

March 1983

16

123.7

302

5949

27.2

493

2.5

5.0

16.1

April 1983–

March 1984

16

123.8

255

5651

23.6

42.7

1.9

4.2

13.1

April 1984–

March 1985

23

111.5

243

5479

19.6

31.7

2.1

7.1

10.3

COW No: _____			
Date	Quarter	Tubes	Sample

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- *The records are in a form that is acceptable and that a cow's lifetime mastitis history is readily available. usable for the person compiling them;*

Whatever system is used, it must be capable of produc-

- *They are easily accessible and regularly reviewed ing information on previous disease, to assist with deci-*

by both the farmer and his veterinary adviser and
sions on culling.

any necessary action is taken following this review.

An analysis of data needs to be carried out at least

Analysis of information

every six months. Intervals longer than this would fail

Regular checks must be carried out by analysing the

to achieve the aims of the monitoring (detailed later

recorded information in order to:

in this section, p. 350). It is also important to be seen to

be using the data, thereby maintaining the interest and

- Assess the incidence of mastitis on each farm and

enthusiasm of the herdsman for continuing with the

hence the efficacy of the control procedures in use;

recording. Wall charts are the simplest method of

- Compare the performance of the farm with ‘stan-

recording; computers may be used, while others use a

dard targets’ or with the herds of other members of

specific mastitis recording booklet, with a page for each

the group being monitored.

cow (Fig. 25.3; Blowey, 1983). This has the advantage

The wide variety of criteria that can be used to monitor mastitis are shown in Table 25.10, which is part of a 'league table' taken from a herd recording programme (Blowey, 1984). Bulk milk cell count is clearly the traditionally used parameter, but this is mainly a reflection of subclinical *Staph. aureus*, *Strep. uberis* or *Strep. agalactiae* contagious mastitis. It provides relatively little information on the incidence of environmental mastitis. The percentage of cows affected gives the proportion of cows in the herd that have been clinically affected by mastitis during a 12-month recording period. Any one of these cows may have had mastitis in one or more quarters and any one quarter may have had two, three or more mastitis incidents over a 12-month period. Defining a case as one quarter affected once, and a remission of clinical signs for six days or more being needed before a new case commences, then the case incidence of mastitis can be calculated. As far

Fig. 25.3

A page from a herdsman's mastitis recording book.
as the farmer is concerned, this will be a more accurate
This is kept during the life of the cow.

reflection of the ‘amount of mastitis’ occurring in the

Table 25.10

A ‘league table’ derived from mastitis data analysis.

Herd

Number

Rolling

Mean

For whole herd

For mastitic cows

number

of cows

mean

herd

Percentage

cell

yield

cows

Cases

Tubes

Tubes

Average

Recurrence

Quarters

count

(annual

affected

per 100

used per

used per

number

(%)

per

(¥103)

sales) (l)

cows

cow

case

of cases

cow

1

65

321

5800

38

60

3.5

5.7

1.6

15

1.4

2

81

745

6000

21

30

0.9

2.9

1.5

12

1.3

3

46

275

5850

19

40

4.1

9.4

2.2

45

1.2

4

134

267

5178

21

30

1.2

4.6

1.2

1.2

6

95

346

29

70

9.8

13.5

2.5

33

1.6

7

110

333

6500

27

70

2.7

3.8

2.6

40

1.6

8

132

272

6036

28

60

1.8

2.9

2.2

29

1.6

9

50

441

30

70

2.1

3.1

2.2

36

1.4

10

60

210

18

60

1.7

2.8

3.4

35

2.2

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herd. If a herd has a low percentage of cows affected,

Table 25.11

Case incidence and tube usage over a 3-year

*but a high number of cases per 100 cows (for example
recording period. Source: Wilesmith et al. (1986).*

herd 7, Table 25.10), then that herd has a problem with

either (i) a large number of down-calving cases, with

1980

1981

1982

maybe all four quarters affected at the same time or (ii)

Cases/100 cows

54.6

49.8

41.2

a proportion of 'chronics' in the herd, which have had

Tubes used/100 cows

280

273

259

repeated attacks of mastitis in one or more quarters

(33–1032)

(23–1112)

(19–1003)

over the lactation. This can be expressed as the pro-

Tubes used/clinical

portion of treated cases that recurred and is clearly very

case

6.1

6.1

5.8

high for herd 7.

(1.1–29.4)

(0.9–53.5)

(0.9–19.5)

These two situations can be separated by monitoring the percentage that recurred, namely the proportion of treated quarters that required a second or subsequent treatment during a 12-month recording period. Addi-

Table 25.12

Targets for herd mastitis incidence. Source:

tional information can be obtained by calculating

Blowey & Edmondson (2000a).

indices for the cows affected, namely for ‘mastitic cows’

(Blowey, 1984). For example, in this category (i) ‘cases

Target

Interference

per mastitic cow’ gives an indication of the average

level

frequency with which a cow gets mastitis during a 12-

month period and (ii) ‘quarters per mastitic cow’ then

Percentage cows affected

gives a further indication of whether it is one quarter

per annum

20

25

being regularly affected, or all four quarters affected

Cases/100 cows per annum

(‘mastitis rate’)

30

40

at once.

Milking cow antibiotic tubes/

In many instances, intramammary antibiotic tube

cow in herd per annum

1.4

2.5

usage is obtained from different sources, e.g. the vet-

Milking cow tubes used

erinary practice or other sales invoices. This has the

per case

4.5

6.0

advantage in that it provides a check on the accuracy

Percentage cases requiring

of the data being supplied by the farmer. For example,

a repeat treatment

if it is calculated that 10 tubes are used for each case

during a 12-month period

10

20

treated, there are several possible explanations:

Percentage dry cows affected

per annum

1.0

2.5

(1)

The cases treated are very slow to respond, either

because the wrong antibiotic is being used, or

because there is an unusually resistant organism

or because treatment is not instigated until the

case is quite well advanced.

Similarly, in the data of Blowey (1986) (Table 25.9),

(2)

Tubes are being used at a rate well in excess of the

tube usage fell from 260 to 210 tubes used per 100 cows

manufacturer's recommendations.

in a period when mastitis incidence declined from 51.0

(3)

*Tubes are being used for purposes other than the
to 31.7 cases per 100 cows per annum. Tube usage would
treatment of mastitis.*

therefore appear to give some indication of mastitis

(4)

*Not all cases of mastitis are being recorded. This
incidence, although it is not a figure that should be used
is the most likely cause of an extremely high tube
in isolation.*

usage per case.

The mean tube usage per cow in the herd and per

Target figures

*case treated as recorded by Wilesmith et al. (1986) was slightly higher than that
recorded by Blowey (1986)*

*Targets for mastitis incidence have been suggested by
(Table 25.9). However, on individual farms Wilesmith
some authors and are shown in Table 25.12. These can
et al. (1986) found that average tube usage over a 12-*

be used by an individual farmer to assess the progress
month recording period varied from 0.9 to 53.5 tubes
of a control scheme.

per case treated (Table 25.11). It was for this reason that
they concluded that tube usage was not a reliable

Uses of mastitis monitoring

indicator of mastitis incidence within a herd. However,
coincident with a decrease in mastitis incidence from

Most points have been referred to already, but an
54.6 to 41.2 cases per 100 cows per annum, there was
appraisal of the uses of recording makes a useful
a decline from 280 to 259 tubes used per 100 cows.

summary of the subject.

Bulk Milk Testing and Mastitis Monitoring • 351

- To monitor the progress of an individual cow. Any

fits of a mastitis control scheme. Veterinary Record, **119**, cow that has had four
or more cases in one quarter

551–3.

during a 12-month period should be considered for

Blowey, R.W., Davis, J.R. & Edmondson, P.W. (2000) Bulk
culling, or at least for special treatment.

milk analysis – an opportunity for greater on-farm involve-

- *To monitor herd status. Is mastitis incidence*

ment. UK Vet, 3, 26–30.

Blowey, R.W. & Edmondson, P.W. (2000a) In Mastitis Control acceptable or should more effort be put into

in Dairy Herds. Farming Press, Miller Freeman, Ipswich, control? The surveys of both Blowey (1986) and

UK, p. 142.

Wilesmith et al. (1986) showed a marked decrease

Blowey, R.W. & Edmondson, P.W. (2000b) In Mastitis Control in mastitis incidence over the recording period and

in Dairy Herds. Farming Press, Ipswich, UK, p. 133.

it is likely that the simple discipline of recording,

Booth, J.M. (1985) Bulk milk somatic cell counting: methods

leading to an increased awareness of the problem,

in use and a proposal for the standard presentation of data.

will lead to improvements.

In Proceedings of the International Dairy Federation

- *A recording system provides an opportunity for*

Seminar Progress in the Control of Mastitis, Kiel, Germany, greater veterinary involvement in on-farm advice

pp. 274–81.

and discussion. Recording in itself often provides

Booth, J.M. (1995) Progress in the control of mastitis. In
the herdsman with greater motivation in mastitis
Proceedings of the International Dairy Federation Seminar
Progress in the Control of Mastitis, Tel Aviv, Israel, **4**, 3–10.

control, and this is particularly the case if league

Booth, J.M. (1997) Progress in mastitis control – an evolving
tables are supplied, indicating how well a specific
problem. Proceedings of British Mastitis Conference, 3–8.
herd compares with the others being monitored.

Bramley, A.J., McKinnon, C.H., Staker, R.T. & Simpkin, D.L.

- If problems occur, an analysis of the records can

(1984) The effect of udder infection on the bacterial flora of sometimes indicate
the likely epidemiology of the

the bulk milk of ten dairy herds. Journal of Applied Bacte-organisms involved
and hence the control measures

riology, **57**, 317–23.

required. For example, a herd with a high percent-

Cousins, C.M. (1978) Milking techniques and the microbial

age of cases requiring repeat treatments is likely to

flora of milk. In Proceedings XXth International Dairy Con-be affected by
staphylococcal or streptococcal cow-

gress, Paris.

to-cow transmitted mastitis. Alternatively, because

David, G.P. & Jackson, G. (1984) The collection and inter-

a heavily contaminated environment tends to

pretation of herd mastitis data. *British Veterinary Journal*, **140**, 107–14.

expose all cows to the same level of infection, a high

Eberhart, R.J., Hutchinson, L.J. & Spencer, S.B. (1982) Rela-incidence of environmental mastitis would be seen

tionship of bulk tank somatic cell counts to prevalence of

in the records as a high percentage of cows affected,

intramammary infection and to indices of herd production.

almost an equal number of cases per 100 cows,

Journal of Food Protection, **45**, 1125–8.

but only low numbers of cases per mastitic cow

Fenlon, D.R., Logue, D.N., Gunn, J. & Wilson, J. (1995) A study and a low percentage of cases recurring. For herds

of mastitis bacteria and herd management practices to

with environmental mastitis, clinical case incidence

identify their relationship to high somatic cell counts in bulk will commonly be high but the cell count remains

tank milk. *British Veterinary Journal*, **151**, 17–25.

low.

Grindal, R.J. & Hillerton, J.E. (1991) Influence of milk flow rate on new intramammary infection dairy cows. *Journal of*

Dairy Research, **58**, 263–8.

Heeschen, W. (1975) *Determination of somatic cells in milk*

References

technical aspects of counting. In Proceedings of the International Dairy Federation Seminar on Mastitis Control, Badinand, F. (1994) Maitrise du taux cellulaire du lait. Recueil Reading, England, pp. 79–92.

de Médecine Vétérinaire, **170**, 6–7, 419–27.

Heeschen, W. & Reichmuth, J. (1995) *Mastitis: influence on Bennett, R. (1993) Lead, follow or get out of the way: the new qualitative and hygienic properties of milk. In Proceedings PMO SCC policy. Dairy Food & Environmental Sanitation, of the International Dairy Federation Seminar Progress in*

13, 74–9.

the Control of Mastitis, Tel Aviv, Israel, **3**, 3–13.

Berry, E.A. (1992) *Mastitis incidence in low cell count herds.*

International Dairy Federation (IDF) (1981) Recommended

Veterinary Record, **130**, 479–80.

methods for somatic cell counting in milk. Document 132,

Blackburn, P.S. (1966) The variation in the cell count of cow's pp. 5–16.

milk throughout lactation from one lactation to the next.

Kingwell, R.G., Neave, F.K., Dodd, F.H., Griffin, T.H.,

Journal of Dairy Research, **33**, 193–8.

Westgarth, D.R. & Wilson, C.D. (1970) *The effect of a*

Blowey, R.W. (1983) Data recording and analysis in dairy

mastitis control system on levels of subclinical and clinical herds. In Proceedings of the Society of Epidemiology and mastitis in two years. Veterinary Record, 87, 94–100.

Preventive Medicine, pp. 19–28.

Kossaibati, M.E., Hovi, M. & Esslemont, R.J. (1998) Incidence Blowey, R.W. (1984) Mastitis monitoring in general practice.

of clinical mastitis in dairy herds in England. Veterinary Record, 114, 259–61.

Record, 143, 649–53.

Blowey, R.W. (1986) An assessment of the economic bene-

Leslie, K.E., Godkin, M.A. & Schukken, Y.H. (1996) Milk

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quality and mastitis control in Canada: progress and

O'Rourke, D.J. (1987) To wash or to wipe – a survey of pre-

outlook. In Proceedings 35th Annual Meeting of National

milking teat preparation. In Proceedings British Cattle

Mastitis Council, pp. 19–30.

Veterinary Association, 1987–88, pp. 130–3.

Milk Marketing Board of England & Wales (MMB) (1985)

Pearson, J.K.L. & Greer, D.O. (1974) Relationship between

The use of mastitis cell counts on individual cow milk

somatic cell counts and bacterial infections of the udder.

samples. Leaflet No. ICCCS/VS/885.

Veterinary Record, **95**, 252–7.

Munro, G.L., Grieve, P.A. & Kitchen, B.J. (1984) *Effects of Schalm, D.W., Carroll, E.J. & Jain, N.C. (1971) Bovine Masti-mastitis on milk yield, milk composition, processing prop-*

tis. Lea & Febiger, Philadelphia.

erties and yield and quality of milk products. Australian

Schukken, Y.H., Grommers, F.J., Geer, D. van de, Erb, H.N. & *Journal of Dairy Technology*, **39**, 1, 7–16.

Brand, A. (1990) *Risk factors for clinical mastitis in herds*

Neave, F.K. (1975) *Diagnosis of mastitis by bacteriological*

with a low bulk milk somatic cell count. 1. Data and risk

methods alone. In Proceedings of the International Dairy

factors for all cases. Journal of Dairy Science, **73**, 3463–71.

Federation Seminar on Mastitis Control, Reading, UK, pp.

Timms, L.L. (1990) *Can somatic cells get too low? Dairy Food* 19–36.

& Environmental Sanitation, **10**, 494–7.

Nyhan, J.F. & Cowhig, M.J. (1967) *Inadequate milking*

Wells, S.J. & Ott, S.L. (1995) *Individual and bulk milk tank machine vacuum reserve and mastitis. Veterinary Record, somatic cell count results: what is the quality of the US milk* **81**, 122–4.

supply? In Proceedings 34th Annual Meeting of National

Olney, G.R., Scott, G.W. & Mitchell, R.K. (1983) *Effect of Mastitis Council, pp.* 11–22.

milking machine factors on somatic cell count of milk from

Wilesmith, J.W., Francis, P.G. & Wilson, C.D. (1986) Incidence cows free of intramammary infection. Journal of Dairy

of clinical mastitis in a cohort of British dairy herds. Veteri-Research, 50, 135–52.

nary Record, 118, 199–203.

Chapter 26

The Milking Machine

D.J. O'Rourke

The equipment

353

the claw. The milk and air travel along the long milk

Functions of main components of a milking machine

353

tube to the container (bucket). A pulsator is normally

Milking machine testing

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situated on the lid of the container and this admits air,

Static test

355

which aids in the collapse of the liners during the closed

Dynamic test

356

phase of pulsation. Air is also admitted through the

Milking machines and mastitis

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regulator, which is situated on the vacuum pipeline.

Developments in milking machine technology

357

The flow pattern is similar in the milking pipeline

Shields

357

Ball claw

358

machine except that the milk and air from each claw

Hydraulic milking

358

flow through the milk pipeline to a common receiver

Automatic milking systems

358

where air and milk are separated (Fig. 26.2). Depend-

Automatic cluster removers

359

ing on the type of milk pump used, air may be admit-

Large-bore pipelines

359

ted when milk is released from the receiver and

High- and low-line plants

359

pumped to the bulk tank.

Liner design

359

The flow pattern of the recorder type of machine is

Backflushing

360

similar to the milking pipeline machine except that air

Automatic teat disinfection

361

admitted at the claw is separated from the milk at the

Control measures

361

recorder jar (Fig. 26.3). Air has to be admitted at this

point through a special inlet or through the teatcups at

the end of each milking operation to force the milk

The equipment

from the recorder jar to the receiver jar (which is under vacuum). Some air may pass along with the milk as the jar empties, especially if the controls are not very expertly handled. This air is separated from the milk at which together provide the flow paths for air and milk. the receiver jar.

The forces necessary to move air and milk through the system arise from the fact that the system is maintained at a vacuum. Thus it is atmospheric pressure that forces

Functions of main components of

air, intramammary pressure that forces milk, into the system and the combination of these forces causes flow.

a milking machine

A milking machine has five basic components and any machine, no matter how big, can be broken down into these components (Figs 26.1–26.3):

Vacuum pump: To extract air and maintain the machine under vacuum.

- *Vacuum pump: to supply the vacuum*

Interceptor: To prevent the ingress of foreign matter

- *Regulator: to control the vacuum level*

into the vacuum pump.

- *Pulsators: to open and close the liners*

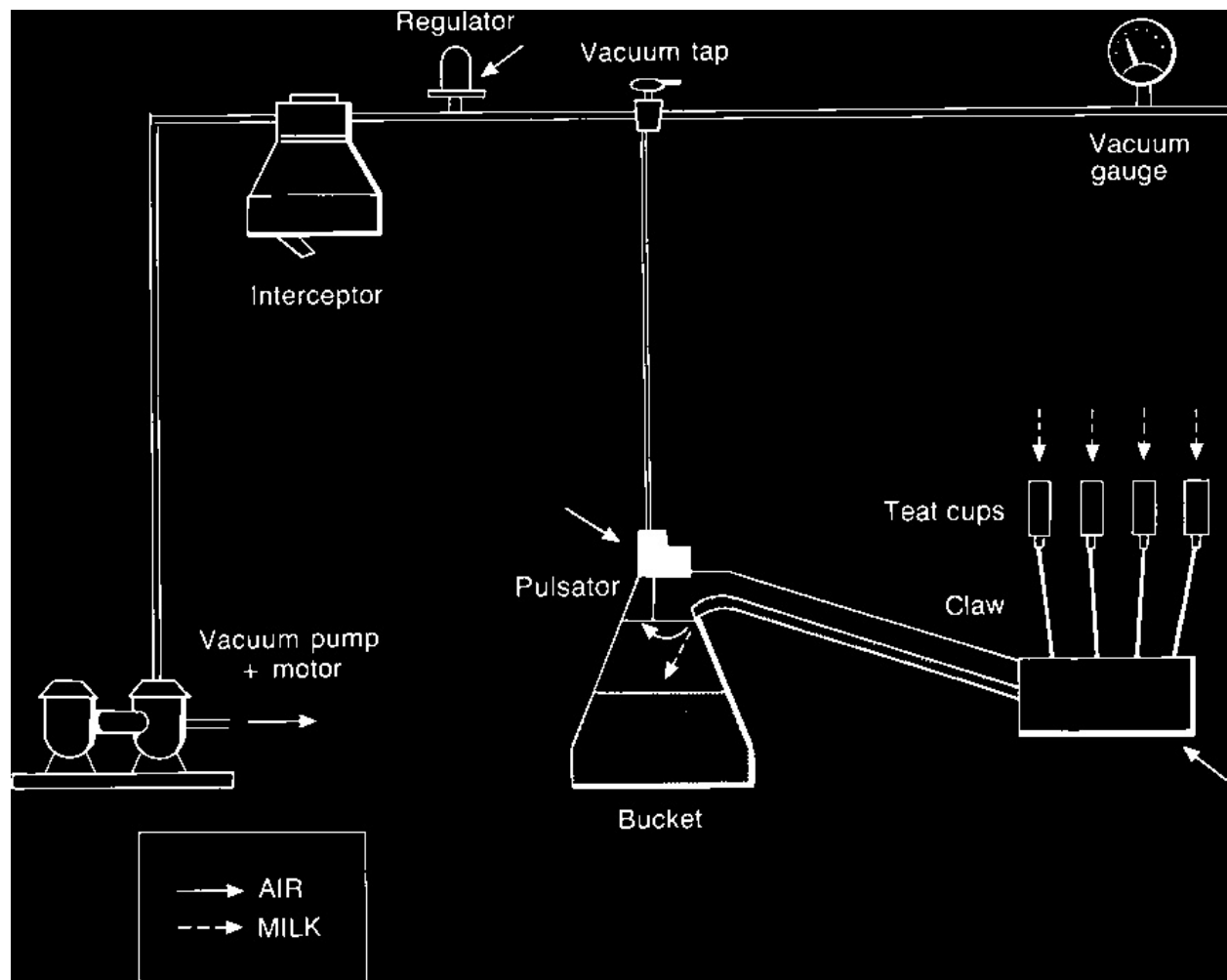
- *Clusters: to attach to the cow*

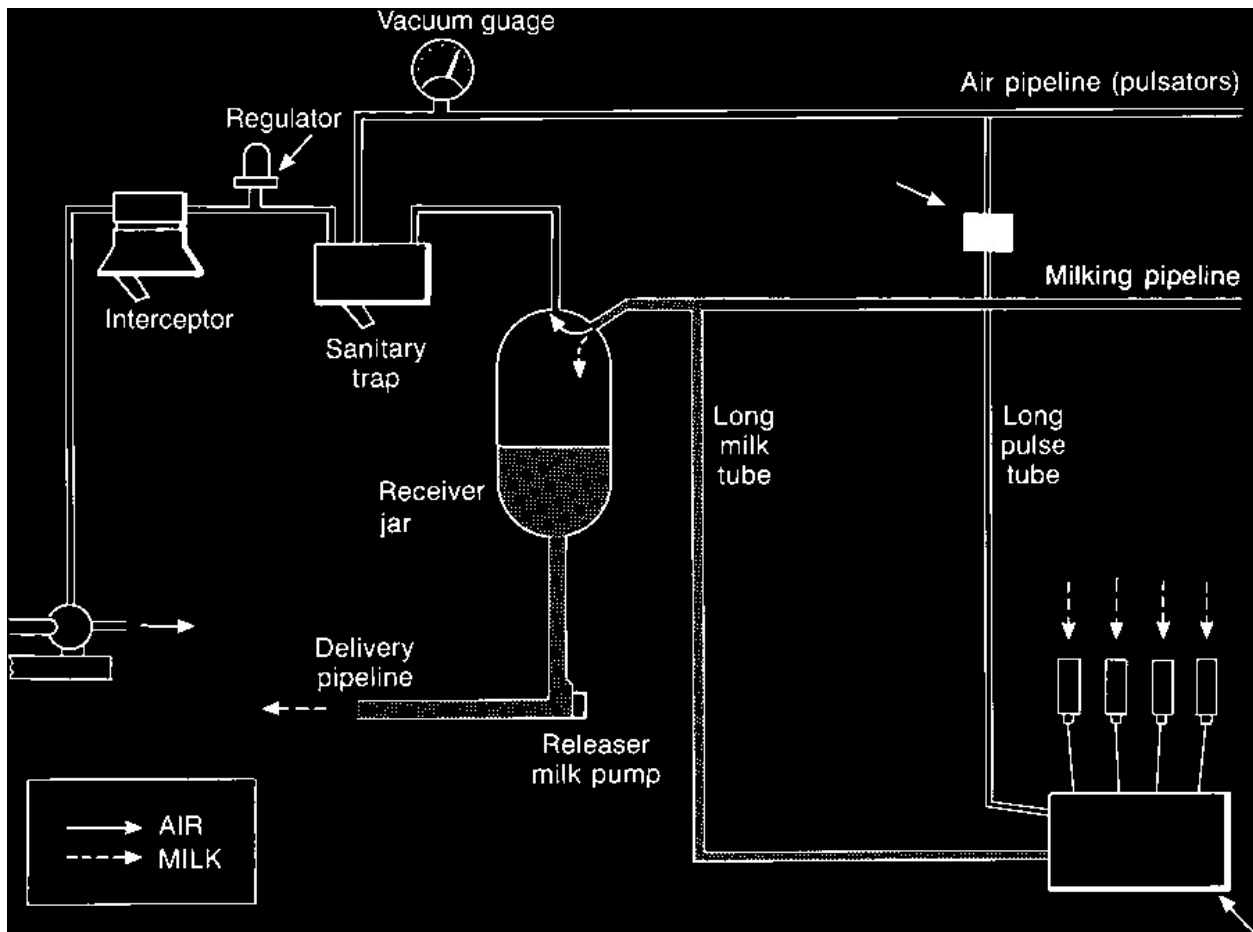
- *Containers: to store the milk*

Sanitary trap: To prevent milk vapour from entering the pulsators.

Figure 26.1 shows diagrammatically the flow of air and milk through a bucket milking machine. Milk enters the teatcups and travels through the short milk

Vacuum regulator: To maintain a constant vacuum tubes to the claw where air is admitted to break up level. It limits the maximum level by admitting air the columns of milk and improve milk flow away from into the plant when a predetermined vacuum level





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Fig. 26.1

Bucket milking machine.

Fig. 26.2

Milking pipeline machine.

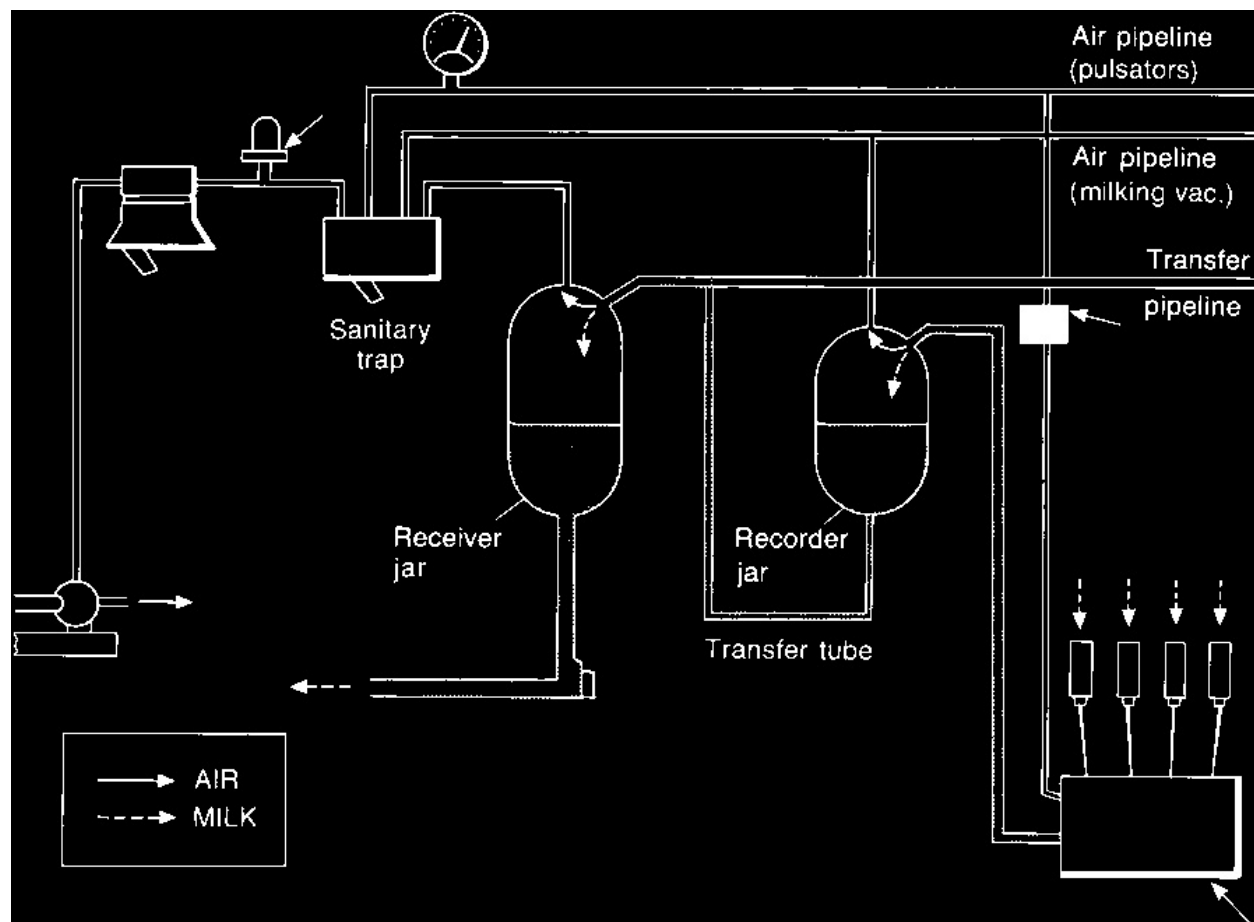
has been reached. When the vacuum level drops, the
sation chamber the liner collapses, milk flow stops and
regulator closes; this stops admission of air and the
the teat is massaged. In fact, milk generally flows when

vacuum level rises until the preset level of vacuum the liner is more than half open. The exact function of is reached, at which time the regulator opens again and pulsation has not been established but is now thought admits air.

to be relief of congestion around the teat orifice. Figure 26.4 shows a typical pulsation chamber waveform

Pulsation: This causes the liner to open and close: when containing four phases: (i) a phase, liner opening; (ii) b

there is vacuum in the pulsation chamber the liner phase, liner fully open; (iii) c phase, liner closing; and opens and milk flows; when air is admitted into the pul- (iv) d phase, liner fully closed.



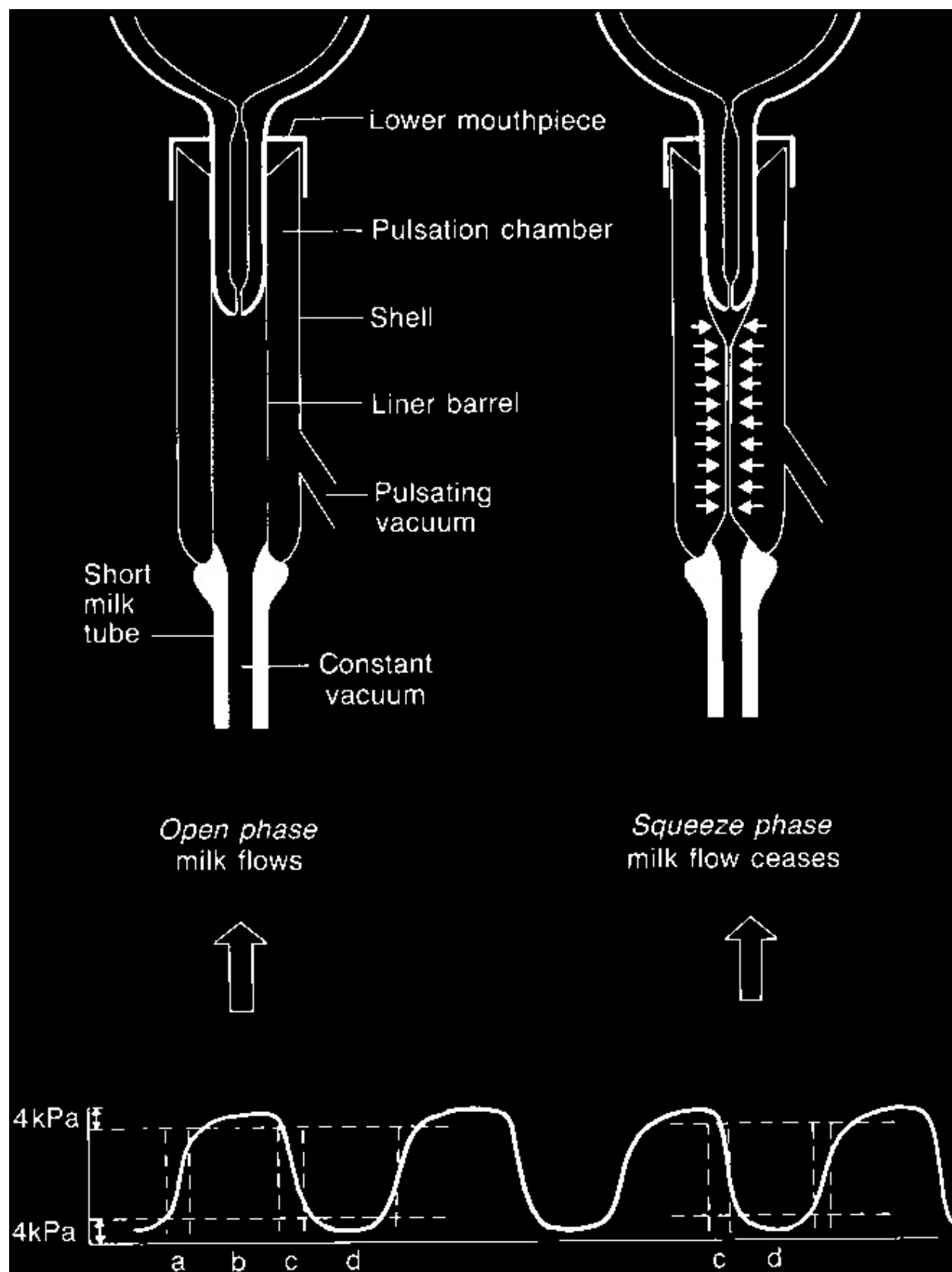


Fig. 26.3

Recorder milking machine.

Milking machine testing

Milking machines are tested to assess whether they are in good mechanical condition and conform to working standards as laid down by the standards institution of that country where used. Static and dynamic tests are used.

Static test

This type of test has been carried out for many years. It is basically an engineer's test to check for correct functioning of the components of the machine. The machine is set for milking and plastic bungs placed in the liner mouthpieces. No milk flows and, therefore, the test does not give an indication of how the machine will perform during milking.

There are three measurements to look for when interpreting a static test report:

(1)

Effective reserve. Vacuum within the plant is

created by the vacuum pump. Air is used by the pulsators, air holes in the claws and leaks in the plant, etc. The spare vacuum capacity left after compensating for these leakages is called the effective reserve. The effective reserve is used to

Fig. 26.4

Pulsation chamber waveform.

compensate for additional leakages during cluster application and removal and cluster fall-off.

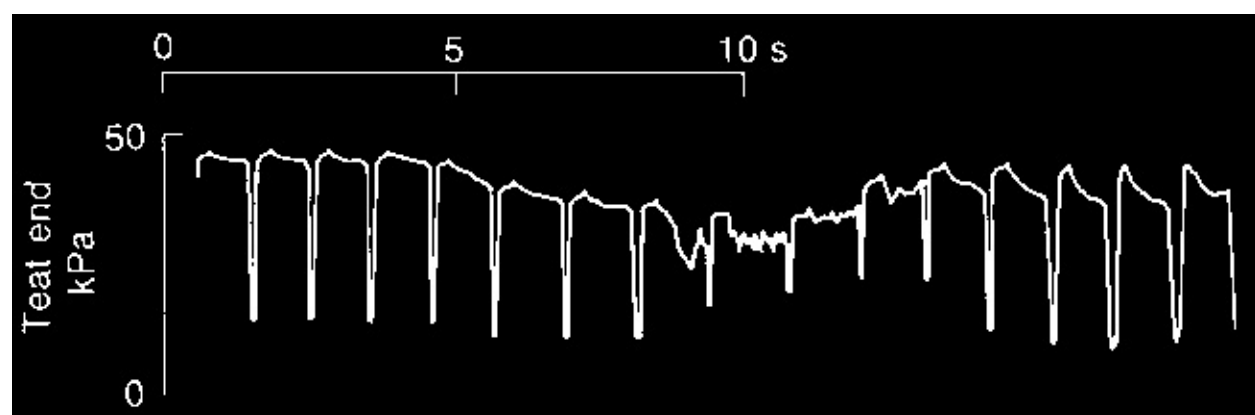
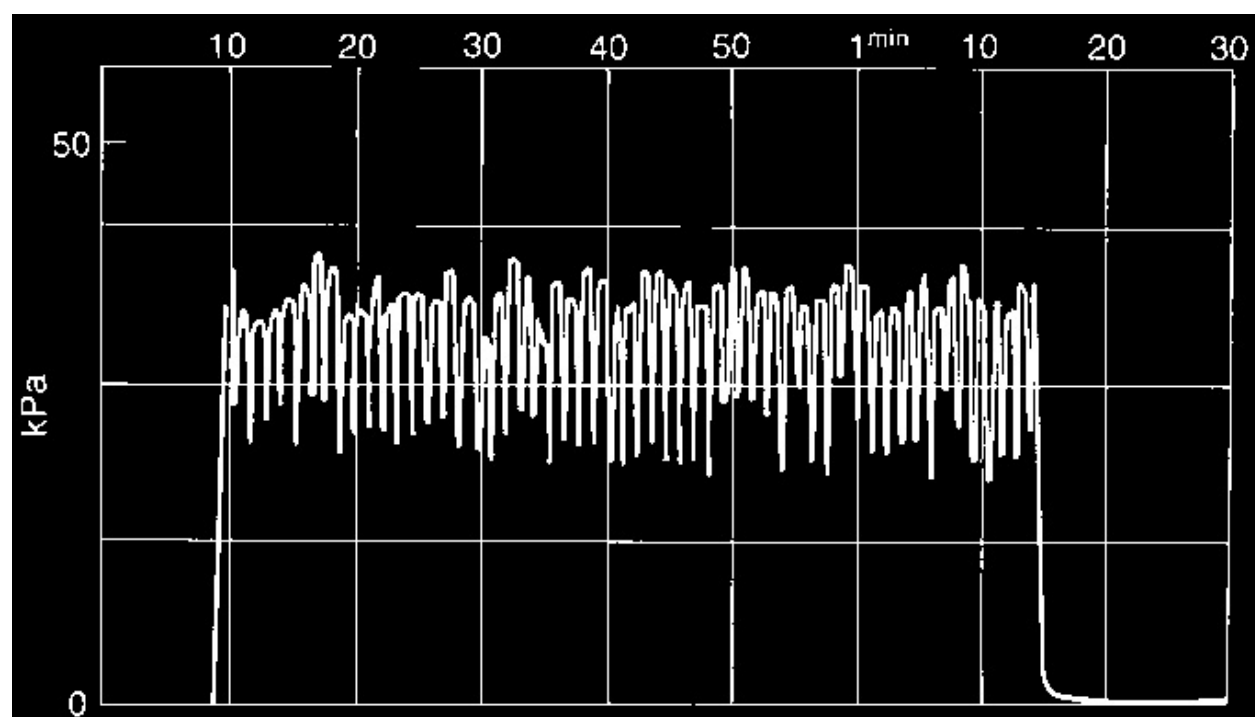
Liners: The performance of liners is related to various (2)

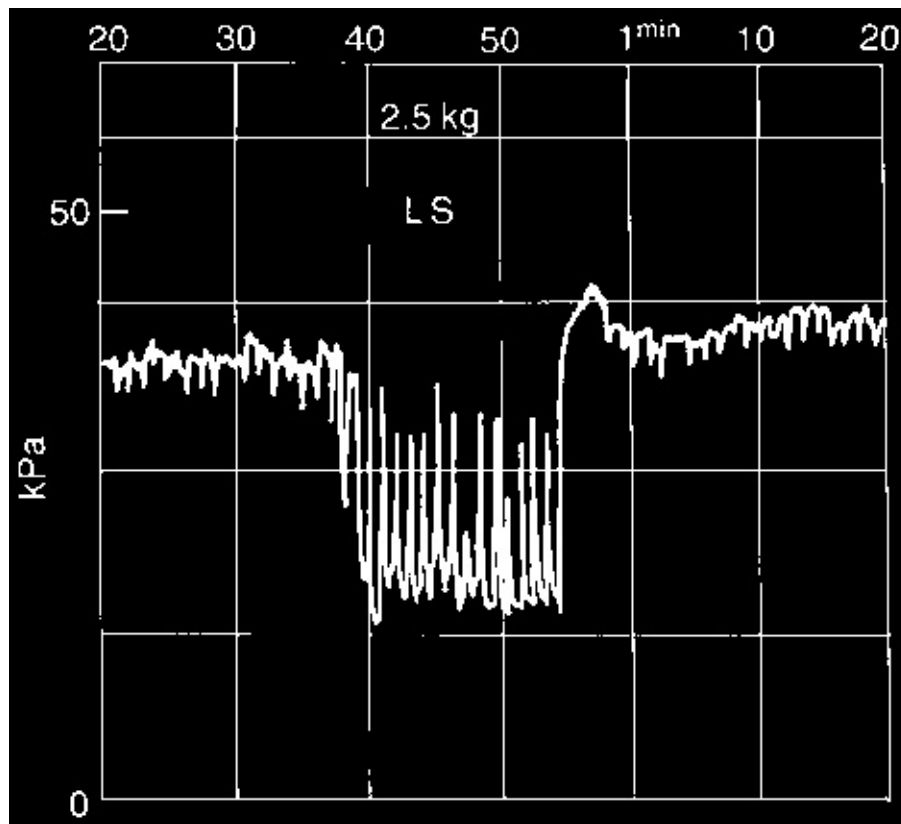
Pulsation. Table 26.1 shows the recommendations dimensions of liners and various other physical properties for pulsation characteristics.

ties of the liner. The liner is the major determinant (3)

Leakages: The static test will measure the air consumption of milking characteristics (O'Shea, 1982).

sumption of the major components in the machine





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Table 26.1

Recommendations for pulsation characteristics.

Source: O'Shea (1982), reproduced with permission.

Parameter

Acceptable range

Rate

53–63 c/min

Ratio (atb : ctd)

55–70%

d-value

Not less than 15%

and there is a set of standards for the maximum consumption allowed. It is worth checking that the results of the test are within the normal range.

Fig. 26.5

Vacuum changes (cyclic fluctuations) at the teat end during milk flow.

Practical tips

As a veterinary surgeon in practice there are two simple ways of checking the effective reserve of a machine:

(1)

Put plant in milking position with plastic bungs in the liners. For every five units, open one and let air in. If the effective reserve is sufficient the vacuum level, as read at the vacuum gauge, should not drop by more than 2 kPa (about 3/

Fig. 26.6

Vacuum changes (irregular fluctuations) at the teat

4

inch Hg)

(O'Shea, 1982).

end during milk flow. Source: Nyhan (1968).

(2)

Again put plant in milking position with plastic bungs in the liners. By letting air in the plant, via the gate valve, drop vacuum to 33 kPa (10 inches Hg). Close off air leakage. The vacuum should return to 50 kPa (15 inches Hg) within 3 seconds (Mein, 1984).

Dynamic test

Dynamic testing was indicated in only 1 per cent of herds (O'Callaghan et al., 1982) where there was a mastitis problem and the static test had revealed no faults.

At that time, equipment was either very sophisticated and expensive (UV recorder) or crude (pulsation pen recorder). In recent years, with the advent of computer

Fig. 26.7

Liner slip at the teat end during milk flow. LS, liner technology, new recording machines specially designed slip; 2.5 kg, flow rate of 2.5 kg/min.

for dynamic testing have been developed.

In dynamic testing the vacuum level is measured at

the clawpiece and other areas of the machine, e.g. the recorder jar, throughout the milking of a number of

- *Irregular fluctuations: occur as a result of changes in cows. This allows the determination of what is happen-airflow during milking, which leads to a drop in ing when milk and air are flowing in the machine and vacuum (Fig. 26.6).*

also assessment of the ability of the milker to use the

- *Liner slip: sudden air admission between the liner machine properly.*

and the teat (Fig. 26.7). A problem exists if more

There are three measurements to look for when

than five slips or falls per 100 cows require correc-

interpreting a dynamic test reading:

tion by the milker(s) (Mein, 1984).

- *Cyclic fluctuations: generated within the claw as a*

It should be noted that 50 per cent of the faults detected

result of pulsation (Fig. 26.5).

in a dynamic test are operator related .



Milking machines and mastitis

most frequent in cows with high milk flow rate (O'Shea et al., 1980).

The milking machine can act as vector in three ways: (i)

Further work carried out on simulated liner slips at

cow-to-cow transfer of bacteria via contaminated clus-

Moorepark in the early 1980s (O'Shea et al., 1981)

ters; (ii) internal flow between clusters within the plant;

yielded the following results:

and (iii) quarter-to-quarter transfer within the cow via

- There were equal numbers of new infections with the claw.*

inaudible and audible liner slips.

A farm survey in Ireland in 1967 showed a highly

- Liner slips at start of milking or the end of milking*

significant regression of bulk milk cell count on effec-

only, or during the total milking caused almost

tive reserve of the milking plant, i.e. as effective reserve

equal numbers of new infections.

decreased cell count increased (Nyhan & Cowhig,

- *Liner slips increased new infections when teats*

1967). Subsequent work showed that unstable vacuum

were heavily contaminated with environmental

(a combination of large irregular fluctuations and

bacteria (Escherichia coli

and

Streptococcus

moderate cyclic fluctuations) caused higher new infec-

dysgalactiae).

tion rates than stable vacuum.

Further work carried out at the National Institute for

Liner slip is now considered to be the major mechanism

Research in Dairying (NIRD) at Reading in the early

of spread of mastitis at milking (O'Shea et al., 1981).

1970s (Cousins, 1972; Cousins et al., 1973; Thiel et al., Baxter et al. (1990)

found that new intramammary

1973) found that large irregular fluctuations or large

infection rates (IMI/100 cow days) were 0.379 in cows

*cyclic fluctuations per se did not increase new infection milked with high-slip
liners and 0.277 in those milked*

rates. However, any combination of large irregular

with low-slip liners ($p = 0.056$). Clinical cases per 100 cow fluctuations with

substantial cyclic fluctuations caused a
days were 0.211 in the high-slip group and 0.158 in the
large increase in new infections. The results also sug-
low-slip group ($p > 0.05$). Rogers and Spencer (1991) gested that infections
were most likely to be initiated
found that variation between cows in machine liner slips,
towards the end of milking.
and manual milking machine adjustments within and
During the late 1970s research workers at Moorepark
across lactation number and days in milk can be partially
in Ireland (O'Callaghan et al., 1976; O'Shea et al., 1976, explained by udder
and teat morphology. Wider teats
1979, 1981; O'Shea & O'Callaghan, 1978, 1982) showed
(distances between teats) were associated with
that plant vacuum stability per se had little effect on increased liner slips and
increased manual milking
new infection rate. It was suggested that the effect of
machine adjustments. More tilted udders (rear quarters
generalized vacuum instability is mediated via
lower than front quarters) were associated with
increased liner slip,
i.e.

sudden air admission

increased liner slips and tended to be associated with between the liner and the teat. Liner slip, in turn, is increased manual adjustments. In addition, poor unit largely a function of liner design and also of vacuum alignments, larger teat diameter and longer teats tended stability.

to be associated with increased liner slips.

When slip occurred in one quarter, impacts of milk droplets were detected in other quarters connected by

Developments in milking

the claw to the teat in which the slip occurred and a

machine technology

higher number of new infections occurred in these impacted quarters.

Shields

This liner slip theory is compatible with many other results. Liner slips are more frequent at foreteats; in- During the late 1970s research workers at Reading fections are more often seen in hind quarters (Rabold developed shields (deflector plates) in the teatcup

& Picher, 1980). Slip frequency is most common at chamber between the short milk tube and the teat end morning milkings and high flow rates and infections are (Fig. 26.8). Trials on 15 herds in England and 16 herds

Fig. 26.8

Shielded liner. Photo courtesy of Institute of Animal Health (IAH), Compton.



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Fig. 26.9

Ball claw cluster. Photo courtesy of Institute of Animal Health (IAH), Compton.

in Australia showed that shields prevented about 10 per cent of quarters from becoming infected (Griffin et al., trials comparing

hydraulic milking with the valve claw-1980a).

piece with airbleeds. Milk flow rate was increased by 20 per cent and milking time was decreased by 26 per cent

Ball claw

with hydraulic milking. Quarter infection ratios were low in both groups with only two out of 88 quarters

In the early 1980s research workers at Reading developed a cluster to prevent the transfer of pathogens and *S. dysgalactiae* in each treatment group.

between teats during milking (Griffin et al., 1980b). It was a simple practical modification of the clawpiece for

Automatic milking systems

each of the four short milk tube connectors (Fig. 26.9).

Field trials showed an overall 14 per cent reduction in

About 100 automatic milking systems are currently in

new infection rate due to the clawpiece (Griffin et al., use on farms in Germany (Schon et al., 1997). Milking

1982). However, there was an unexpected larger reduc-

robots are used at 200 Dutch dairy farms and it was

tion (17 per cent) in new infection rates from environ-

forecast that >800 farms would be using them by 2000

mental sources of exposure (*Streptococcus uberis*,
(Ipema et al., 1998). Data (1996–98) on milk quality
coliforms and others) than from those (8 per cent)
were compared between 28 Dutch dairy farms using
transmitted mainly at milking time within the udder or
milking robots and those with machine milking three
on teat sores (*Staphylococcus aureus*, *Strep. agalactiae* times/day (28 farms)
and twice/day (49 farms). Para-and *Strep. dysgalactiae*).
meters were somatic cell count, pH of milk fat, freezing
point, clarity, fatty acids, milk fat and milk proteins.

The use of a milking robot resulted in increased somatic

Hydraulic milking

cell counts and pH of milk fat, and decreased percent-
Following research on the ball claw, Griffin and Grindal
ages of milk fat (from 4.43 to 4.37 per cent) and milk
carried out an experiment to ascertain the effects of
proteins (from 3.49 to 3.42 per cent). The main differ-
blocking the air bleed on new infections. Initial results
ences are due to the construction, design and cleaning
from a closely controlled small scale experiment in a
of milking robots and cooling tanks (Klungel et al.,

research herd showed that teat end condition was improved (1998).

improved and that it was at least equal in preventing

Working and utilization of a milking robot were

mastitis to milking with the valve clawpiece with air-

studied in a herd of 40 cows during the first 12 weeks

bleeds (Griffin, 1985). This new method of milking was

of lactation (Pomies et al., 1998). After a training period, christened 'hydraulic milking'. Research at Compton

cows had free access to the robot for 18 hours/day.

on the ball claw under hydraulic milking conditions has

During starting phase, frequency of breakdown of the

shown that as the liner starts to open, high vacuum

robot was 1.4 times/week, the rate of automatic attach-

levels (up to 90 kPa) are generated beneath the teat

ment of the cluster when a cow entered the robot was

(Grindal, 1987). Although high vacuum levels have

80 per cent (70 per cent within the first two attempts)

been shown to increase teat damage during conven-

and manual attachment was 2.6 per cent. Five cows

tional milking (Smith & Peterson, 1946), further trials

were removed from the robot due to problems on the
with hydraulic milking have not shown this to be a
front right quarter (reference for attachment of
The Milking Machine • 359

Table 26.2

Recommendations for sizes of milking pipelines.

sonable, while the 62 mm (2 1/2 inches) and 75 mm (3

Source: O'Callaghan et al. (1982).

inches) may not have been thoroughly evaluated
(Spencer, 1981).

Number of units

Minimum bore of milking

pipeline (mm)

High- and low-line plants

2–5

31

Milking systems can be divided into two types depend-

6–8

38

ing on the level of the milking pipeline in relation to the

9–20

cow's udder. Where the milking pipeline is above the udder it is called a high line and a low line if it is below the udder.

cluster). The number of daily milkings was 2.4 and the Midi-level pipelines (1.2 to 1.5 m above cow standing daily milk yield was 27.9 kg. Clinical mastitis and milk level) are recommended. Very high pipelines increase composition and quality were similar to normal values the level of cyclic fluctuations at the teat end (Thiel of the herd, except for butyric acid bacterial counts, et al., 1968). Low-level pipelines (under the cow stand-which were three times higher in the robotic-milked ing level) decrease fluctuations and increase milking cows.

rates by 3 to 8 per cent (Phillips & Copeman, 1969).

However, low-level plants are susceptible to contamination by urine and faeces. In herringbone parlours one

Automatic cluster removers

unit must be used per stall, which is only 60 per cent as Automatic cluster removers (ACRs) are an important

efficient as two stalls per unit (O'Callaghan et al. , 1982).
development in eliminating overmilking and improving
In midi-level pipeline plants vacuum fluctuations are
labour efficiency. Persistent and excessive overmilking
reduced by having air admission holes in the claw (Thiel
can cause external damage to the teat, which may
et al., 1968) but if these holes are not kept patent may increase the susceptibility
to udder infection
titis levels are likely to increase (Hopkirk et al., 1943).
(Shepherd, 1985). However, ACRs are not recommended in two stalls per unit herringbone parlours.

Liner design

Limited overmilking does not increase new infection
rate. ACRs do not reduce the work routine time, unless
Liner design can have a greater effect on milking characteristics
operators have been spending time moving up and
down the pit to remove clusters as soon as cows have
differences in strip yield, eight-fold differences in the
stopped milking. ACRs improve throughput in rotary
incidence of liner slips and 33 per cent differences in

parlours, provided teat disinfection is automated; in this milking times between liner types have been reported. case one operator less is needed. ACRs which admit air Machine-induced congestion and oedema is reduced blasts into the claw (to activate the automatic shut-off) by milking with a 'narrow-bore' liner with a soft mouth-piece. The internal diameter of a liner is usually measured at a point 75 mm below the mouthpiece. Liners removal (400 g/min), when compared to normal level should have a barrel diameter about 1–2 mm less than for cluster removal (200 g/min), decreased machine-on the average diameter of the teats after milk letdown time by 0.5 minutes, increased average milk flow rates (approximately 21–22 mm). Liners should be designed slightly, improved teat condition significantly and to fit teatcup shells. The mouthpiece should not be dis-reduced the change in teat end thickness during milking torted by the shell, but the liner should be held firmly of first lactation cows. Early removal did not affect milk

enough not to twist easily in the shell. Teatcup shells yield or composition or the incidence of subclinical should all be the same length (within 2 mm) to ensure mastitis.

uniform liner tension and to distribute the cluster weight more evenly between the four quarters (National Mastitis Council, 1996).

Large-bore pipelines

In North America narrow liners with a supple mouth These are used in the USA, where it is not uncommon are preferred, as these are thought to reduce the risk of to see milk lines of 75 mm (3 inches) in diameter. At oedema of the teats. It is considered that the diameter present, standards for sizes of pipelines are recommended by the International Dairy Federation (Table after milking, thus providing sufficient friction between 26.2). At a National Mastitis Council Meeting in the the liner and the teat. The liner should also be long USA, it was stated that the American standards for enough to enclose the teat completely. The liner must

38 mm (1 1/2 inches) and 50 mm (2 inches) pipes are re-
be well aligned in the teatcup. Liners may be made from

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natural rubber, synthetic rubber, silicone or a mixture

Results from studies carried out at NIRD (Neave,
of these products; the material used will largely deter-
1971) indicated that circulating water at 85°C through
mine the lifetime of the liner (Levesque, 1998). Most
the cluster for at least 5 seconds is the best way to
manufacturers recommend that rubber liners are used
achieve near sterilization of liners between milkings,
for between 2000 and 2500 cow milkings and then
although a cold hypochlorite flush reduced numbers of
changed. The recommended life of silicone liners is
bacteria to very low numbers (Table 26.3).

5000 cow milkings or 5 months – whichever comes first.

Laboratory experiments showed a large reduction

Theoretically they have a longer life than rubber liners
in bacteria (about 99 per cent) after backflushing with
as silicone does not absorb fat. However, in practice
cold water (Jasper & Bushnell, 1978) and an initial

they are more susceptible to tearing and puncturing flushing with an iodine disinfectant appeared to give better results. The authors concluded that even though on them, as well as being three to four times more complete sterility may not be achieved, backflushing expensive.

should be helpful in reducing cow-to-cow transfer of pathogens on liners. Around this time automatic backflushing systems using cold water rinses with or without

Backflushing

a chemical disinfectant between cold rinses were

Liners are a potent method of disseminating pathogens developed.

both within and between cows. The number of Eriksen and Rasmussen (1994) carried out a 9-week pathogens recoverable from liners after milking experiment with 283 cows in two groups, a control and infected cows is related to the number of bacteria in an experimental group. The teatcup liners of the experimental group. The teatcup liners of the experimental group. The teatcup liners of the experimental group. Consequently, it is recom-

imental group were backflushed with the Airwash
mended that clusters should be disinfected between the
system between milking each cow. The liners were
milking of infected cows. The most common practice is
flushed with a combination of compressed air and luke-
to dip the cluster in a disinfectant solution for a couple
warm water through small inlet valves placed in the
of seconds. Such a practice generally reduces the
short milk tube. The flushing sequence was water for
number of pathogens transferred from cow to cow.
4 s and compressed air for 2 s, repeated for 30–40 s.
However, such practice has often been rendered inef-
Automatic backflushing of liners reduced the total
fective because contamination of milk neutralized the
bacteria count by 50 per cent, coliforms by 68 per cent
disinfectant solution, air locks within the cluster pre-
and *Staphylococcus aureus* by 61 per cent without
vented entry of the disinfectant or the milker failed to
having any effect on udder health and somatic cell
carry out the procedure correctly.
count.

Table 26.3

Disinfection of teatcup clusters after removal from cows with mastitis. Source: Neave (1971), reproduced with permission of The British Cattle Veterinary Association.

Treatment of cluster

Time

No. tested

% positive after

No. of

disinfection

Staph. aureus

recovered per cluster

Cold water flush

5 s

19

100

100 000–800 000

Cold hypochlorite circulation

3 min

19

100

50–2000

(0.03%)

Circulation of water at 66°C

3 min

18

22

0–80

Circulation of water at 74°C

3 min

85

0

0

Circulation of water at 85°C

5 s

530

3

0–15

Table 26.4

A comparison of teat disinfection methods. Source: Anon (1983).

Dipping

Hand spraying

Auto spraying

Time taken (min/cow)

0.08–0.10

0.04–0.08

Nil

Teat-end coverage (%)

95–100

79–97

96–99

Volume used (ml/cow)

5–10

10–13

30

Chemical type

Various

Iodophor

Dilute hypochlorite

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Automatic teat disinfection

milking machine on the rate of new mastitis infections.

Journal of Dairy Research, 40, 289–92.

Several automatic teat disinfection systems are avail-

Eriksen, L. & Rasmussen, M.D. (1994) Automatic backflush-able. These are fitted to the exit race of the parlour ing of teatcup liners by the Airwash system. [Danish] Effek- and consist of a pressure spray unit fitted with a set of ten af afskylning af pattekopperne med Airwash-systemet nozzles. The spray is activated by an electronic control pa bakterieforekomst og yversundhed. Forskningsrapport unit, which detects the presence of the cow (Hunter, fra Statens Husdyrbrugsforsog. Statens Husdyrbrugsforsog, 1985). Table 26.4 shows that with automatic teat spray-Tjele, Denmark: 1994. No. 25.

ing it is possible to achieve as good coverage as dipping.

Griffin, T.K. (1985) The use of valve clawpieces in the control of mastitis. In Proceedings of the International Dairy Feder-Automatic spraying will use more chemical. However,

ation Seminar Progress in the Control of Mastitis, Kiel, as it is outside the parlour it is possible to use the much

Germany, p. 625.

cheaper chemical hypochlorite.

Griffin, T.K., Bramley, A.J., & Dodd, F.H. (1980b) Milking machine modifications in the control of bovine mastitis. In

Proceedings of the International Workshop on Machine

Control measures

Milking and Mastitis, Moorepark, pp. 19–29.

Griffin, T.K., Grindal, R.J., Staker, R.T., Shearn, M.F.H.,

The milking machine is only one component that can

Bramley, A.J., Simpkin, D.L., Higgs, T.M. & Westgarth, D.R.

be involved in mastitis. Mastitis control involves con-

(1982) Development and evaluation of control techniques

tinued implementation of each of the recommendations

for bovine mastitis. Control of intramammary infection by

in the five-point control programme. Changing some of

modification of the design of the milking machine. Report

the components in the milking machine may help in

of the National Institute for Research in Dairying, Reading,

the short term, but only a concerted effort in carrying

pp. 37–8.

Griffin, T.K., Mein, G.A., Westgarth, D.R., Neave, F.K.,

out the full programme will control mastitis in a herd

Thompson, W.H. & Maguire, P.D. (1980a) Effect of deflector in the long run.

shields fitted in the milking machine teat cup liner on bovine The following points are recommendations that

udder disease. Journal of Dairy Research, 47, 1–9.

should help to reduce mastitis resulting from the

Grindal, R.J. (1987) *Effect of ball-valve milking clusters on milking machine:*

udder disease. Veterinary Record, 121, 250–2.

Grindal, R.J. & Griffin, T.K. (1989) *Effect of hydraulic milking*

- *Maintain proper vacuum levels.*

on milking performance, teat condition and lipolysis.

- *Attach and remove clusters properly to minimize*

Journal of Dairy Research, 56, 45–53.

massive changes in vacuum and resulting from

Hopkirk, C.S.M., Palmer-Jones, T. & Whittlestone, W.G. (1943) *entry of air into the clawpiece.*

The effect of closed air admission holes on the health of the

- *Make sure clusters are attached properly on each*

udder of dairy cows. New Zealand Journal of Agriculture, cow.

66, 30–1.

- *Select liners with low slip characteristics. Replace*

Hunter, A.C. (1985) *Milking Routine. In Hygienic Milk Pro-*

rubber liners every 2500 milkings or every 6

duction. The Scottish Milk Marketing Board, p. 42.

months, whichever comes first, and siliconized at

Ipema, B. Smits, D. & Jagtenberg, C. (1998) *Practical experi-5000 milkings or every 5 months, whichever comes*

ences with milking robots. [Dutch] *Praktijkervaringen*

first.

met melkrobots. *Praktijkonderzoek Rundvee, Schapen en*

Paarden, **11**, 37–9.

- Dry teats before attaching clusters in order to

Jasper, D.E. & Bushnell, R.B. (1978) Influence of premilking reduce the number of bacteria.

sanitation on transfer of infection during milking. In

- Have the machine tested at least once a year.

Proceedings of the International Symposium on Milking

Machines and Mastitis, 17th Annual Meeting of the National Mastitis Council, Louisville, Kentucky, pp. 231–41.

References

Klungel, G., Slaghuis, B. & Hogeveen, H. (1998) Milk quality after the use of milking robots requires attention. [Dutch]

Anon. (1983) Effectiveness of teat spraying. *Dairy Farmer*, **30**, Melkkwaliteit bij automatisch melken verdient aandacht.

26.

Praktijkonderzoek Rundvee, Schapen en Paarden, **11**, Baxter, J.D., Eberhart, R.J., Spencer, S.B. & Rogers, G.W.

33–6.

(1990) Effect of milking machine liner slip on new intra-

Levesque, P. (1998) Teatcup liners. (French) *Les manchons*

mammary infection. *Journal of Dairy Science*, **73**(Suppl. 1), trayeurs. *Producteur de Lait Quebécois*, **18**, 34–6.

258.

Mein, G.A. (1984) Simple ‘on-farm’ checks of a milking

Cousins, C.L. (1972) *The relationship between the milking machine installation. Interpretation of machine test report*

machine and new intramammary infection. PhD thesis, University of Reading. Seminar for Veterinarians on Milking Machines and

University of Reading.

Mastitis, Werribee, Australia.

Cousins, C.L., Thiel, C.C., Westgarth, D.R. & Higgs, T.M.

National Mastitis Council (1996) *Liner design influences*

(1973) *Further short-term studies of the influence of the milking characteristics. In Udder Topics, August 1996.*

362 • Chapter 26

Neave, F.K. (1971) *The control of mastitis by hygiene. In*

O’Shea, J., O’Callaghan, E. & Walsh, J.P. (1984) *Machine*

The Control of Bovine Mastitis (ed. by F.H. Dodd & E.R.

Milking Research. In Moorepark 25th Anniversary Publica-

Jackson). British Cattle Veterinary Association, Reading,

tion, Part II: Animal Health and Machine Milking, pp.

p. 55.

166–8.

Nyhan, J.F. (1968) *The effect of vacuum fluctuations on*

Phillips, D.S.M. & Copeman, P.J.A. (1969) *Some recent udder disease. In Proceedings of the Symposium on research on milking machine design. Proceedings of the Machine Milking, Reading, pp. 71–82.*

New Zealand Society of Animal Production, **29**, 26–36.

Nyhan, J.F. & Cowhig, M.J. (1967) *Inadequate milking*

Pomies, D., Vimal, T., Bony, J. & Coulon, J.B. (1998) *Introduc-machine vacuum reserve and mastitis. Veterinary Record, tion of a milking robot on an experimental farm: first results* **81**, 122–5.

obtained at INRA. [French] Mise en place d'un robot de

O'Callaghan, E., O'Shea, J., Kavanagh, A.J. & Doyle, H.J.

traite dans une ferme experimentale: premiers resultats

(1982) *Machine milking and milking facilities. An Foras*

obtenus a l'INRA. Rencontres Recherches Ruminants, No.

Taluntais, p. 24.

5, 335–8.

O'Callaghan, E., O'Shea, J., Meaney, W.J. & Crowley, C. (1976) *Rabold, K. & Picher, D. (1980) Some environmental influ-Effect of milking machine vacuum fluctuations and liner slip*

ences on mastitis of cows in South Germany. In Proceedings on bovine mastitis infectivity. Irish Journal of Agricultural of the International Workshop on Milking Machines and

Research, **15**, 401–17.

Mastitis, Moorepark, pp. 121–7.

O'Shea, J. (1982) *Milking machines – the function and per-*

*Rasmussen, M.D. (1993) Influence of switch level of automatic
formance of components. Irish Veterinary Journal*, **36**, 78–87.

cluster removers on milking performance and udder health.

O'Shea, J. & O'Callaghan, E. (1978) *Milking machine effects Journal of Dairy
Research*, **60**, 287–97.

*on new infection rate. In Proceedings of the International Rogers, G.W. &
Spencer, S.B. (1991) Relationships among*

*Symposium on Milking Machines and Mastitis, 17th Annual udder and teat
morphology and milking characteristics.*

Meeting of the National Mastitis Council, Louisville,

Journal of Dairy Science, **74**, 4189–94.

Kentucky, pp. 262–8.

Schon, H., Wendl, G. & Liebler, J. (1997) *Use of automatic O'Shea, J. &
O'Callaghan, E. (1982) The effect of liner slip on milking systems in dairying.
[German] Einsatz automatis-mastitis infection rates. In Proceedings of the
Conference cher Melksysteme in der Milchviehhaltung. Landtechnik, on Dairy
Production from Pasture, New Zealand Society of* **52**, 322–3.

Animal Production, Hamilton, pp. 77–8.

Shepherd, H.M. (1985) *Milking equipment. In Hygienic*

O'Shea, J., O'Callaghan, E. & Leonard, R.O. (1980) *Milking Milk Production.
The Scottish Milk Marketing Board,*

performances of commercial clusters with standard pulsa-

p. 64.

tion. In *Experiments on Milking Machine Components*,

Smith, V.R. & Peterson, W.E. (1946) *The effect of increasing Moorepark, 1976–1979*, pp. 40–64.

the negative pressure and widening the vacuum release

O'Shea, J., O'Callaghan, E. & Meaney, W.J. (1979) *Relationship ratio on the rate of removal of milk from the udder. Journal between milking machine and the incidence of mastitis*

of Dairy Research, **29**, 45–53.

in dairy cows. Irish Journal of Agricultural Research, **18**, Spencer, S.B. (1981) *Sizing milking systems – a review. Annual* 225–35.

Meeting of the National Mastitis Council, pp. 141–6.

O'Shea, J., O'Callaghan, E. & Meaney, W.J. (1981) *Incidence* Spencer, S.B. (1991) *Effect of vacuum and milking machine*

of new infection rate in dairy cows subjected to solenoid-

liners on liner slip. Journal of Dairy Science, **74**, 429–32.

*induced air blasts during milking . Irish Journal of Agricul-*Thiel, C.C., Clough, P.A., Westgarth, D.R. & Akam, D.M.

tural Research, **20**, 163–83.

(1968) *Factors affecting vacuum within the teat-cup liner*

O'Shea, J., O'Callaghan, E., Meaney, W.J. & Crowley, C. (1976) *during milking. Journal of Dairy Research*, **35**, 303–16.

Effects of combinations of large and small irregular and

Thiel, C.C., Cousins, C.L., Westgarth, D.R. & Neave, F.K.

cyclic vacuum fluctuations in the milking machine on the

(1973) The incidence of some physical characteristics of the

*rate of new udder infection in dairy cows. Irish Journal of milking machine on
the rate of new infection. Journal of*

Agricultural Research, 15, 377–99.

Dairy Research, 40, 117–29.

Chapter 27

Skin Infections of the Bovine Teat and

Udder and Their Differential Diagnosis

J.K. Shearer, J.R. Townsend and E.P.J. Gibbs

Introduction

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is palliative. Differentiation of the diseases affecting the

Infectious lesions of the teat

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teats and skin of the udder is important, however, for

Bovine herpes mammillitis (BHM)

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reasons of public health and exotic disease control;

Pseudocowpox

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cowpox and pseudocowpox are zoonotic diseases

Papillomatosis (teat warts)

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causing skin infections that can affect milking person-

Cowpox

365

nel or herdspersons and any vesicular lesions on the

Vesicular stomatitis

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teats should always raise suspicion of foot-and-mouth

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Diagnostic techniques

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disease.

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In this chapter, particular attention is given to bovine

Prevention and control

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herpes mammillitis and pseudocowpox, the two dis-

Non-infectious lesions of the teat end

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eases that cause greatest concern to dairy farmers in the

Introduction

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industrial nations of the world.

Theleitis

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Infectious lesions of the teat

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(see Table 27.1)

Insect-induced teat lesions

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Differential diagnosis of skin lesions of the bovine teat

Bovine herpes mammillitis (BHM)

Aetiology

Introduction

Bovine herpes mammillitis (BHM) is caused by bovine herpesvirus 2 and is closely related, if not identical, to Teat lesions in milking cattle are due to a variety of Allerton virus (p. 887), the causative agent of pseudo-causes, infectious and non-infectious. Viral skin infections of the bovine teat and udder are principally due to pseudocowpox virus, bovine herpesvirus 2 and papillorhinotracheitis.

lomavirus, but, on occasions, teat lesions may occur in The genome is linear double-stranded DNA, 220 kbp. association with a generalized viral infection, such as The virus produces intranuclear inclusions and syncytia foot-and-mouth disease, vesicular stomatitis, or mucosal in infected cells.

disease. Although the vernacular use of ‘cowpox’ to

describe any teat infection is common, cowpox (as caused by cowpox virus) is an unusual disease and has

Epidemiology

been recognized only in Europe. There are several bacterial and non-infectious skin conditions of the teats

Also known as bovine ulcerative mammillitis, BHM and udder that can be equally as troublesome as viral was first recognized in Scotland. The virus has since infections. Irrespective of cause, when teat infections been isolated from affected cattle worldwide in countries such as the USA, Australia and Brazil.

secondary bacterial infection by *Staphylococcus aureus*, In the northern hemisphere, BHM commonly occurs *Streptococcus dysgalactiae* and other bacteria. Cows, as a seasonal disease between August and December. and particularly newly calved heifers, become difficult In a completely susceptible herd, the disease spreads to milk and the milking time may be prolonged by as quickly with nearly all cows developing infection over much as 50 per cent.

one to two months. In other herds, only the newly
Currently, there are no vaccines in use to protect
calved heifers introduced into the milking herd for the
cattle from any of the viral teat diseases and treatment
first time are affected. Recrudescence of clinical disease

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Table 27.1

Infectious conditions of the teat and udder skin of

Pseudocowpox

cattle.

Aetiology

Cowpox and vaccinia

Initially confused with cowpox virus, it was not until

Pseudocowpox

1963 that it was demonstrated in the USA that pseudo-

Bovine herpes mammillitis (BHM)

cowpox virus was a member of the genus Parapoxvirus.

Bovine papillomatosis (warts)

Pseudocowpox virus is closely related to the virus of orf

As part of generalized disease, e.g.

foot-and-mouth disease

(contagious ecthyma, contagious pustular dermatitis) of

rinderpest

sheep, and also to the bovine papular stomatitis virus.

lumpy skin disease

The virus contains a linear double-stranded DNA,

mycotic dermatitis

130 kpb, and produces intracytoplasmic inclusions in

infected cells.

is unusual, even though latent infections do occur.

Whether latently infected older cattle are the reservoir

Epidemiology

of infection from which the newly calved heifers

Referred to in the past as false cowpox, varicella, water-

become infected is conjectural. It is thought that sus-

pox, udderpox and natural cowpox, pseudocowpox

ceptible pregnant cows may become infected some time

occurs in most countries of the world. It is the most

before parturition, but do not develop the lesions until

common infectious cause of teat lesions in North

calving.

America and the UK. Lesions of pseudocowpox may
The method of transmission of the virus between
be seen in a herd during any season of the year. The
farms with closed herds is unknown, although biting
immunity is short-lived, lasting four to six months; thus,
flies have been incriminated as possible vectors.

pseudocowpox is commonly seen as a chronic problem
in most herds. Although herd morbidity may reach 100

Clinical signs and lesions

per cent, only 5–10 per cent of the herd may be infected
at any one time. Nevertheless, it may occur as an acute

Bovine herpes mammillitis is a disease initially char-
herd problem, which spreads rapidly affecting a major-
acterized by a painful oedematous teat swelling. The
ity of the cows in the herd. Lesions in such primary out-
incubation period ranges from three to seven days. The
breaks are frequently more extensive.

disease is generally more severe than either cowpox or
pseudocowpox and the lesions are ulcerative rather
than proliferative. Newly calved heifers are usually the

Clinical signs and lesions

most severely affected, particularly if they have post-parturient oedema of the udder. Initial lesions are

The clinical presentation of pseudocowpox is variable. followed by the development of an irregularly shaped

Usually no systemic illness is noted and vesicle formation may not be observed. After an incubation period of hours, the vesicles rupture, leaving an ulcerated surface about six days, localized erythematous and oedematous that exudes copious serum (Plate 27.2). Upon drying, areas appear on the teats. These lesions are painful, this exudate forms a thick, dark reddish-brown scab making cows difficult to milk. Within 38 hours, a small (Plates 27.3 and 27.4). In the absence of secondary orange papule develops, followed by the formation of infection, healing is complete in three weeks by granulation and regrowth of the epithelium (Plates

In some cases, a vesicle will form in the centre of the 27.5–27.8). Lesions on the udder may coalesce with papule, but this is rare with pseudocowpox in contrast to

those of the teat and may extend to the perineum
bovine herpes mammillitis. The progressive enlarge-
resulting in vulvovaginitis as a sequel to infection of the
ment of the edges of the lesions (Plate 27.10) leads to
skin of the mammary gland.

umbilication of the central scab. Healing of the lesion is
centrifugal and the primary scab is often shed after 10 to
12 days, leaving the classical raised 'horseshoe' or 'ring'

Differential diagnosis

lesion, which is pathognomonic for pseudocowpox
Bovine herpes mammillitis usually occurs in summer
(Plate 27.11). As adjacent lesions enlarge, they may coa-
and autumn. The oedema of teats, vesication and
lesce to form linear scabs extending the entire length of
ulceration and extensive scab formation over most of
the teat (Plate 27.12). Pseudocowpox lesions are pri-
the teat surface are strongly suggestive of BHM. Epi-
marily found on the teat; however, 5–10 per cent of
demics of BHM can occur in a region, which can lend
affected animals develop lesions on the udder. Healing
support to the diagnosis.

usually occurs within four to five weeks, leaving no scars.

Skin Infections of the Bovine Teat and Udder and Their Differential Diagnosis • 365

In herds where ‘teat chaps’ are common, pseudocowpox infection of the causative BPV strain of a teat wart may be virus can be detected in many of the lesions.

possible through gross examination. BPV-1 has been

In humans, pseudocowpox virus causes localized demonstrated to be responsible for fibropapillomas of infection on the fingers or hands, commonly called the penis and teats. The teat warts have a filamentous ‘milker’s nodule’. These nodules are painful and may or frond-like appearance. These warts are normally extend to the entire arm of the person affected.

seen on younger animals and will typically regress over a period of 1–12 months. BPV-5 produces small ‘rice grain’ fibropapillomas, so named because of their white

Differential diagnosis

and elongated appearance. These warts are seen in

In contrast to BHM, pseudocowpox is a chronic

cattle of all ages, and, if natural regression occurs, it does problem in most herds. Lesions are proliferative, pro-so over periods of greater than a year. The

warts

gressing to small 'ring' or horseshoe' type lesions rather produced by BPV-6 are papillomas that have a frond than ulcerative. Atypical lesions (Plates 27.13–16) may appearance and are considered the most common type be observed and confused with warts or mild traumatic of teat papilloma. These frond papillomas may regress, injuries to the teats and udder but, in general, a careful usually in a time frame of greater than a year.

examination of the herd will allow the clinician to differentiate pseudocowpox from other diseases. It must

Cowpox

be remembered that pseudocowpox is often present in herds where the major problem may be due to BHM or
Aetiology

even cowpox.

Cowpox virus shares with Jenner a central role in the development of vaccines and the control of smallpox in

Papillomatosis (teat warts) (see p. 882)

man. The current use of vaccinia virus (the derivative virus used for protection of man from smallpox) in a

Aetiology

new role as a recombinant vaccine vector for many Bovine teat papillomatosis (teat warts) is a common diseases, maintains attention on cowpox. Few people finding of dairy cattle throughout the world. The realize that cowpox was considered an unusual disease causative agents of teat warts are several distinct strains even in Jenner's time. Occasional outbreaks of cowpox of bovine papilloma virus (BPV). The strains most commonly still occur in Europe, but from the perspective of the dairy industry they are of little importance.

strains of BPV are classified as members of one of two The causative agent of the cowpox is an ortho-subgroups. Strains in subgroup-A, which include BPV-poxvirus, very similar to, but distinguishable from, 1 and BPV-5, produce warts with both dermal and vaccinia virus by its biological properties in laboratory epithelial components (fibropapillomas). Subgroup-B animals and its larger genome (220 kbp compared with strains, of which BPV-6 is a member, produce warts with

185 kbp for vaccinia virus). The virus produces intra-only epithelial proliferation (papillomas). The virus cytoplasmic inclusions in affected cells.

contains a closed, circular, double-stranded DNA approximately 8000 bp in size.

Epidemiology

Epidemiology

The epidemiology of cowpox is unknown. Cowpox virus has been isolated from skin infections in man and The method of infection is thought to be similar to carnivores in which no direct contact with cattle could other bovine cutaneous warts in that transmission is by be established. It is currently thought that rodents are direct contact with infected animals or fomites. The reservoirs of the virus.

virus needs cutaneous damage, such as abrasions, to Once infection is present within a herd, however, the gain entry into susceptible tissue. After infection occurs, disease appears to spread by the milking machine and incubation times of 1 to 6 months have been demon-the hands of milkers. Between and within herds, biting

usually on the teats of cattle in most parts of the world, infected by vesicular stomatitis virus. The vesicles will but if widespread use of vaccinia recombinants develop into erosions on the mouth (especially the becomes popular, either in man or domestic animals, tongue), teats and, rarely, the coronary band. The oral a resurgence in vaccinia mammillitis in cattle is lesions will lead to the clinical signs of excessive saliva-conceivable.

tion and feed refusal. The teat lesions produced by VS are often indistinguishable from those produced by foot-and-mouth disease. For this reason, VS is considered of great regulatory importance.

The incubation period is approximately five days, after which an irregular prodromal fever and tenderness of

Laboratory diagnosis

the teats is noticed. A roseolar erythema occurs at the site of future pock development: oedema may be local-

Sample collection

ized to the area of the erythema or may involve the

An accurate diagnosis of the cause of teat lesions whole teat. A vesicle forms three to four days after may be possible with a thorough examination of both the initial onset of signs (Plate 27.17) and rapidly affected and apparently healthy cows within a herd (or progresses to a pustule, which subsequently ruptures group in larger herds). However, for a definitive diagnosis and suppurates (Plate 27.18). The classical development of a thick red scab ranging in size from 1 to 2 cm in laboratory diagnosis requires adequate tissue samples diameter and said to be pathognomonic for cowpox to be submitted. For submitting good quality samples may now form (Plate 27.19), but more frequently an when a virus is suspected as the causative agent, the ulcerated surface is observed (Plate 27.20). Healing is clinician should remember the following guidelines. centripetal and, in uncomplicated cases, takes place within three weeks. Immunity to reinfection is said to

- During the early stages of the disease, the titre of be lifelong.*

the virus is highest at the affected sites.

- *Viruses require living cells and may be inactivated by disinfectants, light exposure, pH extremes and*

Vesicular stomatitis (see p. 710)

desiccation.

- *Secondary bacterial infection of skin samples is*

Aetiology

common in later stages of the diseases.

Vesicular stomatitis (VS) is a sporadic disease of cattle

- *Skin scrapings should come from animals with early in the US, but is endemic in other areas of the western lesions and be transported on cold packs.*

hemisphere. VS is caused by a vesiculovirus of the

- *Samples for virus isolation should be collected into family Rhabdoviridae. Vesicular stomatitis virus has a virus transport media and transported on cold genome that consists of a single-stranded, negative-packs.*

sense RNA approximately 11 kbp in length.

- *To avoid erroneous results, more than one animal and multiple lesions should be sampled.*

- An area that will be collected as a skin scraping or

Epidemiology

biopsy should be washed with saline, not alcohol

(alcohol will inactivate the viruses).

The epidemiology of vesicular stomatitis virus is not

- *Scrapings should extend to the periphery and base completely understood at this time. The disease is seen of a lesion.*

most commonly in the form of sporadic outbreaks, but

the inter-epidemic host is, as yet, unknown. Outbreaks

A wide range of laboratory tests may be used for

are seen less frequently in more temperate climates

making the diagnosis of the viral agent responsible for

and after the first frost of the year. The virus is prob-

a teat lesion. Brief descriptions of common diagnostic

ably transmitted by direct contact with contaminated

laboratory tests are included below.

milking equipment, ingestion of contaminated ma-

terials, and a number of different biting insects. The

Diagnostic techniques

antibodies produced by natural infection seem to

Electron microscopy

provide little protection from reinfection. Persistent

viral infection has been theorized, but has not been

Electron microscopy has emerged as a very accurate

clearly demonstrated.

and useful test for practitioners. The advantages of

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electron microscopy include a rapid diagnosis and the

thetic ointments have also been recommended, but in

ability to detect non-viable virus and combined viral

large herd outbreaks this type of treatment may be

infections.

difficult to justify financially. Large multiple-use con-

tainers of ointments should be avoided, as they may

become contaminated with virus and serve as a source

Tissue culture

of new infections. Strict hygiene must be followed if

applying ointments by hand, as the hands of treatment

Virus isolation using tissue culture is an alternative

personnel may transfer the infection to uninfected teats.

if electron microscopy is not available. Acceptable

A commonly recommended alternative therapy is the samples may be derived from early scabs, vesicular fluid spraying of a teat dip containing emollients (such as 10 or scrapings of the raw surface of lesions. Some strains per cent glycerin) to reduce bacterial and viral numbers

of pseudocowpox may fail to grow in vitro, but vaccinia and soothe damaged skin.

virus can be distinguished from cowpox virus on tissue

Teat warts, on the other hand, may often go ignored culture.

until an increase in size interferes with milking and requires treatment. Surgical removal of the warts, either by excision or cryosurgery, may be effective. Auto-

Histological identification

genous and commercial vaccines have also been used

Collection of a biopsy of a teat lesion is seldom easy. If as treatments, with varying degrees of success. Vaccine one is collected, the tissue should be preserved in 10 per failures in the past were possibly due to a combination cent formol saline or preferably Bouin's solution. Each

of using one strain of BPV to treat a lesion caused by of the infections discussed above has a characteristic a different strain and a lack of cross-protection between histopathology if an early lesion is sampled. Combined strains. Vaccination has been reported to be effective with immunohistochemistry, this technique is useful for for the prevention of warts caused by BPV-1 and BPV- the differentiation of the specific causative strain of 6, but not for BPV-5. Autogenous vaccines probably BPV responsible for teat warts. The disadvantage of provide the best potential for success.

this diagnostic technique is the difficulty in collecting a teat biopsy and the necessity of an early lesion of sampling.

Prevention and control

Although the treatment of individual cows suffering

Paired serum samples

from teat lesions is necessary, it is also important to

institute prevention and control strategies within the

Paired serum samples collected during the acute stage

herd. Ideally, new herd additions should be quarantined

of the infection and 14 days later may provide a diagnosis for at least 14 days before their introduction into the herd. A diagnostic 4-fold rise in serum antibodies is observed in cowpox and BHM, but pseudocowpox generally does not elicit a serological response. A primary problem with this test is that the cow may already be thoroughly disinfected after use on an infected cow. Milkers should wear gloves in the parlour and disinfect the gloves between cows. Concerted efforts should be made to reduce the fly and insect population. Teat stage serum samples must be taken within two days skin condition must be preserved by removing sources of the appearance of lesions. For practical purposes, of trauma, including improperly functioning milking equipment and poorly maintained housing. Finally, however, a clinical diagnosis of BHM may be made by the detection of high BHV-2 antibody titres in a single

the use of autogenous vaccines (such as in the case of sample.

papillomatosis) may provide some protective benefit.

Treatment

Non-infectious lesions of the

There are many differing opinions on the treatment of teat end (see Table 27.2)

ulcerative teat lesions produced by viruses. However, all the treatments have the similar primary therapeutic

Introduction

objectives of promoting healing and limiting secondary bacterial infection. Many treatment protocols rely on

In addition to infectious lesions of the bovine teat and the use of different combinations of astringents and

udder are those caused by traumatic events, chemical topical antibiotics. Topical corticosteroids and anaes-

injury, environmental conditions, insects and the

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Table 27.2

Non-infectious conditions of the teat and udder skin

ing the cross-breds did not differ in frequency of occur-

of cattle.

*rence. Adjusting production figures for length of
record, cows with one blind quarter produced only 59*

Traumatic, e.g.

kg (130 lb) (1.7 per cent) less milk for the lactation.

barbed wire

*Despite the seemingly insignificant impact on perform-
grazing kale*

*ance, records indicated that animals with blind quarters
mud*

*were culled an average of 57 days earlier than herd
'treads'*

mates.

Irritant chemicals, e.g.

incorrect strength disinfectants

any corrosive agent

Teat trauma (see p. 1127)

Photosensitization

Excessive postparturient oedema

Traumatic lesions of the teat are most commonly the

Milking machine induced

result of the cow stepping on her teats or wire cuts. They

Insect induced

are a troublesome problem for the veterinarian as well

as the dairyman. Histologically, the teat wall contains

an abundance of elastic connective tissue that provides

for expansion and contraction of the teat as it fills and

milking machine. As discussed earlier, teat lesions,

evacuates milk in the lactating cow. The near constant

regardless of cause, are frequently colonized by

movement associated with these physical dynamics of

staphylococci and Strep. dysgalactiae. Lesions occurring the teat combined with milking preparation procedures

at the teat end increase the risk of mastitis. Conse-

and milk collection complicate the normal process of

quently, high new infection rates and increased

healing.

numbers of clinical cases of mastitis are common seque-

The dairyman's challenge is in getting cows with teat

lae in herds where lesions involving the teat end are

lesions milked. Because these lesions are generally

prevalent (Plate 27.21). Lesions of the teat barrel are

painful and cows resist preparation and milking processes less likely to cause mastitis directly, but do cause greater difficulties they are difficult if not hazardous to milk.

milking difficulty. The increased risk of mastitis occurs

A further complication is mastitis. Teat lesions are more as an indirect response to the pain experienced readily colonized by bacteria and thus serve as an important reservoir of infection. Udder preparation and milking speed.

cloths, hands of the milker and milking machine components facilitate the transfer of infectious organisms between quarters of the same cow and can be

Theleitis

responsible for cow-to-cow transmission as well.

This is a non-specific condition of the inner wall of the

Emphasis on milking hygiene procedures becomes teat often associated with inflammation due to traumatic injury or infectious conditions of the teat and lesions are present.

udder. Inflammation involving the teat results in a
Depending on severity and the period of time prior
thickening and hardening of the wall of the teat cistern
to discovery, teat lacerations may be repaired surgically.
that in the most severe scenario could lead to partial or
Fresh superficial lacerations of the teat skin (within 12
complete occlusion of the teat. Theleitis is believed to
hours of occurrence) in which the vascular supply has
be a significant, if not primary, reason for the develop-
not been significantly damaged have the best progno-
ment of non-functional teats or 'blind quarters'.
sis. These are generally amenable to surgical closure. If,
However, a Florida study suggests that many blind
on the other hand, such lesions go unnoticed for a
quarters are due to non-mastitis/theleitis related
couple of days and become heavily contaminated,
factors. Blind quarters are generally described as those
cleansing in mild disinfectant solution and removal of
with an absence of mammary tissue or those which lack
the skin flap tissue is likely to be the best therapeutic
a functional teat or teat sphincter. Duraes et al. (1982) approach.

studied the incidence of blind quarters in 1177 first par-
Teat lacerations (see Chapter 66) that extend into the
turitions in a Florida herd where all five major dairy
teat cistern are of greater concern and generally carry
breeds (Holstein, Guernsey, Jersey, Brown Swiss and
a more guarded prognosis. Exposed edges of the teat
Ayrshire) plus Holstein–Guernsey cross-breds were
cistern lining must be sutured using a suture pattern
represented. A total of 38 (3.2 per cent) heifers had 48
that will turn the edges inward creating an impervious
(1 per cent) blind quarters at calving. The highest inci-
seal. If this is not achieved healing cannot occur and
dence occurred in Holsteins. The other breeds includ-
draining fistulae will often develop. The teat wall

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muscle layers and skin may be closed separately. Most
Activities associated with milking such as udder
advise intramammary and/or systemic therapy for four
preparation, the milking process and postmilking
to five days as a precaution against the development of

teat dipping all exacerbate chapping problems. Chaps mastitis. A protective bandage allowing access to the usually occur as horizontal cracks in the teat skin. teat end for milking is recommended. Some suggest Serum exudates from these cracks result in the formation of linear scabs (Plate 27.22). The surrounding teat of teat cannulas. Mastitis is a frequent secondary skin may appear dry or leather-like and flake. complication.

The primary significance of chaps is that they are Pastured cattle have a lower incidence of teat trauma readily colonized by staphylococci and *Strep. dysgalactiae* than confined cattle. Housing factors of primary importance are associated with the amount of space available affected as well as the herd. Drying of the teats and to the cow for resting and rising. Further, individual cow udders before cows leave the milking parlour, particularly during inclement weather conditions, is an impor-

for teat trauma in some cows.

tant preventive measure. Further, the use of teat-dip products containing hydroscopic skin softening agents such as glycerine or lanolin is helpful in controlling

Chemical injury

chapping problems.

Postmilking teat dipping is widely advocated for control and prevention of new mastitis infections. However, this practice has also increased the potential for chem-

Freezing or frost-bite

ical injury of teats and teat ends. The accidental or inad-

Initially, frozen teats will appear reddened or pale. If

vertent use of concentrated udder wash or cleaning

severe, the lesion progresses to the state where a scab

products in place of teat dip has been observed to cause

forms over the distal half of the teat. In time, usually

serious teat lesions with a single application.

several days, this scab will loosen and fall off, exposing

Teat lesions may also occur as a result of the appli-

a raw, denuded teat end. As a second scab begins

cation of an improperly mixed or defective teat-dip

to develop the duct may become occluded. Milking product. Iodophor-based teat dips, primarily because of becomes difficult and may require opening of the streak their widespread use, are frequently the offenders. canal surgically.

However, the problem has been observed with In less severe cases, scab formation does not occur quaternary ammonium dips, chlorhexidine-based dips, and cows become receptive to milking after only a few dodecyl benzene sulfonic acid and hypochlorite teat days. Cows immediately leaving the milking parlour dips. Lesions appear as dry, roughened, proliferative with wet teats (from dip or milk) to areas with inadequate protection during cold weather may develop by the teat dip. This discoloration may be present on frost-bite on the teat ends. Freezing of the droplet on 40–50 per cent of the teats in the affected herd. Changing the teat end confines the lesion to the teat-end orifice ing to a dip with better conditioning properties will area. The result is as described above for frozen teats.

result in the rapid improvement of teat skin health.

Treatment consists of attempts at keeping the teat

In the case of the iodophor dips, problems have

duct patent and preventing the development of masti-

arisen secondary to freezing of the dip on farm or in

tis. Severely affected cows may need to be culled.

transit. When these solutions freeze they separate,

Drying of the teats and udder prior to exit from the

sending emollients to the bottom of the container while

milking parlour and provision of adequate wind breaks

leaving excessive amounts of the active ingredients in a

and shelter for milking cows is essential. The suspen-

layer suspended above. The subsequent application of

sion of teat dipping procedures should be considered

this concentrated iodine can cause teat irritation and

during extremely cold winters.

lesions. Depending on the degree of insult these lesions

can be quite severe.

Sunburn

Environmental injury

Severe reddening and drying of the teats and udder are

observed in sunburn conditions, when blisters may form.

Teat chaps

*Lesions are most severe on unpigmented skin or where
Teat chapping is a common problem in the more tem-
the skin is devoid of hair. The application of moisturiz-
perate regions of the world where climatic conditions
ers, ointments and salves to the affected areas is advised
favouring dampness and cooler temperatures prevail.
for treatment. For burns caused by fire see p. 934.*

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Photosensitization (see p. 884)

*hyperkeratosis observed. On the other hand, a Wiscon-
sin study indicated that hyperkeratosis of teat ends
This condition occurs when photodynamic agents are
appeared to increase in relation to the compressive load
eaten in their preformed state. Upon exposure to sun-
of the milking machine liner.*

*light, the unpigmented skin areas develop an erythema
and oedema, which results in severe lesions that most
commonly appear on the lateral aspects of the teats and*

Black spot

udder (Plate 27.23). Medial aspects of affected teats remain soft and cool. These lesions are highly susceptible to secondary bacterial infection.

This condition is observed as a lesion of the tip of the teat commonly associated with excessive vacuum levels and overmilking. It is a particularly troublesome problem as the lesions are quite painful and frequently

Milking machine-induced teat lesions

harbour mastitis pathogens, notably *Staphylococcus* sp. *Fusobacterium necrophorum* is also usually present in The milking machine is assumed to be a major cause of the lesions, but difficult to isolate. The specific location of this lesion at the tip of the teat end significantly increases the risk of mastitis in affected animals.

extremely high vacuum or pulsation failure. Damage Control of the condition requires correction of the pre-milking machine may be by direct trauma to the teat or indirect, occurring during milking. The cause and application of an effective teat dip solution over an extended period of time through the induc-

containing emollients or other skin conditioning agents
tion of degenerative changes in the teat. These changes
such as glycerol.

in teat tissue health are primarily associated with cir-
culatory disturbances, resulting in oedema and hyper-
keratosis.

Proper pulsation is essential for the

Insect-induced teat lesions

circulation of blood and lymph in the teat. When
normal circulation is disrupted teat-end health

Summer mastitis is an acute suppurative disease of the
diminishes.

non-lactating mammary gland (see Chapter 24). First

Direct damage to the teat may be caused by exces-

described in Europe, it has been reported in the USA

sive milking vacuum, inadequate pulsation and careless

and other countries as well. It occurs sporadically

use by the operator. Although rare, subcutaneous

throughout the year in Europe, with annual incidences

haemorrhages in the teat epithelium and prolapses of

in England and Wales estimated to be around 2 per cent

teat duct tissue are possible consequences with severe of heifers and dry cows. In the USA, some estimate malfunctions of milking equipment or its use.

incidences of 5–6 per cent on certain farms during the summer months.

While some questions remain, in both the USA and

Calluses and hyperkeratosis of teat ends

Europe epidemics of summer mastitis have been coincident with periods of greatest fly challenge. Further,

Because of its appearance, hyperkeratosis of the teat data indicate that effective fly control reduces disease

end is often mistakenly diagnosed as a prolapsed

incidence. These findings support the possibility of

sphincter. Hyperkeratosis of teat ends is a common

insect involvement. European data suggest that biting

finding in hand milked dairy cows as well as beef cattle.

flies are responsible for the initial damage to the teat

Histopathology of this material will confirm it to be

*end and implicate the cattle fly, *Hydrotaea irritans*, as excess keratin, which some refer to as callouses. These*

the infectious vector in summer mastitis (see Chapter

callouses may be rough or smooth and thus logically
24).

implicated as possible risk factors for mastitis.

However, according to studies in the USA, the presence
of hyperkeratotic lesions at the teat end had no effect
on the prevalence of mastitis unless accompanied by

Differential diagnosis of skin lesions

erosions or scabs.

of the bovine teat

The exact cause of hyperkeratotic lesions in dairy
cattle is unknown, but appears to be related to certain
The following is an aid to the diagnosis of lesions of the
milking practices and milking-machine-related factors.
udder and teats in severely affected milking herds (see
For example, high levels of milk production and
Table 27.3; Plates 27.1–32). With the exception of teat
extended milking times, failure to apply appropriate
chapping and milking machine damage, the aetiology of
premilking stimulation and overmilking increased the
which appears to be complex, most outbreaks of teat
degree of lesions. Vacuum levels and pulsation ratios

lesions in cows are due to three types of virus: (i) (within normal limits) had no effect on the degree of cowpox, (ii) pseudocowpox and (iii) BHM.

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Table 27.3

The clinical appearance and diagnosis procedures for teat conditions (other than BHM, cowpox, bovine vaccinia mammillitis and pseudocowpox).

Condition

Clinical appearance

Confirmatory diagnostic

procedure

Blackspot (Plate 27.27)

Scabby infection of teat orifice with

*Isolation of *F. necrophorum**

Fusobacterium necrophorum but often

(often unsuccessful)

other organisms

Chaps (teat) (see Plate 27.22)

Skin fissures often through dermis.

None. Parapox virus particles

Haemorrhage may occur with scab

may be found but their

formation at the fissure edge. Often

importance unknown. Heavy

horizontal lesions

bacterial contamination of

lesion common

Folliculitis and impetigo

Pustule with surrounding erythema

Isolation of Staphylococcus aureus

Foot-and-mouth disease (see Plate

Vesiculation of teat, buccal and

Presence of virus. Notifiable

27.24)

interdigital mucosae. Pyrexia

in most countries

Mud abrasion (Plate 27.28)

Abrasions on lateral surface of the udder

Photosensitization (see Plate

Lesion progresses from erythema, oedema

27.23)

to serous exudation through skin. There is

then necrosis, ulceration and scab formation.

Pigmented areas of teat unaffected

Ringworm (Plate 27.29)

Typical grey crusty lesions

Isolation of Trichophyton verrucosum

Theleitis and serous exudate from udder

Teat swollen, painful with udder skin

Isolation of bacteria

skin in peracute mastitis (Plate 27.30)

involved. Cow febrile and toxic

Vesicular stomatitis

Vesiculation of teat, buccal and

Virus isolation. As similar to foot-and-

interdigital mucosae

mouth may need to notify

Warts

Filiform (Plate 27.31)

Pedunculated attachment to teat

White nodule (Plate 27.32)

Broad attachment to teat. Variable in size.

Some warts intermediate between filiform

and nodule

Accurate diagnosis is important for various reasons. A

Reference

major factor is the zoonotic potential of cowpox and pseudocowpox. Secondly, there is the need to differen-

Duraes, M.C., Wilcox, C.J., Head, H.H. & Van Horn, H.H.

tiate foot-and-mouth disease as occasionally vesicles

(1982) Frequency and effects on production of blind quar-

ter occur on the teats before their appearance in the mouth

ters in first lactation dairy cows. Journal of Dairy Science, and foot (Plate 27.24).

65, 1804–807.

When investigating an outbreak it is best to examine

as many cattle as possible because the lesions in a single

cow may not be typical. Often mixed disease occurs

Further reading

(Plates 27.25, 27.26) and an assessment of the development of lesions rather than just examination of the most

Farnsworth, R.L. (1996) Observations on teat lesions. In severely affected cattle helps in the diagnosis.

Proceedings of the National Mastitis Council, pp. 93–8.

Farnsworth, R.L. & Seiber, R.L. (1979) Relationship of teat On occasion the clinical appearance of the lesions

end lesions to intramammary infections. In Proceedings of

can be modified by environmental factors, thereby

the National Mastitis Council, pp. 17–24.

making recognition of the condition difficult. Such

Francis, P.G. (1984) Teat skin lesions and mastitis. British factors include the teat cluster, mud or coloured teat

Veterinary Journal, 140, 430–6.

dips. In these problems laboratory diagnosis be-

Gibbs, E.P.J. (1984) Viral diseases of the skin of the bovine teat comes essential, although it is advisable in all disease

and udder. Veterinary Clinics of North America. Large

investigations.

Animal Practice, 6, 187–202.

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Gibbs, E.P.J., Johnson, R.H. & Osborne, A.D. (1970) The dif-Sieber, R.L. & Farnsworth, R.J. (1978) The etiology of bovine ferential diagnosis of viral skin infections of the bovine teat.

teat lesions. In Proceedings of the National Mastitis Council, Veterinary Record, 87, 602–609.

pp. 5–15.

Gibbs, E.P.J. & Rweyemamu, M.M. (1977) Bovine her-

Sieber, R.L. & Farnsworth, R.J. (1984) Differential diagnosis pesvirus, 1, 2 & 3.

Veterinary Bulletin, **47**, 317–43, 411–

of bovine teat lesions. Veterinary Clinics of North America.

25.

Large Animal Practice, **6**, 313–21.

Chapter 28

Factors Affecting Milk Quality

R.W. Blowey and R.A. Laven

Introduction

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(g/kg milk) may be reduced, but the overall fat pro-

Feeding and milk composition

373

duction (g/day) may be increased.

Feeding before calving

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Feed constituents

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Dietary fat

375

Feeding before calving

Dietary protein

376

Severe underfeeding, leading to cows calving down in

Feeding systems

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condition score 2 (overall scale 1–5, see p. 10) or less,

Non-nutritional factors

377

Age of cow

377

will reduce milk yield, fat, protein and lactose and

Stage of lactation

377

although these effects are greatest in early lactation,

Season of the year

377

they will persist throughout it. The extent of the depres-

Disease

377

sion is approximately 20 g/kg (0.2 per cent) in milk fat

Genetic variation

378

and 10 g/kg in protein. Less severe underfeeding has

Management factors

378

little effect on milk protein, and provided that high quality diets are provided after calving, some of the effects on milk quality can be eliminated (Garnsworthy

Introduction

& Topps, 1982).

Feeding a high energy density diet (11.7 MJ/kg DM) precalving significantly increases milk fat concentration

Most countries with a developed dairy industry now in early lactation; however, this effect does not persist pay producers on the basis of both total volume sold and there is no significant effect on fat yield and no and compositional quality, and with the slowly increasing effect on protein concentration or yield.

ing move for a larger proportion of milk to be used for

Very fat cows have an increased fat content in their manufacturing, this trend is likely to continue. In addition, milk, and it has been estimated that milk fat increases tion, many countries also have a milk quota system

by 2 g/kg for each 1.0 unit rise in condition score. This and to maximize profitability, a farmer must produce a effect lasts for the first five to six weeks of lactation only, specified amount of milk of optimum quality. Much and is related to fat concentration only; indeed total fat attention has therefore been paid to factors affecting yield is often depressed in very fat cows.

milk quality, the most important aspects of which are reviewed in this chapter. The composition of average Friesian/Holstein milk is given in Fig. 28.1. Feeding has

Feed constituents (see p. 112)

by far the greatest impact on milk quality and as such The greatest effect arises from the forage : concentrate will be discussed first.

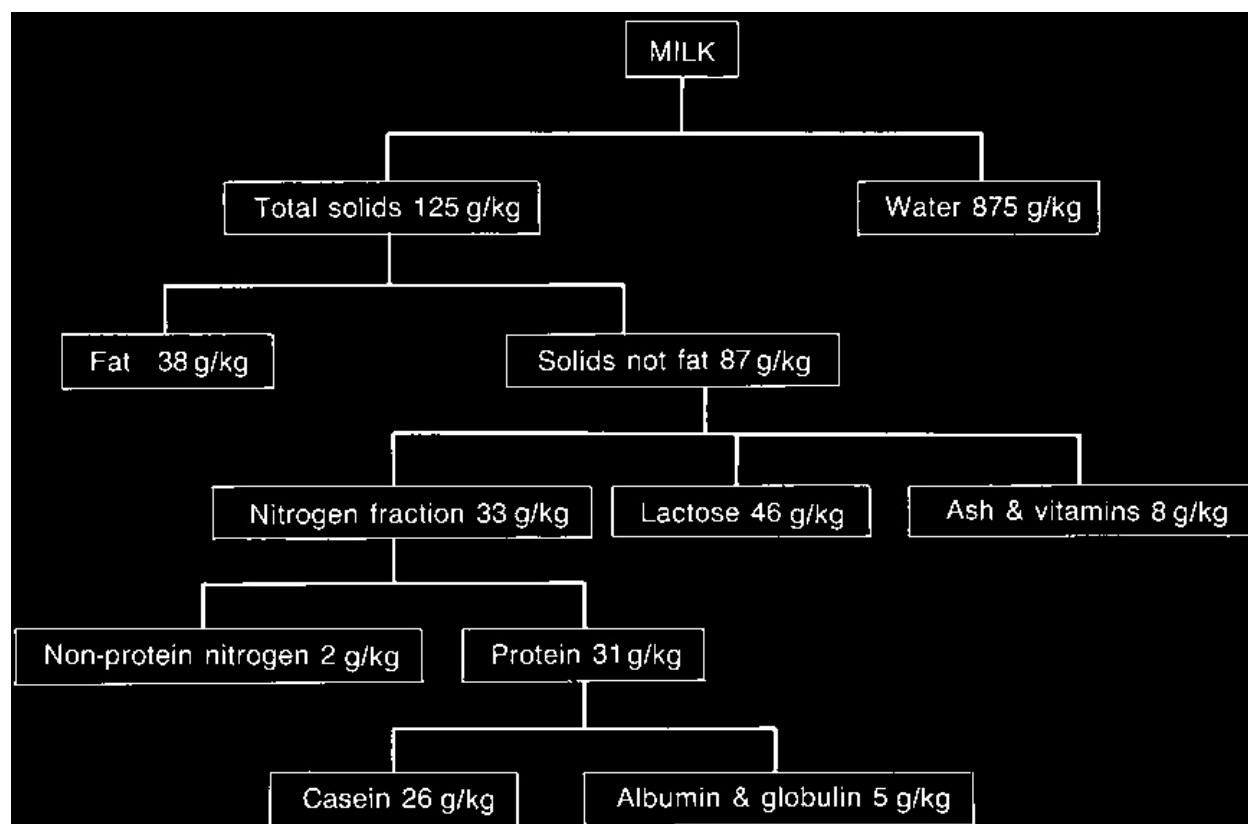
ratio in the ration and its influence on milk fat content.

Milk fat is synthesized from the fatty acids acetate and butyrate, products of the ruminal fermentation of

Feeding and milk composition

forage and other feeds containing fibre. As the level of concentrates in the ration increases, the proportions This is an extremely complex area to study, since it is

of acetate and butyrate fall and that of propionate rises. often difficult to differentiate the separate effects of, There is also a decrease in ruminal pH and a depressed for example, plane of nutrition, system of allocation of activity of cellulolytic degradation, which can eventually result in depressed dry matter (DM) intake. The Superimposed on this are effects of feeding before and extent of the depression varies with the materials being after calving and the problem of differentiating fed, but as an approximate guide, forage : concentrate between compositional values and overall yield. For ratios should not be allowed to fall below 60 : 40 (or example, in a high-yielding cow the milk fat content 50 : 50 if fed as a total mixed ration). For example,



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Fig. 28.1

The compositional quality of milk.

Table 28.1

Effect of proportion and type of concentrate in a mixed diet on milk production in early lactation. Source: Sutton et al.

(1985).

600 (g/kg diet)

700 (g/kg diet)

Starchy Fibrous

Starchy

Fibrous

concentrate

concentrate

concentrate

concentrate

Consumed (kg air dry feed/day)

Hay

7.2

3.5

Concentrates

10.8

14.0

Dietary ADF (g/kg DM)

192

231

136

180

Milk yield (kg/day)

26.3

26.5

32.0

25.5

Fat concentration (g/kg)

41.5

42.9

22.6

36.2

Fat yield (kg/day)

1.09

1.12

0.73

0.91

ADF, acid detergent fibre.

Sutton (1986) calculated that milk fat fell by 5 g/kg (0.05

*• Digestible fibre, for example sugar-beet pulp, cotton
per cent) for every 100 g/kg decrease in the proportion
seed, citrus pulp, etc.*

of hay in the diet, even when the overall energy content

• Fats and oils.

of the ration remained constant. However, there was

Table 28.1 (Sutton et al. , 1985) shows that concentrates a good deal of variation

and probably a better way of containing a high level of digestible fibre cause less estimating dietary effect is to express the overall fibre depression of milk fat and produce an overall higher content of the diet on the basis of acid detergent fibre fat yield (kg/day), even though total milk volume is (ADF). Milk fat has been shown to fall when overall reduced due to lack of starch. This was particularly dietary ADF drops to below 200–250 g/kg DM. This noticeable at the higher levels of concentrate feeding. is equivalent to approximately 450 g long forage/kg It is, of course, the amount of starch in the ration and dietary DM, although on much higher forage diets, milk its conversion into propionate and glucose that is one yield could be depressed.

of the main determinants of the volume of milk pro- The type of concentrate also has an effect. The energy duced. The energy in digestible fibre products such as fraction of a concentrate can be derived from four main sugar-beet pulp is derived from cellulose and hemicel- sources, namely:

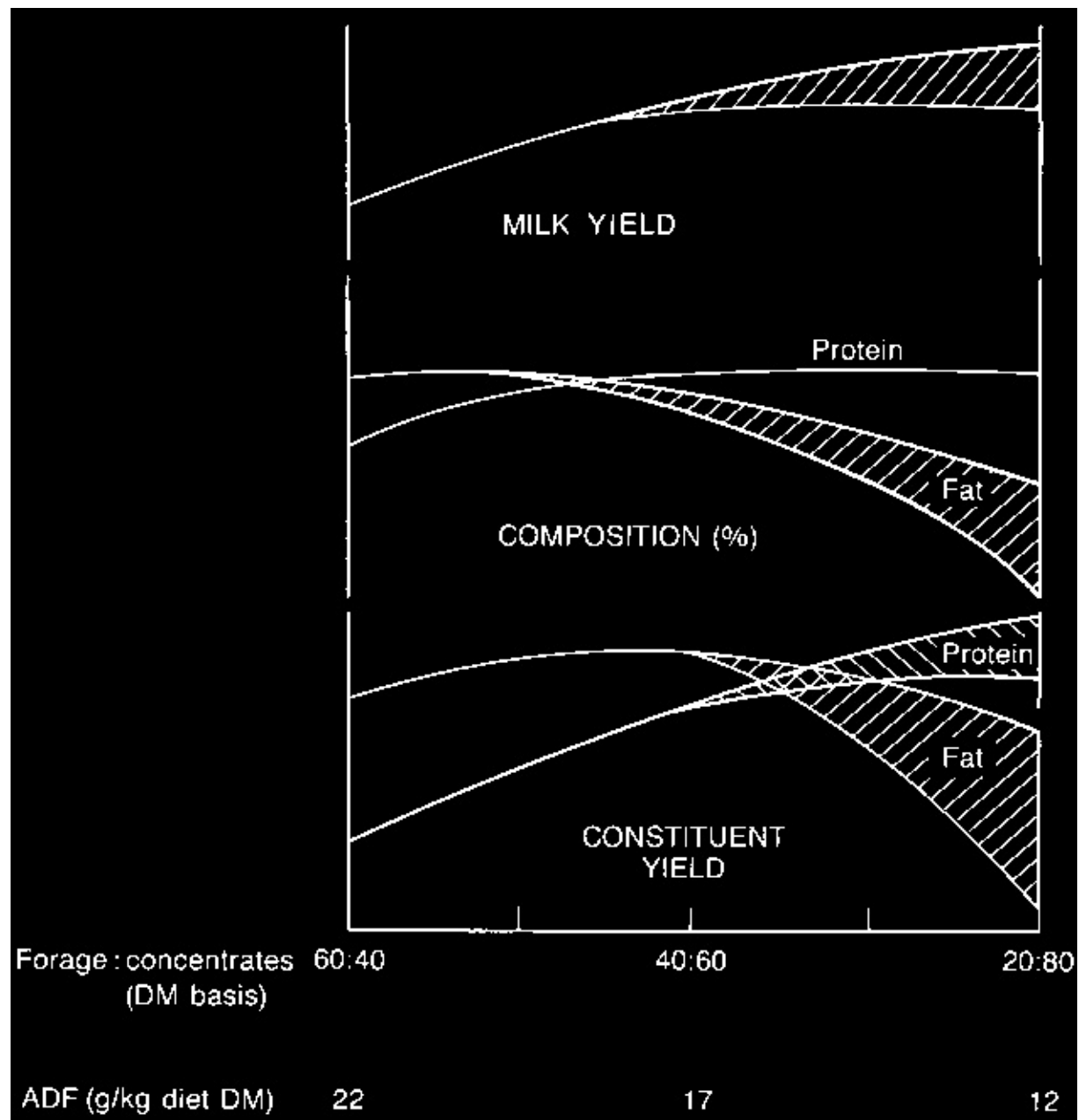
luloses and is slowly fermented. Maize is also slowly fer-

- Sugar, for example molasses.

mented and as such causes less depression of milk fat,

- Starch, for example barley and maize.

compared with other more rapidly fermented starch



and milk quality, but this will depend on the economic milk price : concentrate cost ratio prevailing at the time, and a full discussion is well outside the scope of this review.

Of course, low forage intakes may not occur intentionally. Poor quality fodder such as very wet, very acid or very butyric silages will depress intakes and thereby increase the concentrate : forage ratio. A more common problem is probably inadequate access. Self-feeding consolidated silage, particularly behind an electric wire, may depress intakes of even good quality material by as much as 5–10 per cent and could further exacerbate the problem.

There is no requirement for ‘long fibre’ in the basal ration to maintain milk quality, and diets containing long fibre length generally give the same milk quality as finely chopped silage (Thomas, 1984). However, there is some evidence that silage diets give better milk quality than rations of equal metabolizable energy intake based on hay. Fine grinding of forage will

depress milk fat when used as the sole ration, but if used

Fig. 28.2

The relationship between forage : concentrate ratio or as a supplement to conventional forage it has no effect acid detergent fibre (ADF) and milk yield and composition. (Thomas, 1984).

Dietary fat

sources such as barley. Uncooked ground maize will, to Manufacturers may increase the energy content of a certain extent, pass through the rumen and into the a concentrate by adding fat, either directly into the small intestine. This possibly explains why maize-based product or by fat-spraying the outside of the cubes. Fat concentrates give an overall lower level of yield than 'prills' may also be added to the ration, e.g. a complete conventional barley products. Both features are par-diet, as a separate constituent. Provided that saturated ticularly noticeable at higher concentrate intakes. Root (or 'hard') fats such as tallow, coconut or palm kernel crops, e.g. fodder beet, have most of their energy stored oil are used, and only to a maximum of 5–6 per cent,

as the sugar sucrose. Generally, they produce better this will increase the fat content of the milk. However, levels of fat, but lower yields than a comparable amount milk protein content will be slightly depressed, but as of starch.

milk yield is likely to increase, overall milk protein yield

The effect of diet on milk protein is different from remains constant. Levels of fat above 7 per cent interfere with milk fat. On high-forage diets, increasing fibre with ruminal function, leading to a depression of the proportion of concentrate in the ration (especially both total milk yields and the fat and protein contents. high-starch concentrate) will increase milk protein, but Increased levels of unsaturated ('soft') fats, such as only up to a level of 50–60 per cent of concentrate in maize, cotton seed, groundnut and especially fish oils, the diet. Beyond this, milk yield may rise and milk fat should not be used because they will coat the surface concentration will fall, but protein levels will remain of fibre particles in the rumen, thereby depressing fibre constant. The net effect is an overall rise in protein

digestion and leading to a fall in the milk fat content. yield. These changes are represented diagrammatically. However, if the fat can be protected from rumen degradation in Fig. 28.2.

dation (such as with heating or formaldehyde treatment), the fat will bypass the rumen, resulting in no also increase milk protein, even if requirements for depression of fibre digestion. This will allow the long-metabolizable protein have been met. This effect is chain fatty acids to be utilized more fully and overall enhanced if the amount of rumen bypass starch is milk fat will therefore rise. Protecting unsaturated fat increased, suggesting that the mammary gland requires also has a significant impact on the fatty acid composition of precursors and energy to produce higher quality of milk. As a result of hydrogenation of dietary levels of dietary protein.

fatty acids in the rumen, milk fatty acids are primarily. Clearly, within a quota system there will be an (75 per cent) saturated fat. Protecting unsaturated fat

*optimal level for a farmer, both in terms of milk yield
can significantly increase the proportion of unsaturated*

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fats in milk (and reduce the proportion of saturated

Table 28.2

Effect of number of daily meals of starch-based

fat). Mansbridge and Blake (1997) showed that inclu-

concentrates on milk yield and composition. Source: Sutton

sion of either full fat soybeans or partially oil-extracted

(1986).

rapeseed in the diet resulted in a significant reduction

Concentrates

in the proportion of palmitic acid (the predominant

(kg/day)

10.0

11.5

12.8

14.0

saturated milk fatty acid) in milk. Additionally, feeding

(g/kg diet)

600

700

800

900

oil-extracted rapeseed significantly increased the concentration of oleic acid (a mono-unsaturated fatty

ADF (g/kg diet DM)

192

162

124

99

acid) and linolenic acid (a polyunsaturated fatty acid),

Milk yield (kg/day)

while feeding full fat soyabeans resulted in significant

2 meals

19.3

19.7

20.6

23.0

increases in linoleic and linolenic acid. Milk and milk

6 meals

20.8

20.2

24.5

21.4

products produced from cows fed protected diets could

Milk fat (g/kg)

be a valuable aid in reducing saturated fat intake.

2 meals

34.3

32.6

31.6

17.9

6 meals

36.2

39.2

35.3

29.7

Dietary protein (see p. 106)

Milk protein (g/kg)

2 meals

31.4

33.2

31.7

32.0

*The effects of dietary protein on milk quality, including
6 meals*

31.8

34.1

31.2

33.2

milk protein, are less well defined. It is the energy content of the ration that has the major effect on milk protein, particularly at high forage intakes. Severe dietary protein deficits will depress milk protein, although this may be partly because the requirements are greater, rather than the steady supply of propionate that is available from the ruminal micro-organisms have not been met and this will be a feature of more frequent feeding. The effect of increased total dry matter intake, and therefore the effect of increased levels of insulin is to promote lipogenesis in adipose rather than mammary tissue.

Table 28.2 shows that milk protein is unaffected by

frequency of feeding, or by the level of concentrate fed

Feeding systems

at these high levels of concentrate : forage ratio.

It is possible to overcome some of the effects of high

Complete diets (see p. 120) offer an opportunity for

concentrate, low forage diets by feeding the cows more

even feeds of concentrate throughout the day. When

frequently. In a series of experiments at the National

milk fat is low, due to depression by an excessive con-

Institute for Research in Dairying (Sutton, 1986),

concentrate : forage ratio, feeding the same constituents in

cows were given concentrate diets varying from 600 to

a complete diet will lead to an increase in the fat

900 g/kg (namely 40 per cent to 10 per cent forage) fed

content. Milk protein may also increase on complete

either twice daily or up to six times daily. The results

diets, but this is thought to be due to the effects of an

are given in Table 28.2 and show that for all levels of

increased dry matter intake, rather than any particular

concentrate feeding, milk fat was higher with a greater

effect on frequency of feeding.

frequency of daily feeds. As one might expect, the differential was greatest at the highest level of concentrate p. 24), there has been a greater tendency to feed forage, feeding, where twice daily feeding had produced a and concentrates have been fed on a 'flat-rate' basis, severe depression of milk fat. The concentrate used was rather than the more traditional system of a basic allocation for maintenance and an increasing quantity of Had a highly digestible fibre product been used, it is concentrate depending on yield. Provided that good unlikely that the depression of milk fat would have quality forage is available ad libitum (and this is essential to the success of the system), there is no significant response to more frequent feedings would also have been considerably less. It is effect on milk quality throughout the lactation. Since generally assumed that diets leading to high propionate high levels of 'starchy' concentrates promote high and low acetate in the rumen will reduce milk fat.

yields, cows calving onto a 'flat-rate' system tend to
However, increasing the frequency of feeding does not
'peak' at a lower level in early lactation and hence the
seem to depress propionate concentrations sufficiently
'dilution' effect of high yields depressing milk quality is
to account for the full benefit to milk fat synthesis. It
not seen. Any reduction in milk quality on a flat-rate
would appear that high plasma insulin concentrations
system is compensated for by slightly higher quality
are the main factor depressing milk fat and that the
later in lactation. If access to forage is restricted, then
release of insulin is stimulated by peaks of propionate
clearly decreasing concentrate in early lactation will
production, such as occurs after a large feed of concen-
depress milk protein.

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The depression of milk fat caused by high-concen-

Table 28.3

*The effect of silage feeding at grazing on milk
trate diets can also be counteracted by the use of
quality. Source: Phillips & Leaver (1983).*

buffers. The most commonly used compound is sodium bicarbonate, fed at 12.5–15 kg/t, or 100–125 g/cow per

Grazing

Restricted grazing

day. The extent of the improvement depends on the

and silage

original level of milk fat, the response being greatest in

Milk yield (kg/day)

19.9

18.9

herds where butterfat is already low due to high levels of starchy concentrate being fed twice daily. Feeds such

Milk composition

as sugar beet pulp also have a good buffering capacity

Fat (g/kg)

35.6

39.4

(sometimes also known as the cationic exchange capac-

Protein (g/kg)

35.1

34.8

ity), whereas others, such as maize gluten, can lead to a

Solids yield

more acid fermentation. It is interesting to speculate

Fat (g/day)

708

745

on the effects of the use of sulphuric acid as a silage

Protein (g/day)

698

657

additive. This is a 'strong' acid, thus requiring a greater

buffering, and could exacerbate milk fat problems in a

way that formic or lactic acids would not.

to February) due to the fact that a large number of cows

Non-nutritional factors

are at peak yield and relatively few are pregnant.

Changes in lactose follow an opposite pattern. The

Age of cow

lactose content of colostrum is low, but rises rapidly

after calving to reach a peak by two weeks into lacta-

Heifers generally have the highest milk quality, there

tion. This level is maintained until six weeks, but there being a fall of about 3 g/kg (0.3 per cent) in fat and is then a steady fall, the rate accelerating towards the 7 g/kg (0.7 per cent) in solids not fat (SNF) per lactation thereafter. The rate of decline continues until mirror image of changes in fat and protein.

approximately the fifth lactation, after which it becomes more gradual. The decline in SNF is primarily due to a decline in lactose and mainly occurs during the first

Season of the year

three lactations. Protein declines by only approximately

In the UK there is a sharp fall in milk fat at the end

1 g/kg (0.1 per cent) per lactation over this period.

of winter, when cows are turned out to grazing. This can be partly offset by providing access to hay or straw, although intakes of such forages are often poor when

Stage of lactation

highly palatable grass is available and, if only 1–2 kg are

Milk quality is, of course, very high at calving

consumed, this will have little benefit. To overcome the

(colostrum has at least double the dry matter content, situation (and to improve grassland management and i.e. 25 per cent, of normal milk, see p. 211), but then conservation), a system of 'storage' or 'buffer' feeding declines as yield increases, reaching a minimum at has been introduced, whereby cows are kept in at night about 50–70 days after calving. Milk fat may drop by as on silage. Experiments carried out at Crichton Royal much as 10 g/kg (1.0 per cent) and protein by 3 g/kg (0.3 Farm (Table 28.3) showed that this significantly per cent) over the period. This depression is partly due improved butterfat and although yields were marginally to a 'dilution' effect of high yields and partly to the lower, overall fat yield was increased. However, protein inherent inability of the early lactation cow to consume yields were lower.

sufficient energy to meet the demands of production.

Often there is also a fall in milk protein during the Feeding high levels of 'starchy' concentrates and, in winter housing period (November to March in the UK), so doing, boosting the volume of milk produced, can in

probably due to a lower energy content of the diet at some circumstances exacerbate the decline in milk this time of the year and also to a stage of lactation quality. Both milk fat and protein tend to increase after effect. Protein levels then rise rapidly in April and May, 70 days, but milk protein only rises significantly if the following turnout to spring grazing. There is very little cow becomes pregnant, and the rapid rise in both fat seasonal change in lactose concentrations.

and protein that occurs in later lactation (e.g. six months after calving) is greater in the pregnant animal.

Disease

Farms practising 'block calving', for example from August to October, often experience a marked fall in Liver fluke (see p. 276) can depress both milk fat and milk quality two to three months later (e.g. November milk protein and this depression in milk quality may be

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Table 28.4

Approximate breed variations in milk yield and

Management factors

quality. Source: MMB (1983).

Milking intervals would appear to have an effect on

Milk yield

Milk fat

Protein

milk quality in that on twice-daily milking, the fat

(kg)

(g/kg)

(g/kg)

content is higher in the afternoon. This is entirely

due to a 'carryover' effect (Dodd, 1984). At the end of

Ayrshire

4988

39.0

33.8

milking, some 10–20 per cent of the milk present

British Friesian

5610

37.8

32.6

remains in the udder and cannot be removed. The fat

British Holstein

6292

37.3

32.0

content of this milk is very high, 150 g/kg (15 per cent)

Guernsey

4017

46.4

36.3

or higher, and it is withdrawn at the next milking. If

Jersey

3876

51.9

38.5

there is an uneven interval between milkings, milk produced after the shorter interval will have a higher fat content because of the reduced 'dilution' effect of the additional milk produced. The total daily fat production remains constant, irrespective of the variation in milking interval and therefore of fat content. Increased in the absence of any other clinical signs of fluke.

ing the frequency of milking to three or even four times
Heavy infestations of lice (see p. 741) and gastroin-
daily does not alter milk quality significantly, although
testinal worms (see p. 267) may also reduce milk
yields may rise by 10–15 per cent.

quality, but these are unlikely in adult milking cows.

Mastitis (see Chapter 23) leads to a reduction in
yield, lactose and butterfat. For example, a herd with a

References

cell count of 750 000 cells/ml could be losing 750–900
l/cow per year, 50 g/kg (0.5 per cent) lactose and a

Crabtree, R.M. (1984) Milk compositional ranges and trends.

smaller amount (30 g/kg, 0.3 per cent) of milk fat. Milk

In Milk Compositional Quality and its Importance in Future protein levels will
increase slightly with mastitis, but

Markets (ed. by M.E. Castle & R.G. Gunn), pp. 35–42.

the protein is of lower quality, with increased levels of

BSAP Occasional Publication No. 9, Edinburgh.

globulin and decreased casein. High cell count milk is

Dodd, F.H. (1984) Herd management effects on compositional
therefore of reduced value for manufacture.

quality. In *Milk Compositional Quality and its Importance in Future Markets* (ed. by M.E. Castle & R.G. Gunn), p. 77.

BSAP Occasional Publication No. 9, Edinburgh.

Garnsworthy, P.C. & Topps, J.H. (1982) *The effect of body con-*

Genetic variation

dition of dairy cows at calving on their food intake and per-

formance when given complete diets. Animal Production, It is well known that Channel Island cattle such as

35, 113–19.

the Jersey and Guernsey have higher milk quality than

Mansbridge, R.J. & Blake, J.S. (1997) Nutritional factors

other breeds. Approximate breed values are given in

affecting the fatty acid composition of bovine milk British Table 28.4. Of course there are large variations between

Journal of Nutrition, 78 (Suppl 1), S37–S47.

individual animals within a breed, and this is the basis

National Dairy Council (NDC) (1997) United Kingdom Dairy

of genetic selection. The selection of animals on the

Facts and Figures, pp. 1–208. National Dairy Council,

basis of yield alone could lead to a decrease in fat and

London.

protein contents and care should be taken to ensure

Phillips, C.J.C. & Leaver, A.D. (1983) The effect of offering that bulls with a high improved contemporary compar-silage to set-stocked dairy cows. Animal Production, 36, 507.

ison (ICC) for both fat and protein are selected. There

Sutton, J.D. (1986) In Principles and Practice of Feeding Dairy Cows, pp. 203–18. NIRD Technical Bulletin No. 8.

is less genetic variation within breeds for protein

Sutton, J.D., Bines, J.A. & Napper, D.J. (1985) Composition of and lactose than for fat and hence the greatest rate of

starchy and fibrous concentrates for lactating dairy cows.

genetic progress will be made by selecting for fat. For

Animal Production, 40, 533.

example, the range of fat content within a breed is over

Thomas, C. (1984) Milk compositional quality and the role of

20 g/kg, whereas genetic variation for protein is only

forages. In Milk Composition Quality and its Importance

approximately 10 g/kg (Crabtree, 1984). The genetic

in Future Markets (ed. by M.E. Castle & R.G. Gunn), pp.

variation for lactose is even lower.

69–76. BSAP Occasional Publication No. 9, Edinburgh.

Chapter 29

The Enhancement of Bovine Mammary

Gland Immunity Through Vaccination

K.P. Kenny, T. Tollersrud and F.D. Bastida–Corcuera

Introduction

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Anatomy of the bovine mammary

Anatomy of the bovine mammary gland

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gland (see Chapter 22)

Defence mechanisms of the gland

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Immunoglobulin in lacteal secretions

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Biological activity of antibody

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The bovine udder comprises four mammary glands.

Complement

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The main arterial supply is from the bilateral external

Cells of the mammary gland

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pubdental arteries. The venous return is via the external

Cytokines

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pudendal veins and the subcutaneous abdominal veins.

Intramammary infection

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There are two lymph vascular systems: a superficial and

Virulence factors and immune response to major

a deep set. The superficial set drain the cutaneous area

pathogens

383

and the teat walls. The deep set is associated with the

Escherichia coli

383

finer branches of the arteries and veins. The lymphatics

Klebsiella spp.

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drain into the superficial inguinal (supramammary)

Mycoplasma spp.

385

lymph nodes and the lymph passes to the deep inguinal

The summer mastitis complex

385

Staphylococcus aureus

385

node.

Streptococcus spp.

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The mucosa of the teat or streak canal is arranged in

Mucosal immune system

388

longitudinal folds and is lined by stratified squamous

Generation of immune response

388

epithelium which constantly undergoes keratinization.

Limitations of vaccination

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A slightly projecting fold marks the proximal end of

the teat canal, the Furstenberg's rosette area. The teat

cistern (sinus) is the cavity directly proximal to the teat

canal, and this continues into the parenchyma of the

Introduction

udder as the gland cistern (lactiferous sinus), which pos-

sesses a two-layered columnar epithelium. The secre-

The examination of immunological methods to increase tory tissue is divided into lobes which are composed resistance of the dairy cow to pathogens which cause of lobules and there is an extensive duct system. The mastitis has been ongoing for almost a century. This is epithelium of secretory alveoli is a single layer of a controversial area of bovine immunity with the exis-cuboidal cells. The alveoli are surrounded by a stromal tence of two schools of thought on the role of immuno-layer which is more pronounced in the non-lactating logical intervention in mastitis control programmes. gland.

The first believes it is not possible to generate protec-tive immunity in the mammary gland and control meas-ures should be based on management, therapeutic strategies and genetic selection (Mellenberger, 1977).

Defence mechanisms of the gland

The second opinion is vaccination has a role in a mastitis control programme (Colditz & Watson, 1985). Non-specific and specific defence mechanisms exist in The latter view, founded on an improved knowledge of

the bovine mammary gland (Outteridge & Lee, 1988).
the bovine immune system and the principal pathogens
Non-specific factors include teat and teat duct shape
of the gland, is supported by reports which indicate
and structure, teat duct patency and teat duct keratin.
heightened resistance to infection with certain
Teat duct keratin owes its protective properties to fatty
pathogens. The success of coliform mastitis vaccines has
acids and basic proteins. During milking there is a
spurred novel studies on *Streptococcus uberis* mastitis.
mechanical flushing of the teat canal. The enzyme
Such vaccines will complement conventional control
lysozyme can cleave certain bacterial cell walls, but is
measures which are less effective against environmen-
present at very low concentrations in bovine secretions.
tal pathogens.

Lactoferrin can bind iron and reduce the availability of

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iron to micro-organisms, but citrate inhibits this pro-

Table 29.1

*The concentration of immunoglobulin in serum and
tein. The lactoperoxidase/thiocyanate/hydrogen perox-
colostral secretions. Values are expressed in mg/ml.*

*ide system has activity against micro-organisms. Com-
plement can be activated by the lipopolysaccharide of*

IgA

IgM

IgG1

IgG2

Gram-negative organisms and cause lysis of these

Serum

0.37

3.0

11.2

9.2

bacteria by the alternative pathway.

Colostral whey

4.7

7.1

48.2

4.0

Four specific defence mechanisms operate in the

Milk whey

0.08

0.08

0.5

0.06

gland. These are mediated by antibodies directed against epitopes of the pathogens or their toxins, in conjunction with phagocytic cells and complement. The secretions, but not in milk. Around 50 to 100 per cent mechanisms include prevention of bacterial adherence of milk IgA is produced locally. Plasma derived IgA is to epithelial cells, neutralization of bacterial toxins, selectively transported into most mucosal secretions by opsonization of pathogens and direct lysis of pathogens. vesicular transport following its binding to secretory The key role of humoral immunity reflects the extra-component on epithelial cells. There are conflicting cellular location of mastitis pathogens. A role for cellular immunity has not yet been established although

indicating it is entirely serum derived while others laboratory studies have shown the ability of staphylococci and streptococci to achieve an intracellular localization in cultured mammary epithelial cells. Most milk IgG1 is serum derived. About 90 per cent of IgG2

found in milk is locally produced. To increase specific Two methods can be employed to heighten resistance. IgG1 in milk one must increase the serum level of specific IgG1, whereas for IgA one must generate appropriate plasma cells in the lamina propria.

strategic use of cytokines at key points in the lactation cycle has yielded encouraging results. The administration

Biological activity of antibody

tion of gamma-interferon lessens the severity and duration (see Chapter 22)

tion of experimental *Escherichia coli* infection, but there are no reports of field trials describing the effect

Antibody molecules can be considered as having two

of such administration on naturally occurring infection components. Firstly the two antigen binding sites recognize and bind antigen. Secondly, the Fc portion of the immunity for each mastitis pathogen. The latter is difficult because there are many causative bacteria. The fix complement components and interact with specific challenge is to characterize the virulence factors of the Fc receptors on phagocytic cells. For certain protective principal pathogens, identify their protective epitopes effector functions, antibody must not only be directed and then establish and maintain sufficient protective against a particular epitope but must be of the correct antibody in milk.

isotype.

Secretory IgA serves to protect mucosal surfaces from colonization and penetration by undesirable

Immunoglobulin in lacteal secretions

micro-organisms. It can augment the bacteriostatic (see p. 323)

effects of lactoferrin and can function as an opsonin.

Four classes of antibody have been described in the

Lactateal IgA is largely associated with milk fat globules.

bovine (Butler, 1986): IgA, IgE, IgG and IgM. IgG1 and

IgG1 and IgG2 are efficient at fixation of complement

IgG2 are two subclasses of IgG. The half-life in serum

and able to neutralize bacterial toxins. Cytophilic anti-

of IgA, IgM, IgG1 and IgG2 is 3, 5, 17 and 22 days respec-

bodies are capable of attaching to certain cells in such

tively. Serum IgA of cattle is probably synthesized by

a way that these cells can subsequently adsorb specific

bronchial or gut associated lymphoid tissue, while IgM

antigen. It has been shown that IgG2 is cytophilic for

and IgG are most likely synthesized by the spleen and

bovine blood neutrophils and for ovine mammary neu-

peripheral lymph nodes. Immunoglobulin concentra-

trophils. Bovine macrophages were found to be capable

tions in serum and colostrum secretions are listed in

of binding both IgG1 and IgG2 in vitro. Live vaccines

Table 29.1.

are believed to preferentially stimulate IgG2. IgM is 10

*Antibody present in lacteal secretions may be syn-
to 20 times more effective than IgG in complement fix-
thesized locally or derived from serum via selective
ation. This ability to fix complement may explain why
transport or transudation. Inflammation of mammary
IgM is bactericidal for Gram-negative pathogens. In
epithelium causes an increase in the passage of anti-
this manner it can also opsonize micro-organisms for
bodies from blood to lacteal secretions via transuda-
uptake by phagocytic cells. IgM can also agglutinate
tion. IgA is the major immunoglobulin in most bovine
bacteria and permit them to be flushed out at milking.*

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Table 29.2

The cellular composition of gland secretions (%).

PMNa

Macrophage

Lymphocyte

Epithelial

Colostrum

62

35

4

0

Milk

3

79

16

2

Dry gland secretion

3

89

7

1

a PMN, polymorphonuclear neutrophil.

Complement (see Chapter 58)

Table 29.3

Lymphocyte composition using flow cytometry.

The complement system consists of more than 29 dis-

T cell

B cell

Null

Macrophage

tinct plasma and membrane proteins, several of which are enzymes. The system is activated either by antibody

Peripheral blood

48

20

10

22

binding to the bacterium (classical pathway) or by car-

Milk

52

14

9

25

bohydrate structures on the surface of the organism

Dry gland

43

19

11

27

(alternative pathway). Triggering of the system results in secretion

in a sequence of biochemical reactions in which one component activates another component in a cascade fashion. Complement thus bridges the innate immune system based on recognition of foreign carbohydrates and the induced immune system based on specific anti-

Cells of the mammary gland

body formation.

The terminal part of the reaction is the formation

In dry and lacteal secretions the predominant cell type of a membrane attack complex which causes lytic is the macrophage while in colostrum the polymorphonuclear neutrophil (PMN) serves this role. The organism. Resistance to complement (serum resistance) is the only virulence factor identified among strains of Gram-negative bacteria isolated from cases of mastitis. The cellular composition of gland secretions is outlined in Table 29.2.

bovine mastitis. Serum-sensitive bacteria are either killed or do not grow in normal milk, whereas serum-resistant organisms generally multiply, and only the latter are able to cause mastitis.

Identification of cells in milk and blood can be performed by microscopic examination of stained smears. Flow cytometry is a sensitive technique which allows further differentiation of the mononuclear cell population in blood and gland secretions (Table 29.3). There are several effector mechanisms by which complement aids phagocytosis. When the complement cascade is activated, component C3b is deposited on the surface of the bacterium. This C3b coating promotes gland secretion.

adherence to and ingestion by phagocytes, which Mammary lymphocytes can respond to mitogens possess surface receptors for C3b. Products of complement activation include C3a and C5a which are

responses are lower than those of peripheral blood chemoattractant for neutrophils and C5a can activate lymphocytes from the same animal. T lymphocytes can neutrophils. The inflammatory mediators responsible produce cytokines which effect recruitment and activation for the influx of neutrophils into milk have not been identified. T cells may be further subdivided according to their accessory molecules (CD4 or CD8) or antigen receptors (alpha-beta or gamma-delta). The majority of T cells in milk express alpha-beta T cell receptors. The ratio of CD8⁺ to CD4⁺ cells in blood is 1.5, while in mammary secretions it is 0.85. Some activated CD8⁺ cells may suppress the proliferation of CD4⁺ cells in the course of *S. aureus* mastitis and an increase in

complement components in milk due to this may adversely effect antibody production by B transudation. cells.

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The mammary macrophage functions in phagocytosis may reflect the poor phagocytic efficiency of these cells and killing of bacteria, eliminates fat in the involuting or the unfavourable nature of the non-lactating gland gland and plays a role in local immunity through for them. Fc receptor function is impaired by dry secretion and processing and presentation. After they have tion and colostrum.

phagocytosed bacteria, mammary macrophages generate PMN function has limitations, as evidenced by ate chemoattractants for PMN, while after exposure to chronic infections with some pathogens persisting in the endotoxin they release cytokines. Although critical for face of a high SCC. To increase the efficacy of phago-

phagocytosis of *Strep. uberis* they may provide an intra-cytosis one could increase and maintain a high level

cellular location for Staph. aureus and their phagocytic of opsonin in milk through immunization, select for

capacity is reduced by dry secretion. A considerable cows with superior phagocytic ability or decrease PMN population of macrophages exists beneath the alveolar mobilization time in response to pathogen invasion. and ductal epithelium.

*Antigen-specific inflammatory responses can be estab-
Milk from normal uninfected glands contains less
lished in the gland by parenteral immunization and one
than 300 000 cells per millilitre and the predominant
of the effector inputs of vaccination is the more rapid
cell is the macrophage. Subclinically infected quarters
response of the PMN to infection. One could also
contain around 750 000 cells per millilitre, while the
increase and maintain a high PMN concentration in
secretion of infected quarters contains millions of cells
milk; however, this will adversely affect milk quality.
per millilitre and in both these instances the pre-
dominant cell is the polymorphonuclear neutrophil
leukocyte (PMN). A pre-existing leukocytosis confers*

Cytokines

increased resistance to infection; however, increased Cytokines are a group of regulatory proteins which act as intracellular communication signals and are secreted in very low amounts by a variety of cell types. Most cytokines possess multiple biological properties and regulate the activity of cells which participate in specific pathogens is achieved. Such high cell counts are not and non-specific immunity. Some can also alter physiological processes and cause pathological changes. There is a large pool of mature neutrophils present. The immunomodulatory capacity of these molecules is to fight infection. The half life of the cell in the blood-complex and interactions exist between cytokines – the stream is about nine hours, with a substantial number cytokine network. Tumour necrosis factor (TNF), inter-

developing in the bone marrow. Upon infection of the leukin-1 (IL-1), interleukin 2 (IL-2), interleukin 6 (IL-6) and interleukin 8 (IL-8) have been detected in milk glands, PMNs adhere to capillary endothelium via receptors and pass through it by diapedesis. They then migrate down chemotactic gradients and enter the lumen of the gland through the mammary epithelium.

Antibody and complement promote contact and recognition-adjuvants and therapeutic agents is being evaluated.

nition of the pathogen, which is ingested and exposed to the microbicidal system of the PMN. Both oxygen dependent and independent pathways exist within neutrophils to kill ingested microbes. Some bacteria such

Intramammary infection

as *Staph. aureus* possess defence mechanisms against (see also Chapter 23)

oxidative damage by neutrophils and may survive within the phagosome. PMNs are viable for 1–2 days in To induce mastitis a pathogen must pass the teat canal

the mammary gland, then undergo apoptosis (pro- and enter the gland, survive the bacteriostatic and bactericidal mechanisms and use the available nutrients to Phagocytosis by mammary PMNs is much less efficient than that by blood PMNs. PMN function in the adherence capacity of the pathogen and its ability to mammary gland is impaired by milk fat globules, casein utilize nutrients in lacteal secretion to multiply. *Strep.* and the lower glycogen content of milk PMN compared *agalactiae* and *Staph. aureus* adhere readily to ductular to blood PMN. PMN function can also be impaired by epithelial cells, while *Escherichia coli*, *Klebsiella* spp. bacterial toxins. Cows vary in both the ability of their and *Arcanobacterium pyogenes* adhere poorly. If they PMN to phagocytose and in the capacity of their milk are to maintain infection within the gland, bacteria must to support phagocytosis. Despite high levels of phagocytic cells in the gland during the dry period, the gland

are eliminated. The outcome of infection depends on
is very susceptible at this time to new infection. This
the rate of growth of the organism, the production,

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absorption and activity of toxins and the immune status
all of these quarters had peracute toxic mastitis after
of the host.

parturition.

Following infusion of 500 colony forming units
(CFU) of *E. coli*, newly calved cows mobilize PMN

Virulence factors and immune

slowly and appear to be refractory to the presence of
response to major pathogens

irritants in the udder. This permits great bacterial repli-
cation and a large subsequent release of endotoxin

which is often fatal (Hill, 1979). Killing of some *E. coli* ***Escherichia coli*** (see
also Chapter 23)

strains within the udder requires a neutrophilia only,

Escherichia coli mastitis is an opportunist infection.

whereas for others up to 30 per cent serum products

When 290 *E. coli* isolates were examined, 82 per cent

are required. For virulent strains a PMN response and could be grouped into 63 O-serogroups, the remainder opsonin are typically required. In decompemented being untypable (Linton & Robinson, 1984). Mastitis non-immune serum, colostrum and whey, the main isolates do not possess the virulence factors associated opsonin for *E. coli* is IgM and there is no absolute with invasive or enteropathogenic strains of *E. coli* and requirement for complement in the opsonization of *E.*

cannot be distinguished from faecal isolates. Serum *coli* by the adult dairy cow. Later research found IgG2 resistance, which is the capacity to withstand killing by from immune bovine serum or whey opsonic for *E. coli*. complement, is the key feature of mammary isolates.

Encapsulated isolates are more resistant to uptake by The contribution of a capsule is unclear because most PMN and require greater amounts of opsonin than non-unencapsulated strains are serum resistant. Serum-encapsulated isolates.

sensitive strains are killed rapidly, with over 90 per

The Gram-negative cell wall consists of an inner cyto-

cent killed in 15 minutes by freshly collected 10 per cent
plasmic membrane and a cell wall which is composed
normal bovine.

of mucopolysaccharide–peptidoglycan, phospholipid

When serum-resistant strains gain access to the
protein and lipopolysaccharide. LPS is composed of
lactating gland they commence to multiply and
three distinct subunits. The outermost O polysaccharide
lipopolysaccharide (LPS), also called endotoxin, is
is linked to the core polysaccharide which is covalently
released from the bacterial cell wall. Endotoxin is found
bound to the lipid A. Free lipid A in soluble form
in 50–100 per cent of lacteal secretions and 15 per cent
expresses all the typical in vivo properties of endotoxin.
of blood samples of cows with coliform mastitis. The
Resistance to the bactericidal activity of normal serum
exaggerated response of the host to endotoxin is often
is attributable to the O antigen of the LPS. When com-
responsible for the severity of this mastitis. Fever,
pared to serum-sensitive strains, LPS from serum-
disseminated intravascular coagulation, hypotension,

resistant strains is relatively enriched in long-chain O shock, complement activation and death can occur.

antigen subunits. These O polysaccharides may mask

Macrophages are the principal method by which

lipid A which could activate the alternative comple-

endotoxins are removed. In the presence of LPS

ment pathway. If the longer polysaccharide chains do

macrophages become activated and secrete IL-1, IL-6

bind C3b, the distance from the outer membrane of the

and TNF. These cytokines mediate endotoxic events

bacteria is too far for insertion of the complement

and are responsible for many of the clinical signs asso-

membrane attack complex.

ciated with this condition.

The O polysaccharides are made up of a series of

E. coli does not adhere to mammary epithelial cells.

identical repeating oligosaccharide units containing

The organisms grow in secretions and there is little

three to five sugars each. The O polysaccharide is

tissue invasion. Mammary lesions in *E. coli* mastitis are immunodominant and displays great diversity in length

confined to the superficial layer of the teat and lactiferous sinus where extensive sloughing and necrosis is conserved. Antibody to the O polysaccharide antigen of epithelial cells is evident. Secretory tissue is not or LPS affords protection against homologous challenge. The response of cows to *E. coli* infection of the gland only. Because of the great diversity of the O polysaccharide the gland may be a function of the physiological state.

saccharides of LPS, a search for shared epitopes on the gland. The dry gland is much less responsive to other parts of the molecule has been undertaken in an endotoxin than the lactating gland and mild histopathological effort to find an antigen that could be used for active immunization. *E. coli* is immunization. Gram-negative core antigens have

incapable of establishing itself permanently in the gland immunological homology across bacterial species, but in the first half of the dry period and this may be are typically masked by the immunodominant O due to lactoferrin (Bramley, 1976). One experiment polysaccharide.

revealed that in the 30 days before parturition bacteria
In contrast to smooth organisms, rough mutants,
maintained infection in 14 of 37 inoculated quarters and
called so because of their colonial morphology, have
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incomplete synthesis of O polysaccharide side chains
and dry matter intake was higher in vaccinates. In a sep-
and consequently varying amounts of the core gly-
arate trial of similar design, cows were challenged one
colopid are exposed. The E. coli J5 strain is an Rc
month after calving with a field strain known to cause
mutant of E. coli O11 : B4 and has an LPS devoid of O
mild clinical mastitis. No differences in temperature,
antigen (see p. 1016). Animals immunized with killed J5
milk production and systemic signs were observed
bacteria are able to make antibodies to the core region
between groups, but vaccination reduced the duration
and are protected from challenge with heterologous
of both infection and local clinical signs.
live organisms. Antibody to J5 cross-reacts with LPS
It is known that cows which succumb to natural clin-

extracted from other Gram-negative bacteria which
ical coliform mastitis are in a complex physiological
cause mastitis, in particular *Klebsiella* spp. The protec-state with alterations in
neutrophil function, including
tive factor induced by vaccination is considered to be
receptor expression, rate of diapedesis and phagocytic
antibody to core glycolipid which binds to invading bac-
capacity. Measurement of these parameters can
teria at a time when the core antigens are most exposed,
predict which cows will suffer severe clinical mastitis in
that is, early in their growth phase. This antibody pro-
response to experimental challenge. These animals may
motes opsonization of Gram-negative bacteria and may
receive greatest benefit from vaccination and field trials
also be bactericidal or reduce growth of the organism.
would more accurately reflect this benefit. For immuno-
The protective ability of antibodies to core glycolipid
logical prevention of Gram-negative infection to be a
may also reflect their ability to bind to released LPS,
possibility three requirements must be met. A common
neutralize the toxic effects of lipid A and thereby dimin-

antigenic structure must exist; this must function as an
ish systemic signs. Such release would occur during bac-
immunogen and the response to this immunogen must
terial growth or after antibiotic therapy, phagocytic
generate protective antibodies. Large field trials indi-
destruction or complement mediated lysis.

cate that the J5 bacterin achieves this goal. Cows vac-
Initial studies of IgG1 antibody titres to the core anti-
cinated in the dry period and at calving have a lower
gens of J5 showed that titres of less than 1 : 240 were
incidence of clinical coliform mastitis in early lactation.
associated with 5.33 times the risk of clinical coliform

The vaccine does not reduce the incidence of intra-
mastitis (Tyler et al. , 1988). This vaccine has been the mammary infection with
coliforms at calving, but

subject of several field trials. In one trial, 246 cows
reduces the number of infected quarters which display
received three doses of J5 killed cells (7.5 billion bac-
clinical signs. Two new areas of research include the use
teria per dose) in Freund's incomplete adjuvant, twice
of a *Salmonella typhimurium* Re17 bacterin to prevent

in the dry period and once after calving, while 240

coliform mastitis and the effect on *E. coli* of antibodies unvaccinated cows served as controls. Six cases of clin-specific for membrane proteins involved in iron

ical coliform mastitis occurred in vaccinates with 29

transport.

occurring in control cows (Gonzalez et al. , 1989). In a separate trial which lasted 30 months, cows received

Klebsiella spp. (see also Chapter 23)

either vaccine or placebo at dry-off, 30 days later and

at calving. There was no difference between groups in

Klebsiella pneumoniae (*K. pneumoniae*) and *K. oxytoca* the percentage of quarters infected with Gram-negative

are environmental pathogens and opportunist invaders

bacteria at calving. In the control group, 67 per cent of

of the bovine mammary gland. Within the genus *Kleb-*

these infections became clinical in the first 90 days of

siella there are over 70 capsular types and eight somatic lactation compared to 20% in the vaccinated group

types or O groups. Thirty-three capsular types of *K.*

(Hogan et al. , 1992a).

pneumoniae were isolated from cows belonging to 12

Cows directly challenged with coliform bacteria via

herds and within individual herds up to 13 types could the intramammary route have provided data which are be found. The primary feature of pathogenic strains less supportive of the efficacy of the J5 vaccine. One is their ability to withstand the bactericidal effects of experiment found no difference between vaccinates normal serum. Some are also adapted to grow in the and control cows subsequent to challenge with 150 secretion of non-lactating glands. Following infusion of CFU of a serum-resistant strain (Hill, 1991). Both small numbers of serum-resistant bacteria into normal groups displayed acute clinical mastitis with systemic mammary glands a severe mastitis is evident. This mas-signs, a decrease in milk production and large numbers titis is prevented by a pre-existing leukocytosis or by the of bacteria in the milk. In an American study, cows presence of specific antibody.

immunized at drying off, 30 days later and at calving Cell wall O antigens are responsible for resistance to were challenged with a virulent field strain 30 days into serum bactericidal activity. The capsule confers resist-

*lactation (Hogan et al. , 1992b). Lower milk bacteria
ance to phagocytosis in the absence of specific antibody.
counts were observed in vaccinates but SCC and local
Although type-specific protection can be achieved by
clinical signs did not differ between groups. Milk yield
vaccination with the homologous capsular polysaccha-*

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*ride, the numerous capsule types recovered from lacteal
A. pyogenes does not adhere to mammary epithelial
secretions preclude the use of a polyvalent capsular
cells and this may explain why summer mastitis is typi-
vaccine. Antibody specific for core glycolipid of E. coli
cally associated with the non-lactating gland. This bac-
J5 strain cross-reacts with endotoxin extracted from K.
terium grows best in lacteal secretion when the pH is
pneumoniae. This antibody is reputed to bind to these
greater than 7, as in the dry gland, and secretes
core antigens during the log phase of bacterial growth
haemolysin and proteases. P. indolicus stimulates the and in this manner afford
protection.
growth and haemolysin production of A. pyogenes. High*

titres of protease antibodies are found in the serum of infected cows. *P. indolicus* and the microaerophilic

Mycoplasma spp. (see also Chapter 23)

coccus produce enzymes which can damage tissue.

Mycoplasmas lack a cell wall and are surrounded by a

Early vaccines were composed without regard to the

membrane which is similar to the cytoplasmic mem-

complex nature of the aetiology, typically containing *A.*

brane of bacteria. Several species of mycoplasma cause

pyogenes with or without hemolysin, and the results

mastitis but *Mycoplasma bovis* (*M. bovis*) is most were poor. Sorensen showed that *A. pyogenes* alone

prevalent. Cows in all stages of lactation are suscepti-

had no protective effect but that a triple bacterin prepa-

ble but those in early lactation suffer more severely.

ration composed of *A. pyogenes*, *P. indolicus* and the Antibiotics have little success but after a prolonged

microaerophilic coccus gave encouraging results and

time cows may recover. Resistance to killing by serum

merited further study (Sorensen, 1972). There is a

is a virulence determinant and a pre-existing leukocy-

marked lack of data on the immune response of the cow

tosis does not prevent cows from challenge. Neutrophils to these pathogens and approaches to vaccination will are not capable of killing *M. bovis* unless specific anti-remain empirical for some time. body is present.

Udders with a quarter which had resolved infection

Staphylococcus aureus (see also Chapter 23)

were examined and it was found all quarters of the udder were resistant to challenge. Quarters recovered

Staphylococcus aureus has been described as a persist-from infection were immune for up to six months, but

ent pathogen of the gland due to the failure of therapy over one year postinfection all quarters were susceptible during the lactating period to achieve bacteriological ble to reinfection. Studies of the immune response to cures. It is a contagious pathogen and infusion of 10

M. bovis have failed to differentiate between resistant CFU into the teat sinus can cause mastitis. Much

and susceptible animals. Systemic antibody does not research has been performed to ascertain the benefit appear to protect from infection and the immunity may of immunization with staphylococcal antigens. Early

be based on locally produced IgA and IgG and the attempts at vaccination were unsuccessful and these action of T lymphocytes. Immunization with protein failures can be explained to a degree by the low extracts assists cows in eliminating infection. No attenuated strains have been identified.

bacteria in conventional media which does not support the expression of certain surface polysaccharide virulence factors. Later work demonstrated limited protec-

The summer mastitis complex

tion against homologous strains, a reduction in the (see also Chapter 24)

severity of clinical infection and a reduction in spread

There are conflicting views on what are the causative of *Staph. aureus* mastitis in vaccinated herds.

organisms of summer mastitis and how these organisms

The cell wall is composed of peptidoglycan, teichoic spread and invade the gland. Summer mastitis typically acid and protein A. Protein A can bind to the Fc portion affects the non-lactating gland of heifers or dry cows and

of IgG molecules. Peptidoglycan was once considered involves mixed bacterial infections. *Arcanobacterium* to be the key cell surface component involved in *pyogenes* (*A. pyogenes*), *Peptostreptococcus indolicus* promoting opsonization of *Staph. aureus*. Most bovine (*P. indolicus*), a microaerophilic coccus and *Streptococcus*-mammary isolates of staphylococci produce additional *cus dysgalactiae* are the pathogens believed to have a surface polysaccharides which are located outside the role in this condition. *Fusobacterium necrophorum* and peptidoglycan layer. These polysaccharides may block *Bacteroides melaninogenicus* have also been recovered antibody binding to peptidoglycan or prevent bound from the secretion of affected glands. Sorensen (1972) antibody interacting with phagocytic receptors. Their could induce summer mastitis in heifers with *A. pyo-* precise composition remains obscure and they have genes and *P. indolicus* or with these two organisms and been described as exopolysaccharides, pseudocapsules a microaerophilic coccus. Cultures were more virulent if and slime. Their production is typically associated with grown together and infused rather than grown separate growth in milk. Such growth increases the virulence

rately and then mixed for infusion.

of a strain, induces anti-phagocytic properties and

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increases adherence to epithelial cells. A typing scheme

was not statistically significant (Nordhaug et al. , 1994).

based on chemically characterized capsular polysac-

Isolated exopolysaccharide in liposomes afforded

charides has been described for staphylococci. Serotyp-

sheep protection from infection.

ing of bovine isolates for capsular polysaccharides 5 and

Studies performed in Australia found vaccination

8 typically found 50 to 70 per cent of isolates possessed

with live bacteria protected the gland from infection.

one of these microcapsules, though in some countries

The protection afforded by vaccination with live bacte-

non-typeable isolates predominated.

ria is due to the generation of specific IgG2 which is

Staphylococcus aureus produces around 30 extracel-

cytophilic for PMN. The use of live bacteria has signif-

lular proteins, some of which have been shown to be

icant disadvantages and consequently bacteria grown in

virulence factors (Sutra & Poutrel, 1994). Alpha-toxin
a medium containing milk whey and then killed have
is believed to be responsible for much of the tissue
been used. Killed bacteria stimulate IgG2 when admin-
damage seen in staphylococcal mastitis and beta-toxin
istered with the adjuvant dextran sulphate. In normal
has been shown to cause inflammation of the gland.
glands there is a 24 hour lapse between entry of
Following experimental infection ulcerative and erosive
bacteria into the gland and the accumulation of 500 000
lesions in the epithelial layer are evident throughout
PMNs per millilitre and this delay allows infection to
the ductal system. Toxins can also damage PMNs whose
become established. Vaccination can decrease this lag
role in defence against *Staph. aureus* became apparent period to 6 hours and
PMNs may enter the gland, car-when anti-bovine leukocyte serum converted a
chronic
rying IgG2 on their membrane.
staphylococcal mastitis to an acute gangrenous type.
Australian workers developed a vaccine containing
Neutralizing antibodies can be induced through vacci-
killed bacteria, alpha-toxoid and beta-toxoid, emulsi-

nation and these lessen the damage to secretory tissue. The surface of staphylococci contains proteins which experiments have been performed to establish the efficacy of this Australian vaccine (Watson et al. , 1996). Attachment to ductal epithelial cells is an important step in colonization and is thought to be mediated by fibronectin binding protein. Specific anti-milk yield was lower in the vaccinates. Heifers which body has been shown to prevent this adherence. During received the vaccine had lower SCC and greater milk production after challenge, compared to controls. A tissue components to which the organism can bind in a field trial conducted on seven herds and over 1800 cows specific manner.

found 45 clinical cases of Staph. aureus mastitis in vaccinates and 67 cases in controls. This difference was not

described for their ability to cause food poisoning or statistically significant. One of the herds had a high incidence of toxic shock syndrome in humans. There is variation in the prevalence of *Staph. aureus* mastitis and in this herd clinical between countries in the proportion of bovine *Staph.* and subclinical cases of mastitis were significantly lower in *aureus* isolates secreting these molecules. Recent research suggests they may have a role to play in *aureus* isolates compared to controls. A better understanding of *Staph. aureus* suggests that staphylococcal mastitis by causing immunosuppression. protection depends on antibodies being generated to Enterotoxins may activate CD8⁺ T lymphocytes and several virulence factors. Vaccines should generate anti-decrease the response of CD4⁺ T lymphocytes to antibodies specific for matrix binding proteins to prevent staphylococcal antigens. As superantigens, enterotoxins adherence and to the surface polysaccharides to may also contribute to inflammation by activating cells promote opsonization. Neutralizing antibodies specific in a non-specific manner and causing the release of for alpha- and beta-toxins would lessen clinical damage

inflammatory cytokines. Bovine mononuclear cells produce and protect the cellular defences of the gland. The cells proliferate in response to enterotoxin and secrete IL-2, results of trials with experimental vaccines based on gamma-interferon and TNF. It is difficult to stimulate these three components will clarify whether vaccination neutralizing antibodies to these molecules.

has a role in preventing staphylococcal mastitis. When In a field trial with a staphylococcal bacterin–toxoid attenuated strains are administered by the intramammary preparation, fewer new infections, less culling and lower somatic cell counts were evident in vaccinated cows as compared to controls. The capacity of such strains to stimulate protective immunity is being examined.

10 billion CFU of each of two strains of *Staph. aureus*, whose exopolysaccharide cross-reacted with most field

Streptococcus spp.

isolates. The toxoid component contained alpha- and beta-hemolysins. A second trial found fewer clinical

There are no cross-reactive protective epitopes shared and subclinical cases in vaccinates but this difference by the streptococci which cause mastitis.

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Streptococcus agalactiae (see also Chapter 23)

found in the alveolar exudate with just a small number associated with damaged epithelial cells. After infection

Streptococcus agalactiae (Strep. agalactiae) is a Group B

there are many neutrophils in gland secretions but few

streptococcus and is an obligate parasite of the bovine

contain Strep. uberis. The role of this cell in defence mammary gland where it colonizes the milk ducts. It

against Strep. uberis is unclear because isolates poscan be eliminated from a herd through culture and ther-

sessing a hyaluronic acid capsule resist uptake by neu-

apeutic means. This easy step should take precedence

trophils and capsular material can damage PMNs.

over the task of generating protective antibody. The

Macrophages may be the key defence cell since they

group-specific polysaccharide is common to all strains

can ingest isolates which have been opsonized by IgG

and Strep. agalactiae can be divided into five distinct 1 and IgG serotypes based on the presence of type-specific surface 2.

Quarters previously infected with Strep. uberis are antigens. A protein antigen designated X is frequently significantly protected from subsequent infection by the associated with bovine mastitis isolates. Attachment to same bacteria. Immunization with killed bacteria via epithelia is a prerequisite for colonization and surface the intramammary or subcutaneous route offered pro-lipoteichoic acid may aid in this attachment. Strep. tection from challenge with the immunizing strain and agalactiae produces CAMP factor which is lethal for a strong inflammatory reaction was present in the mice and rabbits.

gland. The bacterin did not afford protection from The bactericidal activity of neutrophils for Strep. infection with wild-type strains. When live bacteria agalactiae is dependent on antibody and complement. were administered subcutaneously and bacterial

Most opsonizing antibody is directed to the weakly extracts by the intramammary route, a degree of pro-immunogenic, type-specific polysaccharide and such tection was again noted. This protection did not extend antibody is a more efficient opsonin when it binds com- to strains not included in the vaccine. As there was only plement. The sera of cows contain low levels of natural a mild inflammation in the gland it was proposed that opsonin. Mackie et al. (1986) found heifers became live vaccine affected the ability of the organism to repli- hyperimmune following systemic vaccination by the cate and a neutrophil response was not critical for pro- subcutaneous and intravenous routes. Despite high tection from infection (Leigh, 1999).

titres there was little real difference between the hyper- *Streptococcus uberis* is unable to produce certain immune and non-vaccinated heifers following chal- amino acids and must find them in its environment.

lenge with around 40 million bacteria and it was

Studies using chemically defined media found all strains concluded that locally produced antibody may be

require eight amino acids for growth, with some required for protection. Antibodies to protein X
ing 13 amino acids. The organism is incapable of
increase the ingestion of bacteria by PMNs but do not
hydrolysing proteins directly, but is able to convert
increase the number of bacteria killed by PMN.
bovine plasminogen to plasmin, a protease which can
break down casein into peptides. The plasminogen activator of *Strep. uberis* is a protein of 30 kD; it is secreted *Streptococcus uberis* (see also Chapter 23)
by almost all isolates and specific neutralizing antibodies are stimulated following immunization.
serologically, biochemically and by molecular biological
In an experimental challenge trial, vaccination with
techniques. It does not fit into the Lancefield classification
plasminogen activator gave a significant degree of
tion with 25 per cent of strains typing as group E and
protection from clinical disease and greatly reduced
smaller numbers belonging to other serogroups. A
bacterial numbers in milk (Leigh, 1999). Both of these
similar percentage of strains is CAMP positive. Sensi-

protection indices were achieved in the presence of a
tive molecular typing methods indicate most isolates
mild inflammatory response and protection was corre-
are genetically dissimilar. This organism can be recov-
lated with the titre of neutralizing antibody specific for
ered from several body sites of the cow and from
plasminogen activator. The studies conducted with
manure and soil. It has become a significant cause of
Strep. uberis could be highly relevant to other mastitis and as an
environmental pathogen is less
pathogens and have underlined the importance of in
affected by conventional control methods.

vivo rather than *in vitro* studies.

The susceptibility of the dry mammary gland to
infection with *Strep. uberis* increases with time and in *Streptococcus*
dysgalactiae (see also Chapter 23)

the second half of the dry period all quarters are
susceptible. This bacterium binds low amounts of fi-

Streptococcus dysgalactiae (*Strep. dysgalactiae*) is a bronectin, fibrinogen and
type II collagen. Adherence

group C streptococcus and possesses a hyaluronic acid

to mammary epithelial cells has been described *in vitro*, capsule. The group C
antigen is a carbohydrate and

but in experimental infections most bacteria were
three type-specific antigens have been identified. This

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bacterium is able to cause mastitis on its own and is one
entation to CD4⁺ T cells and to B cells. These CD4⁺ cells
of a number of pathogens involved in the summer
release cytokines that stimulate the B cells to divide and
mastitis complex. Strains of highest infectivity are those
produce antibodies. A small proportion of B cells
which adhere well to mammary epithelial cells. Strep.

become memory cells and if subsequently presented
dysgalactiae binds well to fibronectin, moderately to fib-with their specific
antigen they will proliferate rapidly

rinogen and poorly to collagen. Protein G is present in
and produce large amounts of antibody. Macrophages,
the cell walls of some Strep. dysgalactiae and can bind T cells and B cells are
present in the mammary gland

bovine IgG. Such non-specific binding of immunoglob-
and administration of antigen to the dry gland stimu-
ulin may protect the bacteria from gland defences.

lates local synthesis of IgA and IgG1. Such production

Cows develop protective antibody following immu-

persists well into lactation and is associated with the nization and hyperimmunized cows showed a degree of development of plasma cells in periductal connective resistance to challenge exposure of the homologous tissue and interalveolar areas. The response is much less strain.

when antigen is administered to the lactating gland. Intraperitoneal administration of antigen followed by local infusion recruits cells from systemic lymphoid

Mucosal immune system

tissues which have been primed by intraperitoneal immunization. Vaccination in the region of the draining lymph node of the mammary gland stimulates serum antibody and promotes production of local antibody. The concept of a common mucosal immune system was proposed following the observation that subsequent to intestinal exposure to antigen, specific immunity was evident at distant mucosal sites. Immunocompetent J5 coliform mastitis was efficacious when administered cells (specific lymphocytes) can migrate from Peyer's

subcutaneously or in the area of the supramammary patch or mesenteric lymph nodes to distant sites, lymph node. The administration of antigen in the region become resident and respond to sensitizing antigen.

of the draining lymph node accompanied by local infusion. This does not seem to be true for ruminants since there is no selective migration of lymphocytes between the lymph node and the mammary gland. The administration of antigen in the region of the draining lymph node accompanied by local infusion of antibodies in lacteal secretions.

mammary gland and the gut of the cow. There are two major groups of recirculating lymphocytes in the cow: The time of vaccination is another important factor since most infections occur at drying off, during the dry period and in the periparturient period. Almost 100 per cent of clinical mastitis occurs during lactation, with 60 per cent taking place in the first 40 days. Vaccination whereas labelled ileac mesenteric lymphocytes were should be designed to offer maximum protection at

recovered from intestinal mesenteric lymph nodes.

these times. If systemic immunization is carried out at Local administration of antigen to the mammary gland drying off, a booster given shortly before parturition by would stimulate lymphocytes in the lamina propria to the intramammary route allows the gland to give a expand clonally and some of these cells would traffic to heightened and prolonged response through the next the supramammary lymph node.

lactation (Watson & Lascelles, 1975).

Two components of a successful vaccine are the antigen and the adjuvant. As knowledge of the protec-

Generation of immune response

tive antigens of mastitis causing bacteria has grown, it is now appreciated the amount of antigen in a vaccine

Immune responses designed to protect the bovine dose is critical and that the numbers of bacteria used in mammary gland from infection can be generated by early studies were too low. Growth of Gram-positive administration of antigen by various routes. Systemic cocci on conventional laboratory media can result in

immunization performed by the intramuscular, subcutaneous or intravenous route generates serum antibody. are expressed during in vivo replication. Adjuvants When mammary epithelium becomes inflamed size-are substances which act in a non-specific manner to able amounts of circulating antibody pass into the augment the immune response to an antigen. Mineral lacteal secretions from the serum by transudation. Anti-oil, saponin and aluminium hydroxide are examples of body has its greatest effect when present in lacteal adjuvants employed in veterinary vaccines. They can secretions before infection is established. Direct injection affect the magnitude, duration and isotypic composition of antigens into the udder stimulates both a local of the immune response. The mechanism of action of and a systemic response.

adjuvants is not fully understood but includes slow The primary course of events for humoral immunity release of antigen from a depot, alteration of lympho-involves antigen processing by macrophages and pres-

cyte circulation and release of cytokines.

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Limitations of vaccination

To maintain levels of antibody in this volume of secretion requires an enormous synthetic rate. Immunization does not confer absolute protection and at best will

The scientific literature contains numerous reports documenting good husbandry and other mastitis control procedures. Vaccines for coliform mastitis will continue intramammary challenge with a pathogen. There are to be used widely while products to prevent Staph.

fewer reports of protective products. These studies

aureus and Strep. uberis should become available in the account for the two opposing views concerning the role

near future. For other mammary pathogens there is

of vaccination in mastitis control programmes. Chal-

little prospect of vaccines becoming available.

lenge studies often have consisted of placing thousands

of bacteria into the teat sinus, which is not an accurate

reflection of natural infections in which only a few

References

organisms will traverse the teat duct and reach the cistern to multiply. A better method to establish the efficacy of a vaccine is to perform field trials in herds where

Bramley, A.J. (1976) Variations in the susceptibility of lactating and non-lactating bovine udders to infections when

natural exposure to the pathogen will occur, with one

infused with *Escherichia coli*. *Journal of Dairy Research*, **43**, half of the herd receiving vaccine and the remaining

205–11.

half receiving placebo. The evaluation of infection, clin-

Butler, J.E. (1986) Biochemistry and biology of ruminant

ical inflammation, persistence of leukocytosis, bacterial

immunoglobulins. *Progress in Veterinary Microbiology and*

shedding and the effect on milk production will help

Immunology, **2**, 1–53.

determine vaccine efficacy.

Colditz, I.G. & Watson, D.L. (1985) Immunophysiological

The dairy cow undergoes periods of severe stress in

basis for vaccinating ruminants against mastitis. Australian each lactation cycle and immune dysfunction occurs at

Veterinary Journal, **62**, 145–52.

certain times. Immunosuppression can occur during

Gonzalez, R.N., Cullor, J.S., Jasper, D.E., Farver, T.B., Bushnell, pregnancy, lactation and parturition. Much interest has

R.B. & Oliver, M.N. (1989) Prevention of clinical col-

been directed to the four-day period prior to and sub-

iform mastitis in dairy cattle by a mutant *Escherichia coli* vaccine. *Canadian Journal of Veterinary Research*, **53**, sequent to parturition, the so-called periparturient

301–305.

immunosuppression. Secretion taken just after drying

Hill, A.W.

(1979) The pathogenesis of experimental

off and at parturition has an inhibitory effect on blas-

Escherichia coli mastitis in newly calved dairy cows.

togenic responses to mitogens. Proliferative responses

Research in Veterinary Science, **26**, 97–101.

to mitogens by blood and milk lymphocytes are low for

Hill, A.W. (1991) Vaccination of cows with rough *Escherichia* one week before parturition and lowest on the day

coli mutants fails to protect against experimental intra-before calving (Kehrli et al. , 1989). This may relate to mammary bacterial challenge. *Veterinary Research Com-the high incidence of clinical mastitis and intramam-*

munications, **15**, 7–16.

mary infection which occur at calving and up to 30 days

Hogan, J.S., Smith, K.L., Todhunter, D.A. & Schoenberger, P.S.

past calving. Certain indices of neutrophil function

(1992a) Field trial to determine efficacy of an *Escherichia* increased two weeks before parturition and decreased

coli J5 mastitis vaccine. *Journal of Dairy Science*, **75**, 78–84.

dramatically by the first week after parturition.

Hogan, J.S., Weiss, W.P., Todhunter, D.A., Smith, K.L. &

Because the concentration of PMNs is used to assess

Schoenberger, P.S. (1992b) Efficacy of an *Escherichia coli* J5

milk quality and a major defence component of the

mastitis vaccine in an experimental challenge trial. *Journal* gland involves a leukocytosis with specific antibody,

of *Dairy Science*, **75**, 415–22.

some authors argue that protection afforded by such a

Kehrli, M.E., Nonnecke, B.J. & Roth, J.A. (1989) Alterations leukocytosis is, by definition, mastitis. Such a protective

in bovine lymphocyte function during the periparturient

response can be interpreted in this manner. There

period. *American Journal of Veterinary Research*, **50**, 215–20.

is mounting evidence indicating that for certain

Leigh, J.A. (1999) *Streptococcus uberis*, a permanent barrier pathogens, appropriate immunization strategies can

to the control of bovine mastitis? *The Veterinary Journal*, reduce the number of clinical cases, reduce the number

157, 225–38.

of new infections, improve the response to therapy and

Linton, A.H. & Robinson, T.C. (1984) Studies on the association of *Escherichia coli* with bovine mastitis. *British Veterinary Journal*, 40, 368–73.

nary Journal, 40, 368–73.

a transient leukocytosis, it is likely farmers will accept

Mackie, D.P., Meneely, P.J., Pollack, D.A. & Logan, E.F.

the benefits.

(1986) The loss of opsonic activity of bovine milk whey fol-

The mammary gland of the cow has limited defensive

lowing depletion of IgA. *Veterinary Immunology and*

capabilities with regard to PMN function and level of

Immunopathology 11, 193–8.

opsonins. In North America, good dairy cows yield on

Mellenberger, R.W. (1977) Vaccination against mastitis.

average 30 litres of milk for each day of their lactation.

Journal of Dairy Science, 60, 1016–21.

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Nordhaug, M.L., Nesse, L.L., Norcross, N.L. & Gudding, R.

Tyler, J.W., Cullor, J.S., Osburn, B.I., Bushnell, R.B. & Fenwick, (1994) A field trial with an experimental vaccine against

B.W. (1988) Relationship between serologic recognition of

Staphylococcus aureus mastitis in cattle. 1. Clinical param-*Escherichia coli* O111, B4 (J5) and clinical coliform mastitis. *Journal of Dairy Science*, **77**, 1267–75.

in cattle. *American Journal of Veterinary Research*, **49**, Outteridge, P.M. & Lee, C.S. (1988) The defence mechanisms 1950–4.

of the mammary gland of ruminants. *Progress in Veterinary*

Watson, D.L. & Lascelles, A.K. (1975) The influence of sys-Microbiology and Immunology, **2**, 1–53.

temic immunization during mammary involution on subse-

Sorensen, G.H. (1972) Sommer mastitis. Den mulige

quent antibody production in the mammary gland. *Research*

beskyttende virkning af to forskellige vacciner overfor

in Veterinary Science, **18**, 182–5.

eksperimentelle infektioner. *Nordisk Veterinar Medecin*, **24**, Watson, D.L., McColl, M.L. & Davies, H.I. (1996) Field trial 259–71.

of a staphylococcal mastitis vaccine in dairy herds, clinical, Sutra, L. & Poutrel, B. (1994) Virulence factors involved in the subclinical and microbiological assessments. *Australian*

pathogenesis of bovine intramammary infections due to

Veterinary Journal, **74**, 447–50.

Staphylococcus aureus. *Journal of Medical Microbiology*, **40**, 79–89.

Chapter 30

Antimicrobial Therapy of Mastitis

D.J. O'Rourke and J.D. Baggot

General considerations

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Mycoplasma sp. (see Chapters 23 and 24). Differences

Antimicrobial resistance

391

in climate, management and national or regional regu-

Pharmacology of the mammary gland

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latory efforts result in patterns of disease that vary

Intramammary preparations

393

markedly. The arid south-western regions of North

Parenteral preparations

395

America have repeatedly identified prevalences of

Selection of antimicrobial agent

396

intramammary Mycoplasma sp. infection exceeding

Response to therapy

397

those observed in other parts of the world (Jasper et al., Therapy of mastitis

397

During lactation

397

1966; Jasper, 1980). In a survey of 50 California dairy

During the dry period

399

herds (23 138 cows) potential mastitis pathogens were

Benefits of antimicrobial therapy for mastitis

402

isolated from 22 per cent of cows (Gonzalez et al., 1988).

Improved milk quality

402

Staphylococcus aureus was isolated from all 50 herds, Increased milk production

402

Strep. agalactiae from 47 herds and Mycoplasma sp.

Effective microbiological cures, eliminating reservoirs

from 24 herds. The prevalence of mastitis pathogens

of infection

403

(per cent cows) was *Staph. aureus* (9.1 per cent), *Strep.*

Costs associated with antimicrobial therapy

403

agalactiae (9.3 per cent), *Mycoplasma* sp. (0.9 per cent), Drug and veterinary costs

403

coliform bacteria (1.2 per cent) and other streptococci

Discarded milk

403

(0.9 per cent).

Slaughter withdrawal periods

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Laboratory testing is an integral part of treatment

Risks of new infections/altered host defences

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and control programmes and identification of the causal

agents will have a direct bearing on subsequent therapy

decisions. Several handbooks (International Dairy

General considerations

Federation, 1981; National Mastitis Council, 1999) and

articles (Higgs & Bramley, 1981; Buswell, 1995) are

Mastitis is the single most common disease of the adult available to assist in the identification of mastitis dairy cow, accounting for 30 per cent of all morbidity pathogens.

(Gardner et al., 1990) and 38 per cent of health costs (Kossaibati & Esslemont, 1997). Additionally, in herds

where the average culling rate was 23.8 per cent, mas-

Antimicrobial resistance

titis was cited as the reason for culling in 10.1 per cent of cases and was the most common cause of death (8.9

Although useful as a guideline for antimicrobial selection, antibiotic sensitivity testing does not guarantee the achieve expected lactation yield as a consequence of efficacy of an antibiotic. Generally, the major streptococcal pathogens of the bovine mammary gland are

Schultz et al. , 1999) and a reduction in milk yield caused sensitive (>90 per cent) to penicillin, ampicillin and

by subclinical mastitis are responsible for even greater erythromycin (Prescott & Baggot, 1988). Actual cure

economic losses (Hortet & Seegers, 1998; Hortet et al., rates vary with clinical

and lactation status.

1999). Efficacious, economical treatment of mastitis

There appears to have been no significant increase in

is therefore an important component of livestock

bacterial resistance of mastitis-causing bacteria in the

medicine.

UK over the past 25 years. Craven et al. (1986) found a The most frequent causes of mastitis are Staphylo-high incidence (69.8 per cent) of b-lactamase produc-

coccus aureus, Streptococcus agalactiae, Strep. uberis, tion which was similar to that (66.1 per cent) reported

Strep. dysgalactiae, Escherichia coli, Enterobacter sp., by Jones and Heath (1985). These studies indicated

Pseudomonas sp., Klebsiella sp., Arcanobacterium little change from the 70.6 per cent of penicillin-

(Actinomyces;

Corynebacterium) pyogenes

and

resistant strains reported by Wilson (1961). Recent

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surveys have indicated little change (Buswell, pers.

antimicrobial agent to which the causative pathogenic

comm.). It should also be noted that no staphylococci micro-organisms are susceptible at the drug concentration of bovine origin that are resistant to cloxacillin have been identified (Jones, 2000), despite its widespread use in both lactating and dry-cow intramammary preparation in conjunction with systemic therapy is highly desirable, but syringes for over 30 years.

the effectiveness of an infused antimicrobial is limited

Sensitivity patterns for the two most common Gram-

negative intramammary pathogens, *E. coli* and *Klebsiella* sp., are reported in Table 30.1 and may be used to **Table 30.1**

Antibiotic sensitivities of selected Gram-negative

guide the choice of antibiotic when treating peracute

bacteria isolated from cattle with clinical mastitis (reported as per mastitis (Mackie et al., 1988; Prescott & Baggot, 1988).

cent of isolates sensitive).

One should be cautioned that sensitivity patterns will vary locally according to antibiotic use. Historical sus-

Antibiotic

Prescott & Baggot

Mackie

ceptibility information drawn from individual farms will

(1988)^a

et al. (1988)

likely prove more useful. Table 30.2 reports in vitro sensitivity (Mackie et al., 1988) and in vivo cure rates (Le

E. coli

Klebsiella

Coliforms

Loudec, 1978) with respect to Staph. aureus for several Amoxycillin

—

—

51–65

commonly used antimicrobial agents. It should be

Ampicillin

35

0

3–41

noted that the percentage of sensitive isolates consis-

Cephalothin

60–73

82–93

—

tently exceeds cure rates. The absence of in vitro activ-Erythromycin

39

25

—

ity indicates bacterial resistance while sensitivity infers,

Framycetin

—

—

83–97

but does not guarantee, clinical efficacy.

Furazolidone

92–98

25–92

88–99

Gentamicin

99–100

100

—

Lincomycin

29

17

—

Pharmacology of the mammary gland

Neomycin

56–70

33–90

19–93

Penicillin

0

0

—

Antimicrobial therapy is applied in the treatment of

Polymyxin

100

100

—

Spectinomycin

—

—

30–59

clinical mastitis during lactation and in treating sub-

Streptomycin

73

77

49–61

clinical mastitis at the end of lactation, while the imple-

Tetracycline

23

42–54

54–88b

mentation of preventive measures is essential for

Trimeth-sulfa

81

100

62–95

decreasing the incidence of infection in the dairy herd.

Clinical mastitis is often treated systemically which

a Compiled from several sources.

entails the repeated parenteral administration of an

b Oxytetracycline.

Table 30.2

Antibiotic sensitivities and bacteriological cure rates following treatment for Staphylococcus aureus after intramammary treatment with various antibiotics (adapted from Mackie et al. (1988) & Le Loudec (1978).

Antibiotic

% sensitive

Staph. aureus

No. of reports

Mean % cure

Range

Penicillin

19–62

32

0–87

12

Cloxacillin

100

41

21–84

14

Cephalosporins

100

72

1

Neomycin

72–98

27

25–36

2

Tetracycline

84–92

54

17–96

8

Tylosin

55

48–59

1

Erythromycin

72–98

63

51–76

2

Spiramycin

70

45–82

2

Rifamycin

66

65–66

2

Penicillin-streptomycin

39

21–78

2

Penicillin-neomycin

96

95–100

2

Ampicillin-cloxacillin

27

27–29

1

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by decreased ability of the drug to ascend partially

respectively; weak organic bases, apart from aminoglycosides, streptomycin and polymyxin B (drugs with infected quarter.

low solubility in lipid), attain milk ultrafiltrate-to-plasma ultrafiltrate equilibrium concentration ratios greater than 1 (Baggot, 2000). The high concentration functional rather than an anatomical barrier, mainly by ratios attained by lipophilic organic bases (macrolides, lincosamides and trimethoprim) are attributed to the ion-trapping effect in acidic milk. Enrofloxacin passively diffuse through the predominately lipid barrier (Baggot, 1977). At a moderate level of N-de-ethylation (a microsomal-mediated oxidative reaction) in the liver, would be expected to attain

lating through the mammary gland to the volume of concentrations in milk that would be effective against milk produced has been estimated to be 670:1; this provides ample opportunity for drugs to passively diffuse Gram-negative aerobic bacteria,

in particular

Escherichia coli.

from the general circulation into milk. The rate of transfer is directly proportional to the concentration gradient within the range 6.9 to 7.2. As a consequence the ion-trapping effect on lipophilic organic bases is reduced

In the presence of mastitis, the pH of milk increases

solubility of the drug. The milk-to-plasma equilibrium

while the concentrations attained by weak organic acids

concentration ratio of total (non-ionized plus ionized)

are somewhat increased. The inflammatory reaction in

drug is determined by (i) the degree of ionization of the

udder tissues enhances the passage of penicillins into

drug, which is pK_a/pH -dependent, in blood and milk, (ii)

milk. The increased pH of milk does not affect the

the charge on the ionized moiety and (iii) the extent of concentrations attained by amphoteric drugs (fluoro-binding of plasma proteins and milk macromolecules. It quinolones, tetracyclines, rifampicin), but antimicrobial has been shown that only the lipid-soluble, non-ionized activity of these drugs is lower in milk than in extracellular moiety of a weak organic acid or base that is free (not bound to proteins) in the plasma can diffuse through the cellular barriers and enter the milk (Rasmussen, 1966). In normal lactating cows (milk pH range

Intramammary preparations (Table 30.3)

6.5–6.8), weak organic acids attain milk ultrafiltrate-to-plasma ultrafiltrate equilibrium concentration ratios

Intramammary preparations are used, often in conjunction with parenteral preparations (depending on drug molecules with moderate and high lipid solubility, severity of infection), to treat clinical mastitis and at attain equilibrium concentration ratios of 0.75 and ca 1, the end of lactation to treat subclinical infection and

Table 30.3

Distribution of antibiotics throughout the udder after intramammary administration (adapted from Ziv, 1980).

Good

Moderate

Poor

Amoxycillin

Penicillin G

Bacitracin

Ampicillin

Cloxacillin

(Dihydro)streptomycin

Cephalexin

Cephalonium

Gentamicin

Erythromycin

Cephapirin

Neomycin

Novobiocin

Cephacetrile

Polymyxins

Penethamate

Cefquinome

Framycetin

Quinolones

Tetracyclines

Spiramycin

Other sulphonamides

Sulphanilamide

Tylosin

Trimethoprim

Rifamycin

Amoxycillin-clavulanic acid

Lincomycin

Pirlimycin

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*prevent the establishment of new infections during the
ping of the infected quarter(s) is recommended in cows
dry period. The various preparations differ in nature,
with clinical mastitis, it follows that only quick-release
content and release pattern of the active ingredient(s)
intramammary preparations are suitable for use in lac-*

and in the length of the withdrawal periods (slaughtering cows.

and milk withholding) but all preparations must be rea-

Intramammary preparations for use in lactating cows

sonably non-irritating to the udder. Intramammary

should contain a readily available form, usually water-

preparations are formulated either to provide quick

soluble salt, of an antimicrobial agent with a low degree

release of the antimicrobial agent or slow release of the

of binding to milk and mammary tissue proteins. The

antimicrobial over an extended period. The former are

vehicle used and viscosity of the formulation should

used mainly in lactating cows while the latter are used

allow rapid release of the drug and ensure that effective

at the end of lactation (after the last milking) and

concentrations are maintained throughout the recom-

in non-lactating cows. While the formulation of an

mended dosage interval. Examples of intramammary

intramammary preparation determines the pattern of

preparations that are formulated as suspensions and

antimicrobial release and may influence the withdrawal

have a recommended dosage interval of 12 hours periods, the chemical nature and physico-chemical include cloxacillin sodium, oxytetracycline hydrochloride, ampicillin sodium–cloxacillin sodium combination govern its uptake and distribution in mammary tissue and trimethoprim–sulfadiazine combination. Cefuroxime sodium and cefoperazone sodium (second- and third-generation cephalosporins, respectively) are formulated as an oily paste and oily suspension, respectively. Drug transfer from treated to untreated quarters of the udder takes place via the bloodstream.

Following intramammary infusion antimicrobials with high lipid solubility (macrolides, trimethoprim, fluroquinolones, rifampicin) are readily taken up by and ration is considered to be usually sufficient for treatment. Quick-release intramammary preparations have

is moderately lipid-soluble and the fraction that is not short withdrawal periods, typically slaughter times of 7 chelated with calcium in milk enters mammary tissue days and milk withholding times of 3.5 days, or either and distributes evenly in a manner that is not subject to may be less depending on the preparation.

the pKa/pH-dependent ion-trapping effect. However, Slow-release intramammary preparations may the antimicrobial effect, which is exerted mainly on contain an antimicrobial agent with a high degree of streptococcal spp. and coagulase-negative staphylo-binding to the secretions and mammary tissue proteins.

cocci, is bacteriostatic. The susceptibility of coagulase-

Either a poorly soluble salt of an antimicrobial agent

positive Staph. aureus, E. coli and Klebsiella spp. to may be used or the formulation of the preparation be

oxytetracycline is variable and unpredictable. The

such that the rate of antimicrobial release is relatively

uptake and distribution of sulfadiazine (pKa 6.4) and

constant, approaching zero order (Baggot & Brown,

sulphamethoxazole (pKa 6.0), which are moderately

1998). The formulation of slow-release preparations of lipid-soluble organic acids, are influenced by the degree determines the antimicrobial concentration–time of ionization in their local environment. Since profile in the mammary gland to a greater extent than sulphonamides are combined with trimethoprim in quick-release preparations. Since only a single dose of intramammary preparations the broad-spectrum anti-a slow-release preparation is infused, the antimicrobial bacterial effect produced is bactericidal. The beta-content is generally higher than in quick-release penicillam antibiotics (penicillins and cephalosporins), preparations. The antimicrobial must remain active (be although they exist mainly in an ionized form in milk, stable) throughout the extended duration in the udder are taken up by mammary tissue and distribute readily and the preparation must not cause irritation. Examples of slow-release intramammary preparations kill bacteria within phagocytic cells (neutrophils and include cloxacillin (as benzathine salt) with aluminium

macrophages). The uptake of hydrophilic antimicrobial monostearate (suspension), ampicillin (as trihydrate)–agents (aminoglycosides and polymixin B) from milk is cloxacillin (as benzathine salt) formulated as an oily low and their distribution is very limited and uneven suspension, procaine benzlypenicillin (oily paste) and because of selective binding to mammary tissue compounds dihydrostreptomycin sulphate–procaine benzylpenicillins. Antimicrobials that avidly bind to mammary cillin (oily paste). The long withdrawal periods for these tissue (polymyxin B, neomycin, streptomycin, spiramycin) preparations preclude their intramammary infusion in ramycin) have long withdrawal periods.

lactating cows.

The frequency of milking and the efficiency of milk–Intramammary administration permits delivery of out greatly influence the concentration of antimicrobial the antibiotic directly to the mammary gland. Penicillins that could be attained in the udder. Since frequent streptomycin derivatives have demonstrated efficacy in

intramammary treatment of *Strep. agalactiae*. Intra-Parenteral preparations with pharmacokinetic properties that meet most of these criteria include procaine single-use syringes for a course of treatment of one, two benzylpenicillin, amoxicillin trihydrate–clavulanate or three syringes at 12- or 24-hour intervals depending on the product. Lactating cow (quick-release) products (potassium combination, trimethoprim–sulfadiazine (or sulphamethoxazole) combination and enrofloxacin. In lactating cow (quick-release) products are generally designed for rapid clearance and, consequently, have shorter milk withholding periods. Anti-racycline hydrochloride (conventional preparation) antibiotics with extensive tissue binding (polymixin B and aminoglycosides) have extended milk withholding periods (Baggot, 2000). Dry-cow formulations, for the are not indicated in the treatment of mastitis. Macrolide

treatment of subclinical mastitis, are designed to antibiotics have good tissue penetrative capacity and produce extended duration of effective drug concentrations. This is achieved by using a higher concentration of the active drug or the benzathine salt of the active drug in oil or a repository vehicle.

ruminal fluid (pH 5.5 to 6.5) where the ion-trapping effect also applies. For antimicrobial therapy with either a parenteral or intramammary preparation, or the concurrent use of tissues and passage into ruminal fluid, high doses are both types of preparation, to be effective an antimicrobial concentration exceeding the MIC for the causative agent is required to attain effective concentrations in milk. In the case of spiramycin, the persistence of tissue residues of pathogenic micro-organism must be maintained at the site of infection for an adequate duration. Provided the

of erythromycin and spiramycin cause tissue irritation appropriate antimicrobial is selected, the intramammary intramuscular injection sites, slow intravenous injection of a slow-release preparation at the end is the preferred route of administration.

of lactation may adequately meet this requirement.

Drugs are transferred from the general circulation to

However, in the case of Staph. aureus infection, the the mammary gland by passive diffusion (Baggot, 1977; administration of a parenteral preparation in conjunction with a slow-release intramammary preparation agents that are not extensively bound to plasma proteins more readily reach the mammary gland (Table strains of the bacterium, increases the effectiveness of 30.4). Because milk is weakly acidic (pH 6.5–6.8), drugs therapy and consequently the recovery rate.

that are weak bases (trimethoprim, macrolides, lincosamides) are preferentially concentrated by ion trapping (Ziv, 1980; Baggot, 2000). Of the antimicrobials

Parenteral preparations

that distribute well in the mammary gland following parenteral administration, only fluoroquinolones have broad-spectrum activity against Gram-negative

Severe mastitis is usually treated systemically, although pathogens. Because of bacterial resistance concerns for intramammary therapy will often be used adjunctively.

humans, the use of this class of antimicrobial in dairy

An ‘ideal’ antimicrobial for systemic therapy of mastitis should have the following properties (adapted from Ziv, 1980):

passage into milk.

- *Low minimum inhibitory concentration (MIC) for the majority of mastitis-causing pathogenic microorganisms.*
 - *High bioavailability following intramuscular* **Table 30.4** *Distribution of antibiotics throughout the udder after injection.*
- parenteral administration (adapted from Ziv, 1980).*
- *Lipid-soluble and predominately non-ionized in the*

blood, and have a low degree of binding to plasma

Good

Moderate

Poor

proteins.

- *A long apparent half-life to ensure that concentra-*

Erythromycin

Amoxycillin

(Dihydro)streptomycin

tions above (preferably several-fold) the MIC are

Spiramycin

Ampicillin

Gentamicin

maintained at the site of infection throughout the

Tylosin

Cephalosporins

Neomycin

Trimethoprim

Penicillin G

Polymyxins

dosage interval (12 or 24 hours).

Baquiloprim

Rifamycin

Spectinomycin

- *Minimal adverse effects in cows.*

Fluoroquinolones

Sulphonamides

- *Short withdrawal periods (milk withholding and*

Tetracyclines

slaughter).

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In the presence of clinical mastitis, the pH of the milk adequate duration in order to achieve a bacteriological increases (6.9–7.2), approaching that of plasma. Under cure. Apart from preparations containing fixed com- these circumstances weak acids (sulphonamides, binations (trimethoprim–sulphonamide, amoxillin– penicillins, amoxycillin–clavulanic acid, cephalosporins, clavulanate), the use of combination preparations rifampin), although typically not concentrated in milk, should generally be avoided.

may reach effective concentrations (Ziv, 1980;

Having made a (tentative) clinical diagnosis of the Durnford, pers. comm.; Baggot, 2000). Lipophilic drugs nature of the mastitis and established the history of of various pharmacological classes will also be concentrated in the affected cow and incidence of infection in milk in spite of the decreased ion-trapping.

in the herd, a pretreatment milk sample for bacterial The site of infection within the udder varies with the culture should be properly (aseptically) collected.

causative pathogenic micro-organisms and, in addition Immediate examination of a milk sample is a particularly useful aid in selecting an antimicrobial for empirical treatment, which should commence at this time.

usually an infection of the milk compartment of the A parenteral and quick-release intramammary preparation, antimicrobial agents that have limited membrane permeability may be used concurrently. The latter should be

brane penetrative capacity and produce a bactericidal infused after stripping milk and other products (cellulose effect, such as penicillins (benzylpenicillin), may be lar debris, pus) from the infected quarter(s). Oxytocin, selected for therapy. Even though coliform mastitis is 5 to 10 units of diluted solution (10 units/ml), can be an infection of the milk compartment, the choice of administered by slow intravenous injection to facilitate the completeness of stripping. The half-life of oxycenrofloxacin or trimethoprim–sulphonamide since the tocin is approximately 2 minutes. In addition to anti-long withdrawal period for aminoglycosides precludes microbial administration, supportive therapy should be their use. *Staphylococcal aureus*, the principal causative provided. micro-organism of subclinical mastitis, may reside in the Results obtained from the microbiology laboratory, intracellular space and within epithelial and phagocytic interpreted in conjunction with the tentative clinical cells (Pyorala, 1995). The treatment of staphylococcal diagnosis and knowledge of the distribution of anti-

mastitis requires the administration (parenteral and microbial agents in the udder, facilitate selection of intramammary) of an antimicrobial agent which can the appropriate drug. Mastitis, with the exception of penetrate epithelial cells and tissues of the udder and summer mastitis, is usually caused by a single bacterial is active against staphylococcal beta-lactamase.

species. When Staph. aureus is the causative pathogenic Amoxicillin–clavulanic acid combination and

micro-organism, it can be rapidly determined whether macrolide antibiotics (erythromycin and spiramycin) the strain isolated produces beta-lactamase (70 per cent meet these requirements. The bacteriostatic effect pro- of strains), although it should be recognized that milk duced by macrolides and their reduced antibacterial induces production of the enzyme. The susceptibility of activity in milk could be disadvantageous.

streptococci can be predicted, but it is necessary to determine quantitative susceptibility, using the broth dilution method (which measures MIC), of coagulase-

Selection of antimicrobial agent

positive staphylococci and enteric bacteria (*E. coli*, *Klebsiella* spp.). Knowledge of the susceptibility (pre-There are several factors to consider when selecting an

dicted or measured in vitro) of the causative bacterial antimicrobial agent, and the preparations (parenteral,

pathogen and of the pharmacokinetic properties of intramammary) to use, for the treatment of mastitis.

the antimicrobial agent of choice provides the infor-

They include the nature and severity of the infection, mation required for calculating the dose and selecting the stage of lactation, the causative pathogenic micro-

the dosage interval for the parenteral preparation.

organism and its susceptibility, the relative cost of the

Dosage calculations are based on MIC which is various antimicrobial preparations, the incidence of assumed to be related to the concentration attained mastitis in the herd and the usage pattern of intra-

at the site of infection in the udder. An intramam-

mammary preparations. Early diagnosis and the

mary preparation containing an antimicrobial that will prompt application of appropriate treatment will reach the site of infection and complement the activity

reduce the extent of mammary tissue damage and will of the antimicrobial in the parenteral preparation increase the rate of recovery and the return of milk should be chosen and administered by intramammary production to or approaching the original level. It is infusion (after stripping the inflamed quarter) at the desirable to select an antimicrobial that produces a recommended dosage interval, which is usually 12 or 24 bactericidal effect and has a narrow spectrum of activity hours, for preparations intended for use in lactating cows and essential that treatment should continue for an cows.

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Response to therapy

infusion mainly in lactating cows.

Amoxycillin

trihydrate–clavulanate potassium and prednisolone are

A combination of methods, applied two to three weeks

combined in a compound intramammary preparation

after the end of treatment, should be used to assess the

(oily suspension) which provides greatly enhanced

effectiveness of antimicrobial therapy. They include activity of amoxycillin against beta-lactamase producing *Staph. aureus* and has short withdrawal periods. collected milk sample, tests based on changes in somatic Steroids (prednisolone) are present in low dose and cell count, protein, enzyme and electrolyte contents of their activity is of short duration. There has been no milk (indication of inflammation) and return to the convincing evidence of depressed antibody production expected level of milk production (Pyorala, 1995). Discrepancy between the expected and obtained response of prednisolone in an intramammary formulation. In to therapy may be attributed to inadequacy of the host defence mechanisms, acquired bacterial resistance, prednisolone is supportable on grounds of cow welfare. altered pharmacokinetic behaviour of the antimicrobial Farmers will claim that the inclusion of the steroid will in the diseased animal or poor distribution in the udder

lead to a reduction in size of the inflamed quarter. Trials with limited access to the site of infection. In the case carried out with an amoxycillin–clavulanic acid formulation of infection caused by *Staph. aureus* bacteria located in lactation containing prednisolone confirm this observation microabscesses, macrophages and fibrotic tissue, the (O'Rourke, 1994) (Fig. 30.1).

bacterial pathogen may be virtually inaccessible to Most compound preparations contain antimicrobial antimicrobial agents or may be in a state of dormancy. agents that are selected on the basis of broadening the The bacteriological cure rate of staphylococcal mastitis spectrum of antimicrobial activity without extending is low, particularly when treatment is applied during lactation the withdrawal period. These compound preparations tation, while the high self-cure rate of coliform mastitis (more than one antibiotic) are of doubtful value and are greatly influences the validity of attributing bacteriological cure to antimicrobial therapy (Sandholm & the concentration achieved by any one antibiotic in

Pyorala, 1995). A reasonably satisfactory response rate such preparations is likely to be lower than the to treatment of staphylococcal mastitis is obtained only optimum required for treatment and could facilitate when appropriate therapy with a parenteral preparation the development of resistance (Egan, 1984). Unless the tion and a slow-release intramammary preparation antimicrobials in a combination preparation act synergistically (Table 30.5) the use of compound preparations should be avoided.

the greater the likelihood of at least a moderately In recent years veterinary practitioners have been favourable response to therapy. The bacteriological debating the concurrent use of an injectable and intracure rate can vary widely.

mammary preparation in the treatment of mastitis.

Therapy of mastitis

25

During lactation

20

Factors to take into account when choosing an intra-mammary preparation include:

15

- *Antibacterial spectrum*

10

- *Cost of the preparation for usual duration of treatment*

5

- *With/without steroid*

0

- *Previous treatment history*

0

2

4

Percentage increase in quarter size

6

24

- *Availability of injectable 'partner' (parenteral*

Time from treatment (hours)

preparation)

- Milk withholding time

Fig. 30.1

Reduction in quarter size, experimentally infected

- Personal preference

with Streptococcus uberis, after treatment with amoxycillin and clavulanic acid formulations with and without prednisolone

Compound preparations containing one or more

(O'Rourke, 1994). 糞 = Lactating low formulation containing

antimicrobial agents and a corticosteroid (hydrocorti-

amoxycillin, clavulanic acid and prednisolone. 糞 = Same formu-

sone or prednisolone) are available for intramammary

lation without prednisolone.

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There have been a number of studies indicating a ben-

Serratia spp. Because of the severity of clinical signs the eficial effect in the treatment of Staph. aureus infections following therapeutic regimen is suggested:

from the concurrent use of an intramammary and an

(1)

Frequent stripping of the affected quarter follow-

injectable (Tables 30.6 and 30.7). However, the com-

ing the administration of oxytocin is probably the

patibility of the injectable and intramammary prepara-

most important, yet most often neglected, components should be taken into account whenever this form of therapy. Evacuation of the udder serves to

of treatment is being considered.
remove both bacterial endotoxins and inflammatory host mediators, thus markedly reducing the
Peracute mastitis
severity of clinical signs.

(2)

Antibacterial therapy: gentamicin, polymyxin B

Toxic mastitis is most commonly caused by the coliform
and potentiated sulphonamides have minimal

organisms E. coli, Klebsiella spp., Enterobacter spp. and inhibitory
concentrations (MIC) and distribution

properties suitable for the treatment of coliform

Table 30.5

Clinically useful antimicrobial drug combinations in
mastitis.

veterinary medicine (adapted from Prescott & Walker, 2000).

(3)

Intravenous fluids.

(4)

Non-steroidal anti-inflammatory drugs.

Indication

Drug combination

Comment

The non-steroidal anti-inflammatory drugs have a

Bovine Staph.

Penicillin–

Synergistic

more selective action than glucocorticoids in that they

aureus mastitis

streptomycin

combination

directly inhibit cyclo-oxygenase, although isoforms of

the enzyme are inhibited to a varying degree depend-

Amoxycillin–

Potentiated

ing on the drug. The consequence of cyclo-oxygenase

clavulanic acid

combination

inhibition is that the synthesis of prostaglandins

Table 30.6

*Bacteriological cure rates for subclinical Staphylococcus aureus infections at 21 days post treatment with amoxycillin and penicillin G (Owens et al., 1988).
Reproduced with permission of the American Dairy Science Association.*

Route**Regime****Bacteriological****cure rate (%)**

Intramammary

62.5 mg amoxycillin

25

(n = 40)

six times @ 12-hour

intervals

Intramammary +

62.5 mg amoxycillin

51.4

injection (n = 35)

six times @ 12-hour intervals

+9 000 000 iu procaine

penicillin daily for 3 days

Table 30.7

Bacteriological cure rates for chronic Staphylococcus aureus infections at 20 days post treatment with spiramycin (Ziv, 1980).

Route

Regime

Bacteriological cure rate

(%)

Injection (n = 18)

10 g spiramycin (IM) twice

16

@ 72-hour intervals

Intramammary (n = 40)

500 mg spiramycin three

40

times @ 24-hour intervals

Intramammary +

10 g spiramycin (IM) twice

68

injection (n = 35)

@ 72-hour intervals +

500 mg spiramycin three

times @ 24-hour intervals

involved in central pyresis and pain perception as well as subsequent lactation when compared with control cows as tissue inflammation is inhibited. Unlike glucocorticoids that received no therapy.

coids, the non-steroidal anti-inflammatory drugs do not

The efficacy of dry-cow therapy for subclinical

produce immunosuppression. The rate of elimination

mastitis is not invariably reliable, especially for chronic

(hepatic metabolism) of these drugs in cattle differs

cases of mastitis caused by Staph. aureus (Table 30.9).

widely. The half-life of salicylate is 0.8 hours (3.5 hours

There are, however, results indicating that an improved

when administered orally as aspirin, due to slow disso-

cure rate can be achieved when intramammary treat-

ment is accompanied by systemic treatment (Table

hours), flunixin (8.1 hours) and phenylbutazone (42–66

30.10).

hours).

They distribute to a similar extent, mainly in

extracellular fluid, and enter milk by passive diffusion.

Strategies for dry period treatment

Of these drugs, flunixin meglumine (2.2 mg/kg, administered by intravenous injection at 24-hour dosage

With consistent application of dry-cow therapy (DCT intervals) may have a place in the treatment of acute E.

in conjunction with other mastitis control measures the coli (endotoxin) mastitis. The withdrawal period is

prevalence of contagious mastitis pathogens has slaughter 7 days, milk 12 hours. The antipyretic, anti-

declined to a low level in many herds (Kirk et al., 1994), inflammatory and analgesic effects produced by the

as is evidenced by bulk milk somatic cell counts

drug are largely dependent on the stage of the inflam-

(BMSCC) of <200 000 cells/ml. Some owners of herds

matory process at which treatment is commenced.

with a low prevalence of contagious mastitis pathogens

Early diagnosis of coliform mastitis would increase the

have stopped using dry-cow therapy and others are ques-

beneficial effect produced by flunixin administration.

tioning the practice because of concerns about residues

in the milk (Kirk et al., 1994). Macmillian et al. (1983) indicated that in herds with low cell counts selective dry-

During the dry period

cow therapy may be preferable to whole herd therapy.

Routine dry-cow therapy provides the following

A fact sheet entitled Dry Cow Therapy (Anderson & advantages:

Cote, 1996) from the Ontario Ministry of Agriculture,

Food and Rural Affairs recommends that selective dry-

- Bacteriological cure of a higher proportion of*

cow therapy should be used when the monthly bulk

Staph. aureus infections (Table 30.8).

milk cell count (BMCC) is below 200 000 and the

- No loss of milk production.*

quarter infection rate is <15%. The following cows

- Prevention of new infections (including summer*

should be treated:

mastitis commonly due to Arcanobacterium pyo-

genes), especially in the early dry period.

- Cows with peak somatic cell count (SCC) >*

- Treatment of infections caused by streptococci and*

250 000/ml.

other bacteria.

- Cows which have had a clinical case of mastitis.
- Cows from which a major mastitis pathogen(s) has

In herds with a high prevalence of contagious mastitis been cultured.

tis pathogens, the use of dry-cow therapy has been an efficacious and economically effective method of reduc-

In New Zealand the SAMM (seasonal approach to ing the frequency of intramammary infections (Heald managing mastitis) plan was devised by the Dairy

et al., 1977). Berry et al. (1997) showed that cows treated Research Corporation. It was adopted by the NZ

with dry-cow therapy at the end of lactation produced

Mastitis Advisory Committee and launched in 1993.

179 kg more milk during the first 120 days of the sub-

The implementation of the plan contributed to a

Table 30.8

Bacteriological cure rates (percentages) for selected Gram-positive intramammary infections using cloxacillin (adapted from Griffin et al., 1982).

Bacterium

Lactation

At drying off

Clinical

Subclinical

Streptococcus agalactiae

85

100

99

Staphylococcus aureus

26

52

61

Streptococcus dysgalactiae

90

98

96

Streptococcus uberis

77

82

87

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Table 30.9

The responses of previously untreated or unsuccessfully treated staphylococcal infections to cloxacillin therapy given in lactation or at drying off (adapted from Griffin et al., 1982).

Previous lactation treatments

Lactation therapy

0

1

2

>2

No previous drying-off treatments

% response

46

21

17

12

1 or more previous drying-off treatments

% response

25

15

13

7

Drying-off therapy

0

1

>1

% response

61

46

24

Previous drying-off treatments

% response

60

24

10

Table 30.10

Bacteriological cure rates for chronic subclinical Staphylococcus aureus infections at 15 days postcalving (Johansson et al., 1995). Reproduced with permission of the International Dairy Federation.

Route

Regime

Bacteriological

cure rate (%)

Intramammary (n = 42)

0.17 g penicillin benzathine

57

+0.4 g dihydrostreptomycin

in all 4 quarters

Intramammary +

20 mg/kg

79

injection (n = 38)

benzylpenicillin procaine for

5 days followed by 0.17 g

penicillinbenzatin +0.4 g

dihydrostreptomycin in all

4 quarters

substantial decline in somatic cell count levels in

tion in clinical mastitis during the dry period and a 3-

subsequent years (national average cell count is

fold reduction in infected cows at calving and clinical

178 000/ml). The criteria used for determining herd

mastitis in the first 21 days of lactation when compared

mastitis status and dry-cow therapy are show in Table

to herds where there was no treatment (Table 30.13).

30.11.

Hovi and Roderick (2000) found that the incidence

The effects of three selection strategies for dry-cow

of new infection during the dry period was three times higher in organic herds when compared to conventional antibiotic usage were compared in a trial involving 1044 herds (28.9 cases per 100 cows versus 9.2 cases per 100 cows in 12 herds (Table 30.12) (Browning et al., 1984). The authors point out that most organic dairy cows). Selective cow treatment was identified as the preferred strategy. Blanket treatment resulted in increased antibiotic usage: 15.5 versus 6.4 tubes per infection eliminated without using dry-cow therapy and culling healthy young cows.

with no additional benefit and selective quarter treatment resulted in a higher new infection rate (6.4 versus 3.9 per cent) during the dry period. Whilst we may implement selective dry-cow therapy, the key question is: are these untreated quarters more

susceptible to infection during the dry period? The

Berry et al. (1999) confirmed that this is still the case physiological process whereby the teat canal becomes

today in a study comparing dry-cow therapy versus no closed through the formation of a keratin plug after treatment. Dry-cow therapy resulted in a 10-fold reducing off appears to be a major defence mechanism.

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Table 30.11

Criteria used in the SAMM plan for determining herd mastitis status and dry-cow therapy strategy (Woolford et al., 1995). DCT, dry-cow therapy. Reproduced with permission of the International Dairy Federation.

Criteria

Herd mastitis classification

High

Medium

Low

Average BMSCC, Jan–Mar (000/ml)

>400

150–400

<150

or

% cows >150 000/ml over Jan–Mar

>50%

20–50%

<20%

or

% cows clinical in first month of lactation

>10%

3–10%

<3%

or

15% cows clinical first 3 weeks of last

Yes

No

No

dry period?

or

Late season outbreak of clinical cases?

Yes

No

No

Recommended DCT strategy

Total

Selective

Selective

herd

cow

cow

Table 30.12

Prevalence of infected quarters at drying off and calving plus the new infection rate during the dry period for three different dry-cow strategies (Browning et al., 1984, reproduced with permission of Australian Veterinary Journal). DCT, dry-cow therapy.

Strategy

Drying off

Calving

New infection rate

prevalence

prevalence

during dry period

(%)

(%)

(%)

DCT

13.2

6.8

2.6

Selective cow

13.0

7.9

3.9

Selective quarter

13.1

10.0

6.4_a

a P £ 0.05.

Table 30.13

Prevalence of clinical mastitis during the dry period, infected cows at calving plus clinical mastitis during the first 21 days of lactation (Berry et al., 1999). DCT, dry-cow therapy.

Strategy

Clinical mastitis

Infections at calving

Mastitis in first 21

during the dry

(%)

days of lactation

period (%)

(%)

DCT

0.0

9.6

5.5

No treatment

10.5

32.8

16.4

Williamson et al. (1995), reporting on the observations during the dry period, 97 per cent occurred in teats that

of the dynamics of teat canal closure for a group of 657 had an 'open' canal.

quarters after drying off, noted that 50 per cent of teat

The dilemma is how to protect quarters that are not

canals were classified as 'closed' at 7 days after drying

treated with dry-cow therapy. In New Zealand a non-

off. A further 45 per cent became closed over the fol-

antibiotic preparation, Teatseal™, is available (Orbe-

lowing 50–60 days and 5 per cent remained 'open' at 90

Seal™ in the UK). Teatseal is a dense, viscous material,

days. During this study 83 per cent of all clinical infec-

*which is infused hygienically into the teat as soon as
tions during the dry period occurred within the first 21
possible after the last milking of lactation. It physically
days. Of the 52 clinical infections that were diagnosed
plugs the teat canal immediately after infusion thus*

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Table 30.14

Numbers of new intramammary infections (IMIs)

lishment of infection, by the presence of a physical

*over the dry period and at calving for the four treatment groups barrier from day
one of the dry period, which remains*

(Woolford et al., 1998).

in place until calving.

Experimental group

NC

PC

TS

TS + Ab

Total quarters allocated

528

528

505

505

Benefits of antimicrobial therapy

at drying off

for mastitis

New dry period clinical IMIs

18

2a

1a

2a

Improved milk quality

New IMIs at calving

Streptococcus spp.

50

5a

4a

4a

Subclinical mastitis affects the quality of dairy products.

Staphylococcus aureus

3

0

1

0

Milk from infected cattle is less palatable, has a shorter

Coagulase-negative

6

1

6

2

shelf-life and produces lower yields of processed dairy

staphylococci

products. Consequently, cooperatives, commercial con-

Coliforms

4

4

1

1

cerns and regulatory agencies impose penalties, based

Other organisms

2

2

0

1

Clinical – no growth

2

0

0

0

*on cell count and bacterial counts of bulk milk. The
at culture*

most severe penalty arises with milk that is deemed

Total non-streptococcal

17

7b

8c

4a

unfit for human consumption and discarded (BMCC >

IMIs

*400 000 cells/ml in EU, see Chapter 25). Although treat-
ment of clinical mastitis may not result in a microbio-*

Total new IMIs at calving

67

12a

12a

8a

logical cure, macroscopic milk quality will return to

Total new IMIs

85

14a

13a

10a

normal more quickly following treatment. The more

Overall incidence of new

16.1

2.7a

2.6a

2.0a

rapid return to the production of saleable milk is an

IMI rate (% quarters)

important benefit of treatment.

Superscripts denote significant differences within rows between treatment groups and the negative control.

a P < 0.01; b P ^a 0.05; c P < 0.1

Increased milk production

NC, negative control; PC, positive control (cephalonium); TS, Teatseal; TS + Ab, Teatseal + cloxacillin.

The largest cost associated with mastitis is reduced milk yield as a result of clinical and subclinical infection.

Lucey and Rowlands (1984) found that the mean reducing bacteria from entering the teat. The teat in milk yield where clinical mastitis occurred was canal then becomes closed from day one of the dry 540 kg, the average lactation being 4830 kg. The reduction period and remains closed for the dry period.

tion was greatest when infection occurred before peak

A large-scale study has been carried (Woolford et al., yield, the proportional reduction being higher in higher-1998) utilizing a total of 1200 cows across seven herds.

yielding cows. Where clinical mastitis occurred before

Cows with late lactation SCCs of < 200 000/ml were

peak yield, both peak yield and length of lactation were identified and sampled aseptically to establish the infection

reduced, though the rate of decline from peak yield

tion status of every quarter at drying off. At drying off

was unaltered. Where infection occurred between peak

uninfected quarters were randomly allocated to a treatment

yield and 10 weeks after peak yield, lactation yield and ment. The numbers of new intramammary infections length were both affected. Clinical mastitis occurring (IMIs) during the dry period and at calving are shown after peak yield appeared to have little effect on lactation yield. Results suggested that cows which contract treatments (Teatseal, cephalonium (Cepravin DC), and Teat-mastitis are unlikely to achieve their full milk-yield seal plus 600 mg cloxacillin (prior to infusion of the seal) potential in the following lactation. In a survey of just showed about a 10-fold reduction in new clinical IMIs over 24 000 dairy cows the daily loss in milk yield during ($P < 0.01$) relative to the negative control (no treat-the first 2 weeks after the occurrence of mastitis varied ment). The incidence of new IMI due to *Strep. uberis* at from 1.0 to 2.5 kg. Total loss over the entire lactation calving was 10-fold lower ($P < 0.01$) for prophylactic varied from 110 to 552 kg and depended on parity and treatments compared to the negative control. It is also the time of mastitis occurrence. Regardless of the time interesting to note that the prophylactic treatments

of occurrence during the lactation, mastitis had a long-reduced the incidence of postcalving clinical mastitis by lasting effect on milk yield; cows with mastitis did not approximately 50 per cent in the subsequent lactation reach their premastitis milk yields during the remain-compared to the negative control. This study indicated der of the lactation after the onset of the disease that use of Teatseal protects the gland against estab- (Rajala-Schultz et al. , 1999).

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Table 30.15

(a) Reduction in test day milk yield (kg)* in rela-

not obligate parasites of the mammary gland, are trans-

tion to somatic cell counts (SCCs) of Holstein cows without clini-mitted from cow to cow (Bramley & Dodd, 1984).

cal mastitis, and (b) for SCCs of 200 000 cells/ml the reduction Identification followed by treatment, isolation or

in test day milk yield (kg)* at days 50, 150 and 250 postpartum eradication can markedly reduce the numbers of

(Hortet et al., 1999, reproduced with permission from Elsevier new infections if coupled with appropriate hygienic

Science).

measures.

(a) Reduction in yield at various SCCs

Costs associated with

SCC (cells/ml)

antimicrobial therapy

100 000

200 000

600 000

Drug and veterinary costs

Primiparous 0.30

0.61

1.09

These costs will not be detailed, but should be con-

cows

sidered when evaluating the costs associated with

Calving 2

0.32

0.63

1.13

Calving ≥ 3

0.30

0.60

1.07

treatment.

(b) Reduction in yield at SCC of 200 000 cells/ml

Discarded milk

Day 50

Day 150

Day 250

Available intramammary antibiotic preparations rec-

postpartum

postpartum

postpartum

ommend milk withholding periods (discard times) that
vary from 36 to 120 hours after the last treatment .

Calving 2

0.63

0.92

1.77

Cattle with clinical mastitis are usually treated three

Calving ≥ 3

0.60

1.09

1.85

times at 12 to 24 hour intervals. The antibiotic-contaminated, and hence discarded, milk may total as much

*Reduction in yield was calculated as the deviation from a reference as 420 litres in a high-producing cow. The decision to

value set at 50 000 cells/ml.

undertake treatment cannot be taken lightly. Dry-cow therapy is particularly cost-effective because it does not
Reduction in milk yield is also associated with sub-require milk discarding.

clinical infection. Hortet and Seegers (1998) found in a review of 19 papers that there was an average loss of

Slaughter withdrawal periods

80 kg of milk in primiparous and 120 kg in multiparous cows for each two-fold increase of the geometric mean

Both systemic and local (intramammary) antibiotic of somatic cell count (SCC) > 50 000 cells/ml. The reduction in test day milk yield (kg) associated with SCC of
Treated individuals cannot be marketed for salvage
less than or equal to 600 000 cells/ml in cows without

purposes, regardless of the success of treatment, until clinical mastitis was assessed using monthly cow records after the withdrawal period for the administrated collected for a one year period in 105 Holstein herds in preparation has ended.

France (Hortet et al., 1999). Results indicated that for 100 000, 200 000 and 600 000 cells/ml the reduction in

Risks of new infections/altered

daily milk yield was approximately 0.30 kg, 0.60 kg and

host defences

1.10 kg, respectively (Table 30.15).

Intramammary infusion of antibiotics is far from being an innocuous procedure. The infusion procedure may

Effective microbiological cures,

remove the keratin lining of the streak canal, an impor-

eliminating reservoirs of infection

tant barrier to intramammary infections (see pp. 313,

A very real, but often overlooked, benefit of antimi-

400). Strict hygiene must be practised to prevent acci-

crobial therapy relates more to the overall herd health

dental introduction of micro-organisms at the time of

status than the infected cow. This is because the infected treatment. Only commercially available formulations of cow is a reservoir of transmissible infection. Each known efficacy and guaranteed safety are suitable for infected cow may represent not only an individual with intramammary therapy. Special care must be exercised suboptimal health and production, but also a reservoir when large numbers of cattle are being treated on a of infection. Each infected cow thus poses a threat to single occasion. Explosive outbreaks of peracute and the productive capacity of non-infected cattle. Strep. often fatal mastitis have been known to follow either agalactiae is an obligate parasite of the mammary gland. poor hygiene during infusion of an intramammary *Staphylococcus aureus* and *Mycoplasma* spp., although product or use of a contaminated product.

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References

Herd Management (ed. by A.J. Bramley, F.H. Dodd & T.K. Griffin), pp. 95–109. Technical Bulletin 4, NIRD, Reading.

Hortet, P., Beaudeau, F., Seegers, H. & Fourichon, C. (1999) Anderson, N.G. & Cote, J.F. (1996) Dry Cow Therapy.

Reduction in milk yield associated with somatic cell counts

Factsheet 90-003. Ontario Ministry of Agriculture, Food

up to 600 000 cells/ml in French Holstein cows without clin-

& Rural Affairs.

ical mastitis. Livestock Production Science, 61, 33–42.

Baggot, J.D. (1977) Principles of Drug Disposition in Domes-Hortet, P. & Seegers, H. (1998) Calculated production losses tic Animals: The Basis of Veterinary Clinical Pharmacology.

associated with elevated somatic cell counts in dairy cows:

W.B. Saunders, Philadelphia.

review and critical discussion. Veterinary Research, 6, Baggot, J.D. (2000) Principles of antimicrobial drug bioavail-497–510.

ability and disposition. In Antimicrobial Therapy in Veteri-Hovi, M. & Roderick, S. (2000) Mastitis and mastitis control nary Medicine (ed. by J.F. Prescott, J.D. Baggot & R.D.

strategies in organic milk. Cattle Practice, 8, 259–63.

Walker), 3rd edn, pp. 50–87. Iowa State University Press,

International Dairy Federation (1981) Laboratory Methods

Ames, Iowa.

for Use in Mastitis Work. Document No. 132. International

Baggot, J.D. & Brown, S.A. (1998) Basis for selection of the Dairy Federation, Brussels, Belgium.

dosage form. In Development and Formulation of Veterinary

Jasper, D.E. (1980) Prevalance of Mycoplasma mastitis in Dosage Forms (ed. by

G.E. Hardee & J.D. Baggot), 2nd edn, Western states . Californian Veterinarian, **34**, 24–6.

pp. 7–143. Marcel Dekker, New York.

Jasper, D.E., Jain, N.C. & Brazil, L.H. (1966) *Clinical*

Berry, E.A., Cocks, V. & Hillerton, J.E. (1999) Dry cow treat-and laboratory observations on bovine mastitis due to

ment strategies. In British Mastitis Conference, Stonleigh, Mycoplasma. Journal of the American Veterinary Medical

p. 94.

Association, **148**, 1017–29.

Berry, S.L., Mass, J., Kirk, J.H., Reynolds, J.P., Gardner, I.A. & Johansson, J., Fumke, H. & Emanuelson, U. (1995) Systemic

Ahmadi, A. (1997) Effects of antimicrobial treatment at the

treatment of chronic subclinical *Staphylococcus aureus*

end of lactation on milk yield, somatic cell count, and inci-

mastitis at drying off. In Proceedings of the Third IDF Interdence of clinical mastitis during the subsequent lactation in

national Mastitis Seminar, Tel-Aviv, Israel (ed. by A. Saran a herd with low prevalence of contagious mastitis. Journal

& S. Soback), Book 2, pp. 54–7. National Mastitis Reference of the American Veterinary Medical Association, **211**, 207–11.

Center, Bet-Dagan.

Bramley, A.J. & Dodd, F.H. (1984) Reviews of the progress

Jones, T.O. (2000) Mastitis bacteriology: further problems,

of dairy science: mastitis control – progress and prospects.

pitfalls and comments. Cattle Practice, 8, 333–6.

Journal of Dairy Research, 51, 481–512.

Jones, T.O. & Heath, P.J. (1985) Beta-lactamase production in Browning, J.W., Mein, G.A., Brightling, P. Nicholls, T.J. & Staphylococcus aureus isolated from bovine mastitic milk.

Barton, M. (1984) Strategies for mastitis control: dry cow

Veterinary Record, 117, 340.

therapy and culling. Australian Veterinary Journal, 71, Kirk, J.H., DeGraves, F. & Tyler, J. (1994) Recent progress 179–81.

in treatment and control of mastitis in cattle. Journal

Buswell, J. (1995) Simple mastitis bacteriology for the prac-

of the American Veterinary Medical Association, 204, tice. In Practice, 17, 426–32.

1152–8.

Craven, N., Anderson, J.C. & Jones, T.O. (1986) Antimicrobial Kossaibati, M.A. & Esslemont, R.J. (1997) The costs of

drug susceptibility of Staphylococcus aureus isolated from production diseases in dairy herds in England. Veterinary

bovine mastitis. Veterinary Record, 118, 290–1.

Journal, 154, 41–51.

Egan, J. (1984) Mastitis – a review. Veterinary Update, 1, 5–11.

Le Loudec, C. (1978) Efficacites des antibiotiques contre les

Esslemont, R.J. & Kossaibati, M.A. (1997) Culling in 50 dairy mammites

bovines staphylococci et streptococci.

herds in England. Veterinary Record, 140, 36–9.

Annales de Recherches Vétérinaires, 9, 63.

Gardner, I.A., Hird, D.W., Utterback, W.W., Danaye-Elmi, C.,

Lucey, S. & Rowlands, G.J. (1984) The association between

Heron, B.R., Christiansen, K.H. & Sischo, W.M. (1990)

clinical mastitis and milk yield in dairy cows. Animal Production, mortality, morbidity, case-fatality, and culling rates for

duction, 39, 165–75.

California dairy cattle as evaluated by the National Animal

Mackie, D.P., Logan, E.F., Pollock, D.A. & Rodgers, S.P. (1988) Health Monitoring system, 1986–87. Preventive Veterinary

Antibiotic sensitivity of bovine staphylococcal and coliform

Medicine, 8, 157–70.

mastitis isolates over four years. Veterinary Record, 123, Gonzalez, R.N., Jasper, D.E., Farver, T.B. & Bushnell, R.B.

515–17.

(1988) Prevalence of udder infections and mastitis in 50

Macmillan, K.L., Duirs, G.F. & Duganzich, D.M. (1983) Asso-California dairy herds. Journal of the American Veterinary Association between dry cow therapy, clinical mastitis and

Medical Association, 193, 323–7.

somatic cell count score with milk and fat production in

Griffin, T.K., Dodd, F.H. & Bramley, A.J. (1982) Antibiotic ten New Zealand dairy herds. *Journal of Dairy Science*, **66**, therapy in the control of mastitis. In *Proceedings of the*

259–65.

British Cattle Veterinary Association 1981–82, pp. 137–46.

National Mastitis Council (1999) Laboratory Handbook

Heald, C.W., Jones, G.M., Nickerson, S. & Bibb, T.L. (1977) on Bovine Mastitis. *National Mastitis Council, Washington, Mastitis control by penicillin and novobiocin at drying-off*.

DC.

Canadian Veterinary Journal, **18**, 171–80.

O'Rourke, D. (1994) *Treatment of clinical mastitis in cattle*.

Higgs, T.M. & Bramley, A.J. (1981) *Laboratory techniques for Irish Veterinary News*, **48**, 54–9.

the examination of milk samples. In *Mastitis Control & Owens, W.E., Watts, J.L., Boddie, R.L. & Nickerson, S.C.*

Antimicrobial Therapy of Mastitis • 405

(1988) *Antibiotic treatment of mastitis: comparsion of intra-*

Bovine Udder and Mastitis (ed. by M. Sandholm, T.

mammary and intramammary plus intramuscular therapies.

Honkanen-Buzalski, L. Kaartinen & S. Pyorala), pp. 148–60.

Journal of Dairy Science, **71**, 3143–7.

University of Helsinki, Faculty of Veterinary Medicine,

Prescott, J.F. & Baggot, J.D. (1988) *Bovine mastitis*. In *Antimi-Helsinki*.

crobial Therapy in Veterinary Medicine, pp. 321–31. Black-Williamson, J.H., Woolford, M.W. & Day, A.M. (1995) *The*

well Scientific Publications, Boston.

prophylactic effect of a dry-cow antibiotic against Stre-

Prescott, J.F. & Walker, R.D. (2000) Principles of antimicro-ptococcus uberis. New Zealand Veterinary Journal, 43, 228–

bial drug selection and use. In Antimicrobial Therapy in
34.

Veterinary Medicine (ed. by J.F. Prescott, J.D. Baggot & R.D.

Wilson, C.D. (1961) The treatment of staphylococcal mastitis.

Walker), 3rd edn, pp. 88–104. Iowa State University Press,

Veterinary Record, 73, 1019–24.

Ames, Iowa.

Woolford, M.W., Hook, I.S., Eden, M.T. & Joe, A.T. (1995) The Pyorala, S. (1995) Staphylococcal and streptococcal mastitis.

‘SAMM PLAN’ a seasonal approach to managing mastitis.

In The Bovine Udder and Mastitis (ed. by M. Sandholm, T.

Proceedings of the Third IDF International Mastitis

Honkanen-Buzalski, L. Kaartinen & S. Pyorala), pp. 143–8.

Seminar, Tel-Aviv, Israel (ed. by A. Saran & S. Soback), pp.

University of Helsinki, Faculty of Veterinary Medicine,

59–63. National Mastitis Reference Center, Bet-Dagan.

Helsinski.

Woolford, M.W., Williamson, J.H., Day, A.M. & Copeman,

Rajala-Schultz, P.J., Grohn, Y.T., McCulloch, C.E. & Guard, P.J.A. (1998) *The prophylactic effect of a teat sealer on*

C.L. (1999) Effects of clinical mastitis on milk yield in dairy bovine mastitis during the dry period and the following

cows. Journal of Dairy Science, 82, 1213–20.

lactation. New Zealand Veterinary Journal, 46, 12–19.

Rasmussen, F. (1966) *Studies on the Mammary Excretion and*

Ziv, G. (1980) Drug selection and use in mastitis: systemic vs Absorption of Drugs. Carl Fr Mortensen, Copenhagen.

local therapy. Journal of the American Veterinary Medical

Sandholm, M. & Pyorala, S. (1995) Coliform mastitis. In The Association, 176, 1109–15.

Adult Cattle

Lameness

Chapter 31

Lameness in the Foot

R.W. Blowey

Introduction

409

approach to hoof trimming and the causes and control

Incidence and prevalence

409

of lameness.

Structure of the foot

411

Hoof

411

Corium

413

Incidence and prevalence

Bone and associated structures

413

Weight bearing surfaces, hoof overgrowth and wear

413

The incidence of lameness is defined as the number of

Hoof overgrowth

413

Hoof trimming

415

cases recorded over a given period, usually a year. It is

Lesions causing lameness

417

a longitudinal measure and is often expressed as the

Sole ulcers and white line defects

417

number of cases per 100 cows per annum. A single case

Sole ulcers

419

of lameness is defined as one lesion in one claw. A

Heel and toe ulcers

419

repeat or new case can be a different lesion in the same,

White line defects

419

or another, claw, although it may be a recurrence of the

Causes and control of coriosis (laminitis)

420

same lesion after a period of time. Prevalence is the

Other causes of foot lameness

425

number of cases of lameness present at a single point

Hoof disorders

425

in time, for example when the whole herd is examined

Foreign body penetration of the sole

425

on the same day, and is known as a cross-sectional

Slurry heel

425

measure.

Vertical fissures (sandcracks)

425

Hardship lines

426

A further distinction must be made between the inci-

Horizontal fissures (sandcracks)

426

dence or prevalence of lameness and the incidence or

Axial wall fissures

426

prevalence of lesions, and care must be taken when

Skin disorders

426

comparing data. The majority of studies record the

Interdigital necrobacillosis

426

prevalence of lesions, as this produces a larger number

Digital dermatitis

427

of observations. Very few studies have examined the

Interdigital dermatitis

429

proportion of lesions that eventually translate into

Interdigital skin hyperplasia

429

clinically detectable lameness, and yet this is an ex-

Mud fever

429

tremely important issue if we are to continue to use

Bone and joint disorders

429

lesion prevalence studies to investigate the causes of

Fracture of the pedal bone

429

lameness.

Apical necrosis of the pedal bone

429

Deep pedal infections

430

The results of studies of lameness incidence will

General treatment and control of foot lameness

430

depend on the person recording. Inevitably, studies by

Footbaths

430

veterinary surgeons will give lower figures than those

Nursing, dressings and footblocks

431

by farmers or stockmen, because rarely are all lame

cows on a farm dealt with by a veterinarian. It is diffi-

cult to provide an average figure for the proportion

Introduction

of lame cows seen by veterinary practitioners. For

example, the study of Clarkson et al. (1996) showed

Lameness remains a major problem for dairy herds

that 3 per cent of lame cows were examined by the

worldwide. There are few other problems which
veterinarian in Cheshire, 20.4 per cent in the Wirral,
produce as much pain and distress to the cow and very
21.7 per cent in Wales and 77.3 per cent in Somerset.
few other problems where the herdsman spends so
An American study where observers went to each farm
much time and effort on routine prevention, namely
twice to record the number of lame cows showed that
hoof trimming. This chapter examines the incidence
their recorded prevalence was 2.5 times higher than
and costs of lameness, the anatomy of the digit, an
that estimated by the herd managers (Wells et al. , 1993).

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Table 31.1

The incidence levels of lameness in several British Isles surveys.

Authors

Date

Recorders

Incidence (cases/100

cows per year)

Hedges et al.

2000

Veterinarians

68.9

Clarkson et al.

1996

Veterinarians and farmers

54.6

Esslemont & Spincer

1993

Veterinarians and farmers

36

Prentice & Neal

1972

Veterinarians

30

Booth

1989

Farmers

30

Arkins

1981

Farmers

28

Whitaker et al.

1983

Veterinarians and farmers

25

Esslemont & Kossaibati

1996

Veterinarians and farmers

24

Collick et al.

1989

Veterinarians

17

Bell & Miller

1977

Veterinarians

11

Eddy & Scott

1980

Veterinarians

7

Russell et al.

1982

Veterinarians

6

Leech et al.

1960

Veterinarians and farmers

4

Whitaker et al. (1983) estimated that in Britain veteri-An American study of 18 farms showed a median

nary surgeons saw only a quarter of the total lameness

prevalence of 11.8 per cent of lactating dairy cows were

cases, the remainder being treated by the farmer. This

clinically lame in the summer and 14.8 per cent in the

was based on a study of 185 herds where the veterinary

winter (Wells et al. , 1995). No overall prevalence was surgeon treated 25.2 per cent and the farmer 74.8 per

provided because of the management differences.

cent of all lamenesses. As herds increase in size, the

A two-and-a-half-year UK study of 37 farms where

stockman becomes more competent and the number both farmers and vets recorded lameness produced a of lame cows seen routinely by the veterinary surgeon mean annual incidence rate of 54.6 new lamenesses per decreases.

100 cows, varying on individual farms from 10.7 to 170.1

The use of data produced by farmer recording

(Clarkson et al. , 1996). The mean incidence rates for the produces problems of terminology and there will be

winter and summer were 31.7 and 22.9 respectively, with

errors in recording the causes of lameness. Hence many

78.7 per cent of the total in the outer claw. The mean

studies refer to 'under-run sole' or 'a foot abscess', when

prevalence rate was 20.6 per cent, ranging from 2.0

in fact the primary lesion was a white line disorder or

per cent to 53.9 per cent, with winter and summer rates

a sole or heel ulcer. This produces further errors in our

being 25.0 per cent and 18.6 per cent, respectively

understanding of the incidence and causes of lameness.

(Clarkson et al. , 1996).

It is generally accepted that the incidence of lameness

The trial reported by Hedges et al. (2000) was an 18-month intervention study involving only five farms in a study provided by both veterinary surgeons and one area (Gloucestershire) of the UK. All lame cows farmers gave an incidence of 3.88 per cent (Leech et al. , were examined by veterinarians, so the study produced 1960). The last major British survey was in 1977 and useful data on the incidence of lesions causing lameness involved 48 veterinary practices which recorded 7526 cases of lameness treated by both veterinary surgeons and farmers in 1821 dairy herds (Russell et al. , in 90 well-recorded UK 1982). The average incidence amongst cattle was 5.5 per dairy herds is shown in more detail in Table 31.2. The cent, although this varied between the veterinary practices, authors concluded that it should be a reasonable target from 1.8 to 11.8 per cent. Most lesions (88.3 per cent) occurred in the feet and 84 per cent of foot lesions for farmers to reduce lameness incidence to second quartile values, i.e. 11 per cent of the herd affected, were in the hind feet. The remaining 11.7 per cent of

giving a target of 14 cases/100 cows per annum. From lameness occurred in the legs and trunk and the major- other figures in Table 31.1 this would appear to be a ity of these involved trauma (Russell et al. , 1982). A rather optimistic target. New Zealand farmer-recorded study of three herds in Although enormous research effort over the past 25 1989/90 showed variations in lameness incidences of 38 years has led to an increase in our understanding of per cent, 22 per cent and 2 per cent (Tranter & Morris, lameness, the incidence has not declined significantly. 1991).

One major reason for this is the advent of digital

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Table 31.2

Incidence of lameness in 90 well recorded DAISY herds (Esslemont & Kossaibati, 1996).

Weighted average values

Overall

Lowest quartile

Second quartile

Third quartile

Fourth quartile

Number of herds

90

22

23

23

23

% of herd affected

17.4

4.4

11.1

18.3

33.8

by lameness

Cases per affected cow

1.4

1.0

1.2

1.4

1.8

Cases per 100 cows

24

4.7

14.1

24.6

47.4

dermatitis. First reported in the UK in the mid 1980s

Hoof

(Blowey & Sharp, 1988), digital dermatitis now ac-

The hoof, or horn capsule, is keratinized epidermis,

counts for around one third of all cases of lameness

similar to hair or finger nails. There are five distinct

seen. In a UK study of five UK dairy herds involving

parts to the hoof, namely the periople, the wall, the sole,

over 1100 cow lactations over 18 months, where all lame

the white line and the heel. The wall, sole and heel

cows were examined and recorded by a veterinarian,

consist of tubular horn, produced by the underlying

the incidence of digital dermatitis was 12.0 cases per

papillary corium (or dermis). Keratinization of the epi-

100 cows per annum, compared with sole ulcers at

dermal cells is a physiological process involving a high

13.9 cases/100 cows p.a. and white line disease at 12.7 rate of synthesis of keratin proteins inside the cell, plus cases/100 cows p.a. (Hedges et al., 2000).

the production of intercellular cementing substances.

Several studies have attempted to record the fre-

This must be carried out entirely by diffusion, because quency of lesions causing lameness, although results the epidermis is avascular. The strength, or quality, of must be interpreted with care. Studies which rely on the horn is determined by three main factors:

veterinary records of cases for which the farmer has requested attention for treatment will undoubtedly underestimate the incidence of conditions such as

- The amount of keratin fibrils within the cell and the strength of their cross-linking disulphide bonds to digital dermatitis and foul in the foot, as many farmers form keratin masses.

would treat these themselves. On the other hand, studies involving farmer recording may use vague terms

- The amount and quality of the intercellular cementing substances which cement the keratinized cells

such as 'under-run heel' or 'sole abscess', without defining together.

ing the initial cause of the lesion (sole ulcer, white line, foreign body penetration, etc.), and these studies would

- The architecture of the horn itself, namely the ratio of tubular to non-tubular horn, which is effectively lack accuracy. Table 31.3 summarizes five sets of data the density of the horn tubules.

on lesions causing lameness.

Heel ulcers are a recently defined condition (Blowey

Horn tubule density is approximately 80 tubules/mm²

et al., 2000a) and may have been classified as under-run in the wall, decreasing to 20 tubules/mm² in the central

heel or foreign body penetration in other surveys. Axial sole area. There is no tubular horn in the white line. The wall fissures also appear to be of increasing importance wall is therefore the stronger part of the hoof, especially worldwide (Vermunt, 1998) and may be a form of white at the toe when it is more mature. As the number of line lesion as many follow the line of the axial groove. horn tubules is fixed at birth, enlargement of the hoof,

for example in older cows, is by an expansion of the

Structure of the foot

intertubular cement. The very large flat foot of a

cow is generally softer and weaker than the small

compact foot of the heifer, because of its lower tubule

The bovine claw consists of three main components,

density.

namely (Fig. 31.1):

The periople is the hairless band of soft horn that sep-

- *Hoof*

arates the wall from the skin at the coronary band. It is

- *Corium*

continuous from one claw to the other and merges with

- *Bone and associated structures*

the bulbs of the heel to give the smooth, waxy coating

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Table 31.3

Types of lesion recorded as causing lameness.

Study No.

1

2

3

4

5

% of

% of

% of

% of

Cases/100

lesions

lesions

lesions

lesions

cows per year

Sole ulcer

12.0

28

13.90

White line disease

18.0

39

16.8

22

12.70

Interdigital necrobacillosis

14.7

15.0

5

7.20

Foreign body penetration

12.3

3.10

Digital dermatitis

8

12.00

Interdigital dermatitis

Interdigital skin hyperplasia

4.2

5

1.20

Interdigital foreign body

2.0

5.6

5

Overworn sole/bruising

1.9

42

8

2.00

Vertical fissure (sandcrack)

1.1

0.54

Aseptic laminitis

4.7

Under-run heel

7.7

Deep sepsis

3.1

8.9

0.45

Heel ulcers

5.80

Axial wall fissures

1.07

Foot lesions

88.3

82.3

Leg lesions

11.7

Hind foot lesions

65.0

35.70

Key to study numbers:

(1) Russell et al. (1982), UK. Lameness reported by farmers and examined by veterinarians.

(2) Tranter & Morris (1991) New Zealand study of three dairy herds. All lame cows examined by veterinarians.

(3) McLennan (1988), Australia. Cases reported to veterinarians.

(4) Clarkson et al. (1996), UK. Lesions recorded by farmers and veterinarians

(5) Hedges et al. (2000), UK study of five dairy herds. All lame cows reported by farmers but examined by veterinarian.

Papillary corium

Laminar corium

Digital

cushion

Wall

Pedal bone

Heel

horn

Wall

Horn

Laminar

Intertubular

tubule

horn

Papillary corium

horn

leaflets

of the sole

Sole

Interdigitating

horn

White

Fig. 31.1

The structure of the hoof wall

line

and white line. (Diagram: Jane Upton.)

seen on good quality hooves. Its main function is to

The wall, produced by papillary corium beneath the prevent dehydration of the horn. Deterioration with coronary band (Plate 31.1), flows over the laminar age or with hot, dry or windy conditions will predispose corium at approximately 5 mm per month. As the length to vertical fissures.

of the anterior wall, from the coronary band to the toe,

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is approximately 75 mm, new horn takes 15 months (75

pedal bone, the corium is impregnated with fat,

[5 = 15) to come into wear at the toe.

fibrous and elastic tissue, to form a shock absorber

The lamellae of the deeper layers of the epidermis

and vascular pump. Trauma to the digital cushion

(namely the hoof capsule) interdigitate with the

results in a permanent replacement of fat and

laminae of the dermis (the corium). This produces a

elastic tissue by scar tissue, with subsequent loss of

structure that is firmly attached to and protects the

function.

corium, but allows free movement for the horn wall and

- On its internal aspect the corium provides nutrients acts as a shock absorber during locomotion.

and vascular support for the pedal and navicular

Sole horn is produced by the papillary corium of bones. Arteriovenous shunts allow blood to bypass the sole and therefore consists of tubular horn and the foot during periods of weight bearing.

intertubular matrix (Fig. 31.1). The sole thickness varies from 10 to 15 mm and thus sole horn comes into wear

Bone and associated structures

two or three months after formation.

The white line is the cemented junction between the

The third phalangeal (pedal) bone, the distal sesamoid

wall and the sole and runs from the heel, along the

(navicular) and the distal interphalangeal (pedal) joint

abaxial wall to the toe, caudally along the axial wall,

are all contained within the hoof capsule. The deep

then dorsally along the axial groove, to end in the inter-

digital flexor tendon is attached to the flexor tuberos-

digital cleft. The white line (Plate 31.2) consists of

ity of the pedal bone and within the heel is separated

laminar horn leaflet cells and interdigitating horn cells.

from the distal sesamoid by the navicular bursa. Small

Both are produced from the adjacent corium at the toe,

fibrous strands of tendinous structure, sometimes

where the laminar corium of the wall joins the papillary

visible in the base of deep sole ulcers, are fragments of

corium of the sole. There is no tubular horn and hence

the deep flexor tendon. The suspensory apparatus of the

white line horn is less mature and considerably weaker

pedal bone (Fig. 31.8) supports the caudal edge of the

than the wall or the sole.

bone. It is firmly attached to the corium on the abaxial

The heel, or bulb of the hoof, is a continuation of the aspect and joins the suspensory ligaments of the leg

perioplic layer, with horn tubules running obliquely in

axially.

an anterior/ventral direction from the heel towards the

sole. The softer heel horn can expand and contract

during locomotion and, in conjunction with the under-

Weight bearing surfaces, hoof

lying digital cushion, acts as both a shock absorber and

overgrowth and wear

vascular pump preventing venous stasis. Consequently heifers which remain stationary, standing for very long As the primary objective of hoof trimming is to restore periods, may develop anoxia of the corium with subsequent poor horn production.

stand the correct weight bearing surfaces of the foot and the distortions that occur due to overgrowth. In a correctly shaped hoof, weight is taken on the heel, on

Corium

the abaxial wall and, to a lesser extent, on the white line The corium is the modified dermis providing nerve and and 10–20 mm of adjacent sole, and on the axial wall vascular supplies (and therefore nutrients) to the hoof running from the toe caudally along the first third of the horn externally and the bones and associated structures axial space. The remainder of the axial surface of the internally. Arteriovenous shunts across the top of the claw should be non-weight bearing (Fig. 31.2). Other foot enable blood to bypass capillaries during weight

dimensions of the claw are given in Fig. 31.3.

bearing, although if the shunts remain open for too long, for example as a result of laminitis/coriosis, anoxia

Hoof overgrowth

and consequently dyskeratotic horn formation result.

The corium is structurally modified, enabling it to per-

The size and shape of the hoof at any one time will be

form differing functions in various areas of the foot,

a balance between the rate of growth and the rate of

namely:

wear. As one might expect, there are a variety of factors that influence both processes. For example, horn growth

- *Papillae on the wall and sole extrude tubular horn.*

is faster:

- *The laminar corium is a support structure where it interdigitates with lamellae on the hoof wall and in*

- *In young animals*

its distal extremities it produces white line cement.

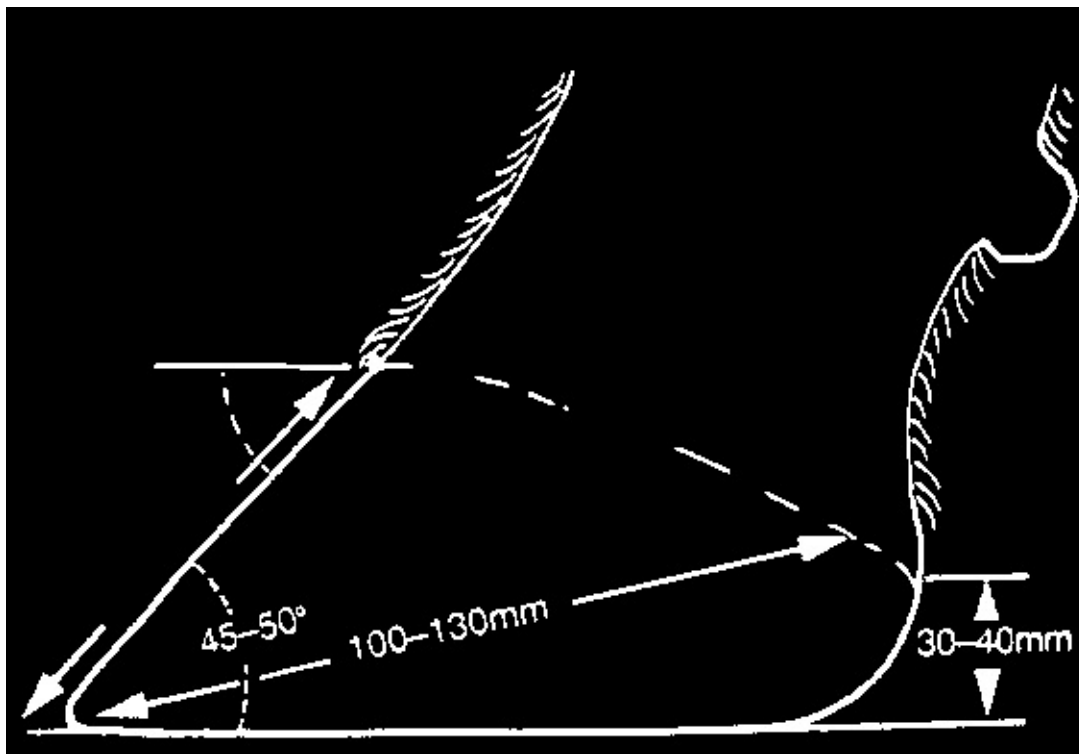
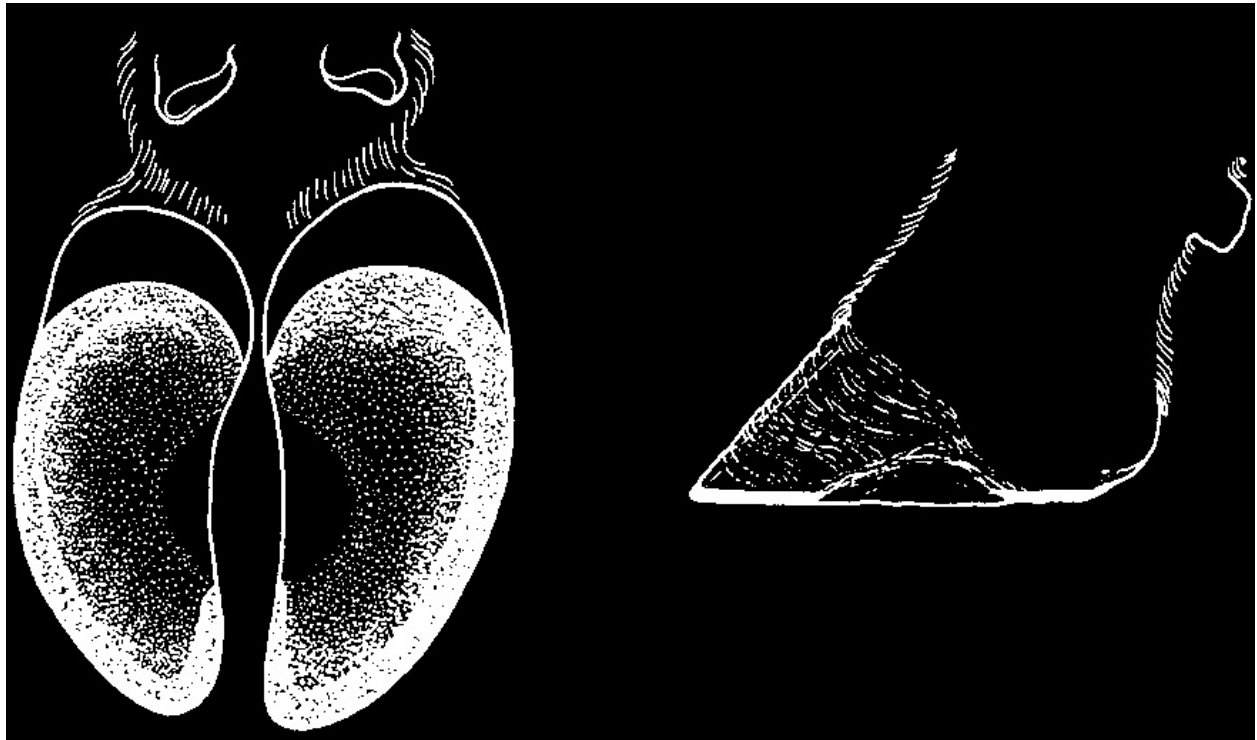
- *With high concentrate feeding*

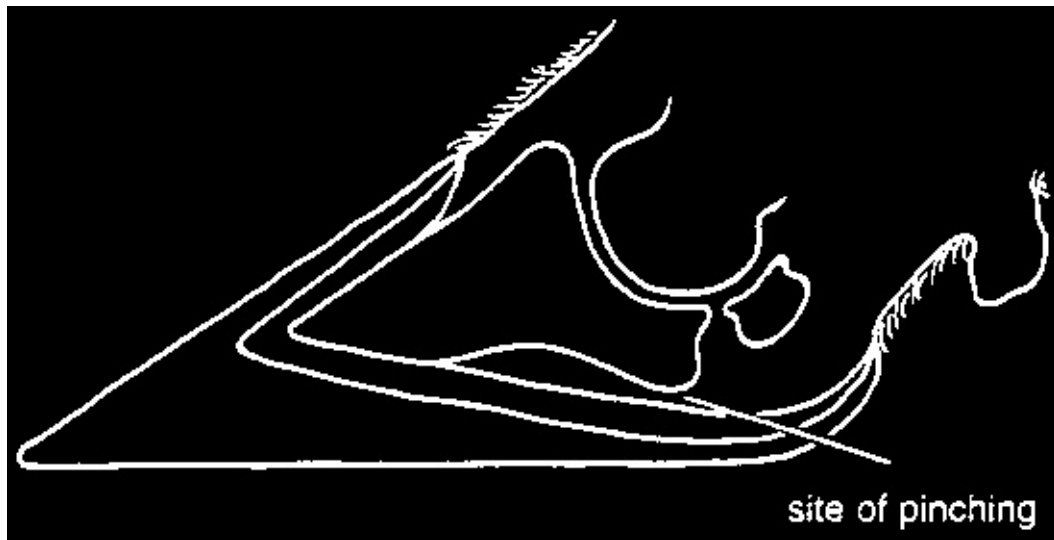
- *The digital cushion: within the heel and extending*

- *With more exercise*

forwards to a point beneath the caudal edge of the

- *On rough surfaces*





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Fig. 31.2

The correct weight bearing surfaces of the foot.

60–80mm

Fig. 31.4

Overgrowth at the toe leads to a caudal rotation of the pedal bone.

Fig. 31.3

Approximate dimensions and angles of a normal claw.

the pedal bone remains the same size, irrespective of the degree of overgrowth. In this respect the hoof is

The rate of wear is increased by factors such as:

very different from the cow's horn, where the cornual

- *Wet conditions underfoot, leading to softer horn*

bone lengthens with increasing length of the horn.

which wears faster

- *Excessive standing and walking*

Overgrowth of the lateral wall

- *Hard and/or abrasive floor surfaces*

In some animals the wall of one claw grows faster than the other and starts to curl under the sole. This produces

Overgrowth at the toe

a corkscrew effect at the toe. Corkscrew claw may be a

The horn of the wall is generally harder than the horn genetic trait or can be a result of coriosis/laminitis.

of the heel, so although both may grow at the same rate, horn is worn away more slowly from the toe than from

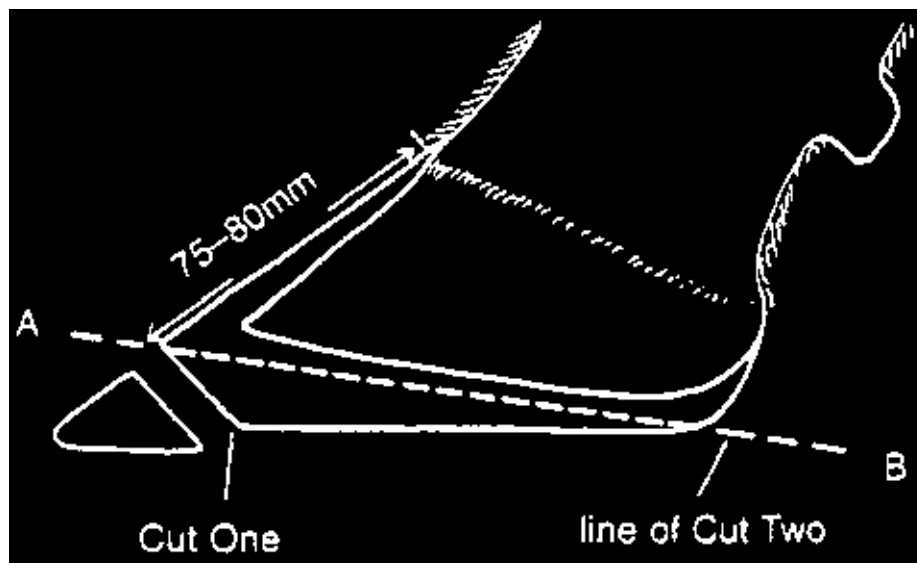
Overgrowth of the sole

the heel. This results in overgrowth occurring primarily

at the toe. The additional horn at the toe lifts the front A ledge of horn is commonly seen growing from the

of the foot and the front wall then forms a more shallow sole and extending into the axial space (Fig. 3.17, Plate

angle, decreasing from 45° to 30° or 20° , or even to the 31.4°). In some instances it may even overlap the adjacent horizontal. In extreme cases the front wall becomes a claw, and may be so pronounced that it becomes concave and the toe is lifted off the ground (Plate 31.3). These changes are shown in Fig. 31.4. Internally the importance in the pathogenesis of sole ulcers, as the pedal bone is rotated backwards, thereby putting even overgrowth of sole horn is immediately beneath the more pressure on its rear edge (the flexor tuberosity) flexor tuberosity of the pedal bone and in an area where and further increasing the risk of sole ulcers. However, weight bearing should be minimized.



Disparity of claw size

of the abaxial wall, across the dorsal wall to the sole surface of the axial wall. 'Hardship lines' can be used

The lateral claw of the hind foot is often considerably larger than the medial claw. There is no single reason to establish the chronology of previous episodes of coriosis/laminitis, and therefore the potential causes of for this and suggested causes include:

current hoof problems.

- *The lateral claw is naturally slightly larger than the medial (Paulus & Nuss, 2002).*

Hoof trimming

- *Poorer suspension of the pedal bone within the lateral claw, leading to pinching of the corium and stimulating the growth of horn. The caudal edge of one common aim, namely to restore the foot to its the pedal bone is suspended within a 'hammock', correct shape and weight bearing surface. The manner known as the pedal suspensory apparatus (fig. 31.8).*

in which this is achieved is less important and the fol-

This is connected to the laminae on the abaxial wall,

lowing text simply describes one approach (Blowey,

and axially to the suspensory ligaments of the leg.

1998), with emphasis being placed on the anatomical

- A greater variation in load bearing on the lateral
corrective stages, rather than the precise method used.*

claw compared with the medial claw when the cow

*Other approaches are described elsewhere (Toussaint-
is walking (Toussaint-Raven, 1985).*

Raven, 1985). Although described as a four-stage pro-

- A leg conformation in which the hocks point in-
cess, in reality one part of the trimming process merges
wards and the toes outwards.*

with the next.

- Excessive engorgement of the udder at calving,
forcing the legs apart.*

Cut one

- The hind feet are the major propelling force of
the cow during locomotion, pushing her forwards,*

The overgrown toe should be cut to its correct length,

whereas the front feet are the major weightbearing which is approximately 75–80 mm from the coronary structures.

band to the toe, or one handspan. Most experienced hoof trimmers would simply estimate this distance but,

In the front feet the position is reversed: the medial when learning, it would be better to measure specifically claw is commonly larger than the lateral claw.

cally. After cut one the cow is left with a 'square-ended toe'. In Plate 31.5 it can be seen that the white line now

Negative net growth

passes across the end of the toe and clearly the wall,

At housing, and especially when housing and calving

which should be the correct weight bearing surface, is

coincide, heifers invariably undergo a period of negative

no longer weight bearing at this point. Although the

net growth. The rate of hoof horn growth is reduced but anterior wall may now be the correct length, the toe is

at the same time there is a rapid increase in wear, espe-

still too high and this means that the front angle of the

cially at the toe. This leads to a shortening and increased

anterior wall is too shallow. This is demonstrated in Fig. 31.5.

some heifers the changes may be sufficiently extreme for the horn at the toe to become totally eroded, expos-

Cut two

ing the corium. Such heifers are said to have 'soft soles' or 'toe ulcers'. The syndrome is seen especially in heifers

The next stage is to remove the excess horn from the that have to walk long distances to and from pasture, for sole surface of the toe, thus dropping the toe relative to example in New Zealand and Uruguay, and also in the heel and bringing the front wall back to a more young bulls introduced into a dairy herd. It may be occasionally seen in maiden heifers when they are housed at the end of their first grazing season.

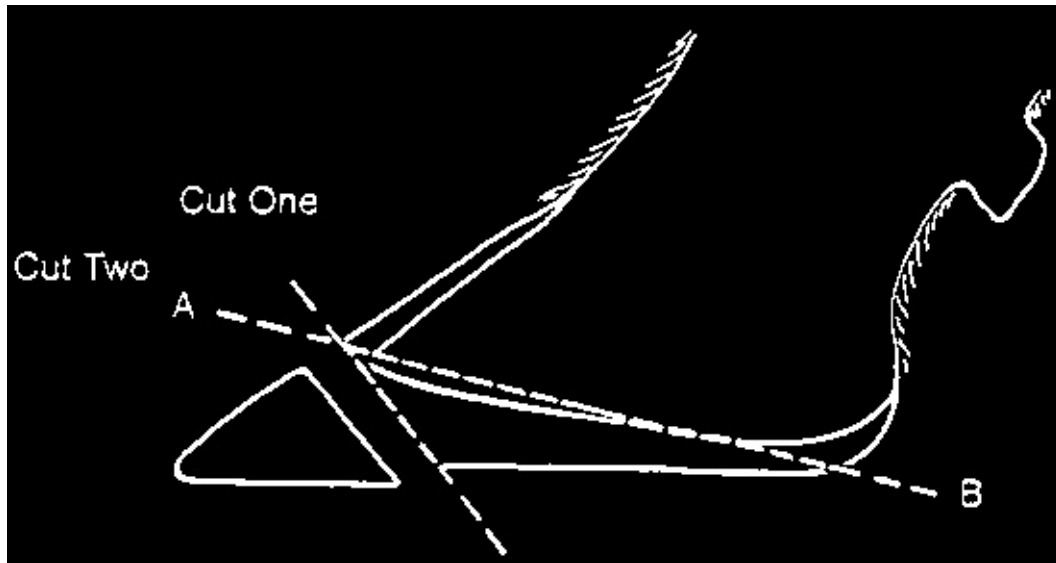
Hardship lines

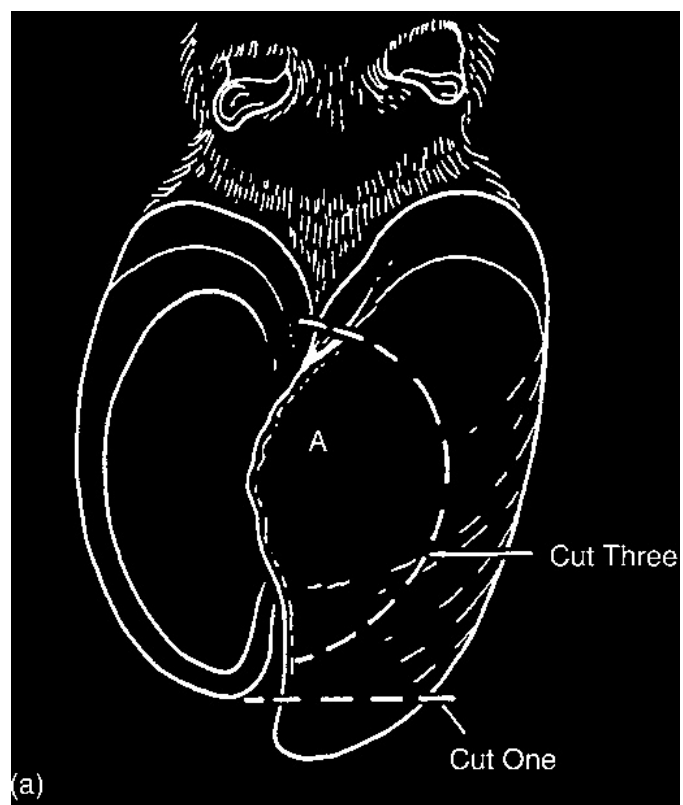
Temporary disruptions of hoof formation in the anterior wall lead to circumferential rings of variable thickness known as 'hardship lines'. When horn production is poor, a groove is formed and in extreme cases results

in total cessation of horn formation, leading to a horizontal fissure. Because the dorsal wall is longer than the heel, the hardship lines often run from the sole surface

Fig. 31.5

Hoof trimming cuts one and two.





(a)



(b)

Fig. 31.6

Hoof trimming. Cut one should not be made too short.

upright position. The horn to be removed in cut two lies beneath the line AB (Fig. 31.5), which is a line joining cut one to the base of the heel. The first part of cut two can be performed by removing part of the wall using hoof clippers, but later stages should be carried out with a hoof knife, using thumb pressure to check this area of the sole regularly for signs of softening. A softening of the horn should not occur, but if it does then trimming must stop. There will only be a few millimetres of horn before the corium is penetrated and exposure of the corium in this area of the foot can lead to quite severe and protracted lameness.

If cut one was in the correct position it should be possible to remove horn from the sole surface of the toe until the white line and adjacent wall are once again clearly visible at the toe. The wall is now the weight

bearing surface. It is important that this is achieved, otherwise the weaker white line structure would become weight bearing, with obvious adverse consequences.

It is vital that cut one does not make the hoof too short; this scenario is shown in Fig. 31.6. Because cut one was too short, a line drawn from the top of cut one to the bottom of the heel (AB in Fig. 31.6) would lead to exposure of the corium at the toe and this can produce severe lameness.

Cut three

Fig. 31.7

Hoof trimming. Cut three and the finished foot.

Cut three consists of removing any axial overgrowth of the sole, followed by dishing the axial sole surface of both claws (Fig. 31.7a), so that weight bearing beneath the flexor tuberosity of the pedal bone is minimized.

Cut three also slightly increases the space between the longer. This usually means removing additional horn digits. This makes impaction by dirt and foreign bodies from the outer claw of hind feet and the inner claw of less likely, decreasing the incidence of diseases such as

front feet, bringing the legs back to the upright position.

foul, interdigital dermatitis and interdigital skin hyper-

This produces more even weight bearing.

plasia ('corns').

General points

Cut four

When trimming is complete, points 1, 2, 3 and 4 on Fig.

The final stage is to trim the two claws back to ap-

31.7b should all be on the same longitudinal horizontal

proximately the same size with the lateral claw 4–5 mm

plane, to provide adequate weight bearing. The two

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claws should also be of equal size and their two sole sur-

• Conditions of the skin: digital dermatitis, inter-

faces on the same transverse horizontal plane. Removal

digital necrobacillosis, interdigital skin hyperplasia

of the axial (inner) wall CD (Fig. 31.7b) is a common

and mud fever.

mistake made by some herdsmen who feel that the toes

• Disorders of the pedal and navicular bones.

should not be touching once trimming is complete. This

is a fallacy. If the wall CD was lowered the claw would be seriously destabilized, causing it to rotate inwards

Sole ulcers and white line defects

and allowing overgrowth of the lateral wall. In the Pathogenesis

worst case excessive removal of the axial wall might expose the corium, leading to severe lameness.

The horn of the white line and sole is produced by the

It is preferable not to remove any heel horn unless it corium. Damage or disruption of the corium will there- is badly under-run, other than as part of cut four. If the fore be the primary change that later leads to the for- heel is only slightly pitted, it is best left alone, since mation of poor quality horn, and this is eventually removal of the heel could lead to backwards rotation of seen as defects such as sole ulcers and white line disor- the pedal bone and so predispose to sole ulcers.

ders. The syndrome is sometimes referred to as 'claw-

One theory of hoof trimming recommends that it is horn disruption', although once again, as the primary advisable first to trim the medial claw to the correct

defect is in the corium, then this is the area to which shape and then use this as a template for trimming attention should be focused. Although the term 'laminitis' is often used, the majority of changes affect the lateral claw. Whilst this system may have its merits, it is the opinion of the current author that it is not whole corium, and particularly the papillary corium always a correct course of action, and especially when of the sole, where there are no laminae. Hence use of dealing with lame cows. This is because it may be the term 'laminitis' in cattle is not particularly accurate. beneficial on welfare grounds to leave the medial claw. It is doubtful if laminitis occurs as a single entity slightly larger than normal in order to increase its in cattle, and the more general term of 'coriosis' is weight bearing potential when there is a lesion in the preferable.

lateral claw.

Changes associated with the corium

Timing of foot trimming

Coriosis can occur as a result of trauma, infection, nutri-

tional imbalance/excess and toxic states and commonly

There is no one correct time to trim feet. Potential

is a sequel to a combination of causes. The overall result

options include:

will be the same, namely altered horn formation with

- *When overgrowth occurs. Leaving a foot with over-
the risk of lameness when the poor quality horn reaches
grown horn not only makes walking uncomfortable
the bearing surface of the sole.*

but it also predisposes to the development of more

*Ossent (1995) describes three stages in the patho-
serious lesions such as sole ulcers.*

genesis of sole ulcers and white line disease, all of which

- *When the cow is lame. Clearly all lame cows
arise as a consequence of coriosis:*

should have the affected foot lifted, trimmed and

(1)

*Disruption of blood flow within the corium, leading
examined.*

to sludging, poor oxygenation of epidermal tissues

- *At drying off. As many of the management and*

and consequently poor keratin synthesis.

feeding 'insults' leading to lameness occur at the

(2)

The laminar suspension of the pedal bone within time of calving, it is ideal to have feet in optimum the hoof is disrupted and the bone sinks within the shape at this stage. In addition, at the end of lacta-hoof.

tion there may be a build up of horn from insults

(3)

Compression of the corium, especially beneath the suffered in the previous lactation

flexor tuberosity of the sinking pedal bone, leads to further ischaemic necrosis, disrupted horn formation and consequently the production of a sole

Lesions causing lameness

ulcer.

The changes at the dermal–epidermal junction are

The majority of the lesions causing lameness are in the still not fully understood. It is thought that they involve foot. In this section these will be subdivided as follows:

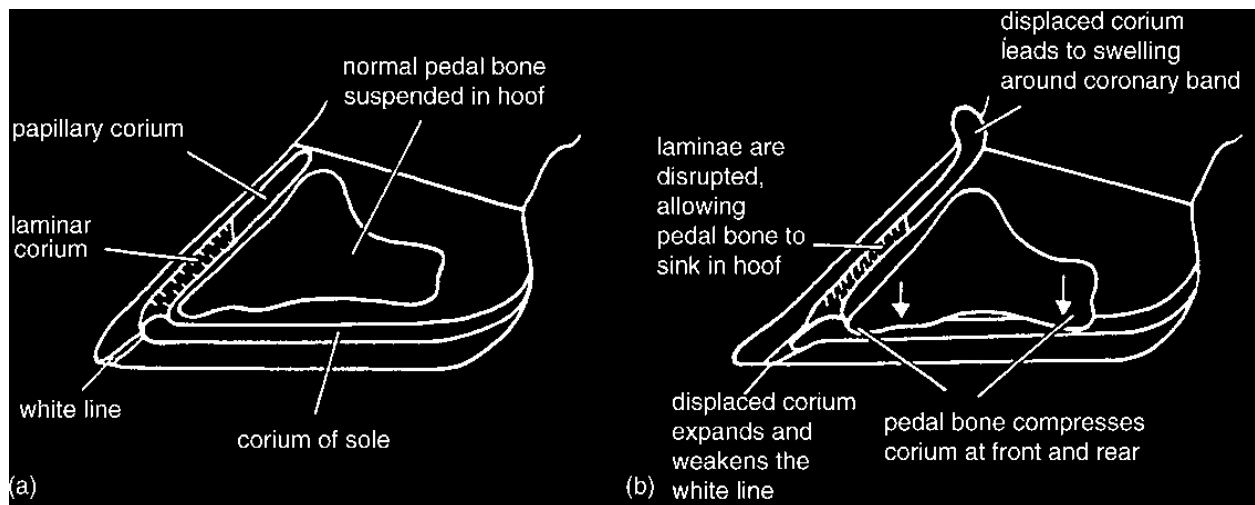
an initial release of vasoactive substances. Vasodila-

- Sole ulcers and white line defects.

tion in the corium leads to vascular stagnation and

- Other hoof problems: foreign body penetration, the opening of the arteriovenous shunts exacerbates horizontal and vertical fissures.

this. The ensuing hypoxia leads to transudation,



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thrombosis and ischaemic necrosis, and consequently

months later. The nature of the horn defects will

poor keratin synthesis and lamellar disruption.

depend on the severity of the initial corium damage,

Haemorrhage on the sole is often referred to as

and are likely to appear as:

‘bruising’. This may be a correct term, although it

- Yellow discoloration – if serum only was released.*

should always be remembered that the ‘bruise’ was

- Haemorrhage – if the blood vessels ruptured (Plate 31.6).*

the horn now on the surface of the sole was being pro-

- A sole ulcer – if the damage to the corium was so*
duced. As such, bruising of the sole cannot be impli-
severe that horn formation was totally disrupted
cated as a recent cause of lameness. The effect of mixing
(Plate 31.7).

serum or blood with the horn can be likened to mixing
sawdust with concrete – it weakens it considerably. This

If there is a generalized inflammation of the corium,
is particularly the case for the white line, which is an
then the suspension of the pedal bone within the foot
inherently weak structure, and at the sole ulcer site
is disrupted, allowing the bone to sink within the foot,
where there may be almost ‘neat sawdust’ because so
as shown in Fig. 31.9. The corium is then displaced and

much haemorrhage is present. The whole process is the following changes occur:

very similar to the changes which occur when a finger-

- The 'sinking' bone puts further pressure on the nail is bruised: the blood spot often starts at the corium corium, especially beneath the flexor tuberosity, the of the skin–nail junction and then slowly grows to the heel or the toe. Ulceration of the sole, heel or toe distal extremity of the fingernail over the next few may result.*

months.

- Lateral displacement of the corium into the white line area leads to weakening and widening of the*
Changes associated with the pedal bone
white line cement with increased risk of white line defects.

The ventro-axial border of the pedal bone is arched in shape. The pedal bone is suspended within the hoof by the laminae, with a much stronger attachment to the abaxial wall than to the axial wall (Fig. 31.8) of the hoof.

Digital suspensory

ligament

When weight is transmitted down the leg the bone rotates slightly on the axial surface, putting increased

Firm attachment

weight on the flexor tuberosity. Increased weight of ligament to

bearing puts extra pressure on the corium and if it is

P3

P3

abaxial wall

already in a fragile state, then it is likely to become damaged. Pinching of the corium between the flexor tuberosity of the pedal bone above and the horn of the

Fat pad in suspensory

sole beneath leads to 'bruising' of the corium. This leads

ligament at caudal

edge of P3

to defective horn formation and the changes will be seen on the bearing surface of the sole one or two

Fig. 31.8

Suspension of the pedal bone within the hoof.

Fig. 31.9

Coriosis leads to disruption of the laminar suspension of the pedal bone.

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- *Dorsal displacement of the corium may produce*

Note that no dressings are recommended: in fact many a circumferential thickening above the coronary would consider them to be counterproductive. This is band.

referred to later.

- *Anterio-ventral displacement of the pedal bone (i.e. movement towards the toe) may leave a hori-*

Heel and toe ulcers

zontal depression or 'furrow' in the heel horn. This is a classic sign of disruption of the laminar sus-

Although sole ulcers are by far the most common, there pension with subsequent movement of the pedal can be areas of haemorrhage or even total perforation bone.

at other areas of the sole. Toe ulcers are thought to occur when the pedal bone sinks within the hoof 'bows

The poor laminar suspension on the axial wall com-

first' – that is, the front of the pedal bone drops before
pared with the abaxial wall (Fig. 31.8) leads to the axial
the flexor tuberosity at the rear.

aspect of the pedal bone, and especially its flexor

Heel ulcers are seen as small dark red/black marks
tuberosity, having the greatest contact with the corium

(Plate 31.8) in the central sole area towards the heel

of the sole. This explains the typical site of the sole ulcer

(Blowey, et al., 2000a). Plate 31.8 shows a typical heel in zone 4 (mid sole) of
the hoof. Once the pedal bone

ulcer on the left claw and haemorrhage at the site of an
has dropped within the hoof it will never regain its orig-

early sole ulcer on the right claw. Some heel ulcers

inal position. In addition, a corium that has been

simply track down to the corium and fade to nothing.

damaged by ischaemic necrosis heals by fibrosis and is

Others lead to under-running and abscessation of the

therefore less able to produce good quality horn in the

sole at the sole–heel junction and can produce a marked

future. As a consequence, heifers affected by coriosis

lameness. Heel ulcers represent a significant cause of

during their first lactation are more susceptible to lameness (Table 31.3). They often occur in association with sole ulcers, although they are seen more commonly in later lactations. For example, in one study, heifers that developed lameness in their first lactation on the medial claw of hind feet. The cause of heel ulcers is not known, although one theory is that they are produced by pinching of the corium under the caudal edge of the pedal bone. At this point the bone is suspended in the pedal suspensory apparatus (Fig. 31.8), within term lameness control measure.

Optimal management of the precalving and periparturient first lactation heifers is therefore vital as a long term lameness control measure.

which there are three fat pads acting as shock absorbers. Continual compression of the corium of the sole can lead to generalized poor horn formation, sometimes shown to undergo cartilaginous change, and it may be seen in older cows where a sole ulcer totally fails to

the effect of this that leads to the formation of heel
heal. In such animals a layer of very poor quality 'horn',
ulcers. Heel ulcers are hence in the central sole region sometimes little more
than a layer of fibrous tissue,
at the junction of zones 4 and 6 (zone standardization
may form over the ulcer site, and the tip of the flexor
from the Liverpool International Ruminant Digit Sym-
tuberosity of the pedal bone can sometimes be palpated
posium 1990, cited by Greenough et al., 1997). Sole
as a hard lump immediately beneath.
ulcers typically occur in a more anterior and axial posi-
tion on the axial aspect in the centre of zone 4, and toe ulcers are seen in zone 5
at the toe.

Sole ulcers

A sole ulcer (Plate 31.7) is a physical condition, caused

White line defects

by trauma to a fragile corium, and treatment must
Weakening of the white line, brought about by the
therefore be aimed at minimizing this trauma. The main
inflammation associated with laminitis/coriosis, can
steps for treatment are:
result in a range of white line disorders. The most

- *Dish the sole ulcer site so that weight bearing is*

common of these are:

minimized.

- *Remove any under-run horn around the ulcer, to*

- *Sterile abscessation.*

eliminate pockets of necrotic horn and infection,

- *White line separation.*

thus allowing the formation of new horn.

- *White line penetration and abscess formation, with potential complications of infection tracking along*

- *Remove protruding granulation tissue.*

the laminar corium towards the coronary band.

- *Reduce the size of the affected claw as much as possible, and maximize weight bearing on the sound*

Sometimes the internal inflammation within the

claw. For more advanced cases, apply a block to the

corium is so severe that pockets of necrotic tissue are sound claw (e.g. Plates 31.7 and 31.20).

formed. These can produce a sterile internal abscess

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and as there may be no obvious tracts running from the

- It leaves a pit which can easily become impacted outside, they may be quite difficult to locate and treat.

with stones or dirt, thereby impeding drainage and

In severe forms of coriosis the whole sole becomes predisposing to further white line impaction.

separated by an accumulation of inflammatory fluid.

- By creating a pit, areas of under-run horn and

This is known as a false sole.

pockets of infection are much more likely to be

More commonly fissures develop vertically into the

missed. If a small area of adjacent wall is removed, it

weakened white line, a process known as white line sep-

is much easier to expose and drain the affected area.

aration (Plate 31.9). This occurs especially if the cows

Protruding granulation tissue is often an indication

are walking over rough or stony ground, or when they

that there is adjacent under-run horn that needs to be

make sudden turning movements, as when escaping

removed, and use of a block on the sound claw to

from an aggressive cow. Small stones may then become

prevent weight bearing on the affected claw improves

*impacted and with continued walking these may even-
the rate of healing. A swollen, hot and painful coronary
tually penetrate the corium. The most common point
band is caused by infection tracking into deeper struc-
for white line separation and penetration is at zone
tures such as the navicular bursa or tendon sheaths, and
3, where the rigid abaxial hoof wall joins the flexible
in such instances antibiotic therapy and/or more radical
heel. During locomotion this is where there are the
treatment is indicated.*

*greatest sheer forces between the rigid hoof wall, the
suspended pedal bone and the movements of the flexi-
ble heel horn. Once the corium has been penetrated,*

Causes and control of coriosis (laminitis)

*the invading foreign body (usually a stone or grit) intro-
duces infection. For white line abscesses near to the
Coriosis is clearly the primary factor responsible for
heel, natural drainage is through the soft horn of the
defects in the horn capsule. In this section the aetiol-
heel. White line abscesses close to the toe do not have
ogy, and consequently the control of coriosis, is*

such an easy escape route and often infection tracks described under the headings of parturition, excessive upwards through the laminae, to discharge at the coronary band, nutrition and general management.

nary band. This produces a more severe lameness because at the toe the pedal bone is tightly attached to

Parturition

the hoof, and there is therefore very little room for the pus to expand.

Many authors have shown an association between par-

Whereas a sole ulcer results in damage to the under-

turition and increased incidence of lameness, with the

lying corium, the majority of white line lesions prima-

peak incidence of white line lesions in first lactation

rily produce separation of the horn from the underlying

hoof occurring nine weeks after calving, and sole

horn-forming corium. Uncomplicated white line lesions

ulcers at 14 weeks postpartum (Leach et al., 1997). In therefore normally heal much more quickly than sole

cows the incidence peaks slightly later. Green et al.

ulcers. White line lesions in the axial groove are

(2002) showed that lameness from all causes peaks in described on page 426.

the second and third month after calving. The rings on

The treatment of a white line abscess is very similar

a cow's horns, one for each calving (Plate 31.10), are said

to that for a sole ulcer, namely:

to be a reflection of the natural disruption in horn for-

mation (Blowey, 1998) that occurs at calving. Livesey

- Remove all under-run horn, even if this means*

et al. (2000) suggested a decreased incorporation of

removing the wall from the sole to the coronary

amino acids into hoof horn at the time of parturition,

band, or the whole of an under-run sole. A few

presumably associated with a repartition of nutrients

authors recommend leaving a bridge of hoof wall to

(sulphur amino acids) towards milk production. Hirst et

prevent movement of each side of the bisected wall,

al. (2000) showed that cows that develop lameness in

but the danger of doing this is that the under-run

their first lactation are more likely to become lame in

infection will not be fully drained.

subsequent lactations, and suggested yield as a contrib-

- Reduce the size of the affected claw, to minimize

utory factor. Green et al. (2002), in an intervention

weight bearing, and leave the sound claw large.

study involving 1109 cow-years on trial, showed that

Blocks and dressings are discussed on page 431.

high-yielding cows were more likely to become clini-

When using a hoof knife to drain infection from the

cally lame than lower-yielding animals, but in the

white line, it is important to remove the short section of

affected group, lame cows produced 396 litres per cow

adjoining hoof wall. Digging a deep pit into the white

less milk than their non-lame counterparts.

line with the curved point of the hoof knife has two dis-

The changes that occur in all periparturient cows

advantages, namely:

appear to have a marked impact on the corium, horn

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strength and subsequent lameness. There is a decrease

Although the average lying time of the heifers was ten

in the rate of rumination, and this leads to a 25–30 per

hours, some animals lay down for as little as five hours
cent decrease in dry matter intake, at a time when the
each day. This group showed the highest incidence
cow's requirements (for the fetus and milk produc-
of lameness and quite severe haemorrhage persisted in
tion) are rising rapidly. There is an increased risk of
the sole horn for up to four months after calving. In
rumen acidosis and a marked immune suppression.
most dairy systems heifers are forced to spend longer
Dietary change, namely moving from a low concentrate
on their feet after calving. They will stand waiting to be
dry-cow ration to a high performance production diet,
milked, they spend longer standing to feed, because
further increases the risk of rumen acidosis and
they are often last to feed, and need to eat more as
coriosis.

lactation proceeds. They will have recently been mixed
Whatever the cause, the increased fragility of the
with the main herd and are now having to compete with
corium during the periparturient period means that it
older cows and fear may restrict their entry into a

is particularly susceptible to trauma. However, in most cubicle shed, especially if they are of low social dominance and have had no previous cubicle training.

corium occurs, due to excess periods of standing

Excessive standing may be bad for the immediate and aggressive interactive movements between cows postpartum cow, but standing still is even worse. If the animal does not move, the vascular pumping mechanism.

nisms of the heel and digital cushion will be impaired.

Even if they calve outside in a field, for a few days

Vascular stasis predisposes to anoxia and damage to the after calving cows, and especially heifers, will spend corium, with resulting poor horn formation. It is essential that there are adequate loafing areas to allow the decreased. There is then more weight on the corium cows to walk around freely. Overcrowding should be and a greater potential for bruising. It is not known

avoided, even in collecting yards. Animals that are whether the decreased lying times are due to inherent packed tightly together have little option but to stand nursing behaviour (attending to the calf), discomfort still. Adequate loafing areas also help to improve from the perineum, an enlarged udder or to some other oestrus expression and hence fertility. factor.

The incidence of sole ulcers and white line disease Diseases such as mastitis and metritis are more will therefore be markedly reduced if animals are common immediately after calving. This also increases encouraged to maximize lying times in the immediate the fragility of the corium and in extreme cases will postcalving period, for example for the first two to six produce hooves with hardship lines and horizontal weeks after calving.

fissures. Heifers are likely to be worst affected, partly because they have often had no prior experience of

Post-calving comfort: Of all the above factors, cubicle the housing and milking system and partly because in

comfort is probably the most important. Cubicles may make the management of cows easier, but they are not always ideal in terms of cow comfort and lameness. Several surveys have shown that the incidence of lameness immediately postpartum, when immune suppression is at its maximum, and this further depresses horn formation. This must point to cubicles being less than ideal, especially for the immediate postcalving period. This leads to long periods of standing, producing increased hoof wear at a time when hoof growth is minimal. A small but increasing proportion of farms are now housing their freshly calved cows in 'maternity' loose around parturition and housing is referred to below and yards for the first two to six weeks after calving and then on page 415. transferring them to cubicles. Experience from such

systems suggests that in heifers especially, a postcalving period of loose housing leads to:

Excessive standing

- *Increased yields.*

Anything that leads to a decrease in lying times, espe-

- *A decreased incidence of lameness.*

cially in the immediate postcalving period when the

- *Improved cubicle acceptance when the heifers are*
corium is in its most fragile state, will increase the inci-
eventually transferred from the yard to the cubicles.

dence of sole ulcers and white line disease.

One experiment (Leonard et al., 1996) deliberately

The third factor is perhaps the most surprising. One

housed heifers in an overstocked cubicle building (17

might have expected that cows and heifers that had got

cubicles for 35 heifers) immediately after calving.

used to a straw yard would be very difficult to retrain

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to use cubicles. The fact that the reverse is true proba-

Nutrition

bly indicates that parturition is a stressful experience

High starch/low fibre diets that lead to rumen acidosis and that it is only when animals have fully recovered undoubtedly predispose to coriosis and subsequent lameness, especially if fed both pre- and postpartum.

system. The major problem with straw yards is the Blowey et al. (2000b) used 48 multiparous cows to increased incidence of environmental mastitis and compare high starch (wheat-based) with equal energy hence perhaps the ideal situation would be a 'maternity high fibre (sugar beet pulp) rations fed pre- and post-group' housed in a low stocking density system of partum. Sole haemorrhage scores at 24 weeks post-luxury cubicles.

partum were significantly higher in the high starch group. Livesey and Flemming (1984) showed that 64 per Cubicle design: Cubicle comfort is obviously all-cent of cows on a low fibre diet developed sole ulcers important. Ideally, cubicles should be long enough and compared with only 8 per cent on the high fibre ration. wide enough (1.15 metres wide and 2.4 metres long) to

Concentrate intakes should be built up slowly after accommodate the larger Holstein cows and with sufficient space at the front to allow the cow to lunge calving for average yielding cows and probably three forward 1–1.5 metres as she stands up. If there are two weeks for higher yielding animals, which peak later. facing rows of cubicles, a length of 2.2 metres is adequate. Ideally no more than 4.5 kg of feed should be given in the parlour. Cantilever-type divisions are ideal. Also recommended are a 100 mm fall from front to rear, a step of well mixed with the ration, helps to stimulate rumination, thereby promoting a good flow of saliva and not more than 130 mm down into the dunging channel and a brisket rail at the front 1.75 m from the kerb, decreasing acidosis. There is evidence that maintaining which prevents the cow from shuffling too far forwards, an ideal dietary cation–anion balance (DCAB) is beneficial but at the same time provides ample space for lunging official (see p. 787). Acidosis can be recognized clinically

as she stands up.

by loose faeces, an increased incidence of digestive

When attempting to stand, the cow lunges forward

upsets, cud regurgitation and a sweaty coat.

1 to 2 metres and lifts herself first onto her hind feet,

Although high protein diets have occasionally been

then up onto her front feet. When she is lying down or

suggested as a cause of coriosis, most consider protein

half standing, therefore, much of her weight is taken

to be of less importance than other factors. High intakes

on her knees. If the floor surface is hard, and par-

of poorly fermented grass silage have also been impli-

ticularly if it is also rough, cubicle acceptance will be

cated, although this could be due to toxic amines rather

low. The worst possible cubicle floor is a stone base,

than high protein.

poorly compacted and with insufficient straw. In an

Even feeding during rearing influences the incidence

attempt to get comfortable cows will shuffle forwards,

of sole haemorrhage, with heifers fed high levels of con-

until they are so close to the wall that they are unable

concentrate being the worst affected. High fibre diets are to lunge to stand. If a proportion of the cows are too now recommended for rearing dairy heifers.

far forward in the cubicles, cubicle comfort should be re-examined.

Most cubicle bases are made of concrete. This is fine
Trace elements and vitamins: Many attempts have been provided that it is deeply bedded, although it is often made to improve hoof condition by mineral, vitamin difficult to retain a well-matted straw bed. A variety of and trace element supplementation. The use of zinc, mats and mattresses are available and these are certainly much better than concrete alone. However, particularly zinc methionine, is often promoted as a feed supplement having beneficial effects. If one of the some bedding should be used, even with mats, other-reasons for the production of poor quality horn at wise hock sores will develop. A disadvantage of mats calving is a temporary deficit of sulphur amino acids is that it is difficult to get large amounts of straw (see p. 419) then logic would suggest that supplemen-

bedding to adhere to them, although the cows enjoy tation with zinc methionine might be beneficial at this standing on rubber mats. Deep sand cubicles (for time, since methionine is a sulphur amino acid and zinc example 70 mm) also work well and reduce the inci-promotes healing.

dence of mastitis, although sand may lead to problems Biotin has been shown to influence the differentia-with slurry disposal systems. Choosing the correct type tion of epidermal cells into hoof horn; it boosts the pro-of sand, which does not compact and consolidate, is duction of keratin and it stimulates the production of essential.

intracellular cementing substance (ICS). ICS is a vital The best cubicles are comfortable cubicles. While component of non-tubular horn, such as is found in the design and dimensions may be important, comfort is of white line, and this is perhaps why biotin supplementa-even greater significance (see also pp. 40–42).

tion has a major effect on white line disease. Although

1.00

Unsupplemented = 0

0.95

Supplemented = 1

0.90

0.85

Fig. 31.10

Effect of biotin supplementation on white line lameness in dairy cows.

0.80

No effect was seen until after 130 days of

Proportion of cattle alive

supplementation (Hedges et al., 2001). Re-

0.75

produced with permission of the American

0

100

200

300

400

500

600

700

Dairy Science Association.

Time (days)

*one might expect ruminal synthesis of biotin would
ness, placing particular emphasis on those factors which
meet the requirements of the cow, Da Costa Gomes
might damage the corium, especially in the early lacta-
et al. (1998) showed that with high concentrate diets
tion animal.*

*producing rumen acidosis, in vitro biotin synthesis
was reduced from 1.5 to 0.3 mg per day. Herds with sig-*

*Wet hoof: This is softer than dry hoof and therefore the nificant problems
associated with horn quality may*

sole is more likely to become penetrated or the corium

*benefit from biotin supplementation, although atten-
bruised if the feet are damp. Cubicle passages should
tion to dietary management would also be important.*

be scraped twice daily and the addition of small quanti-

In a study with 100 first lactation heifers, Midla

ties of slaked lime to the cubicle beds once a week will

et al. (1998) showed that supplementation with 20 mg help to dry the feet as well as control mastitis (see p. biotin/day from calving produced a significant improve- 391).

ment in white line lesions at 100 days of lactation. In a more extensive split herd intervention study involving

Floor surfaces: These should not be too rough, stony or over 1100 cow lactations in five UK dairy herds, sup-have broken concrete, all of which could damage the

plementation with 20 mg/day biotin significantly halved

corium. On the other hand, very slippery surfaces can

the incidence of lameness caused by white line lesions

lead to leg damage. An excellent demonstration that

(Hedges et al., 2001). Survival analysis demonstrated

cows do not like walking on concrete is shown in Plate

that supplementation needed to be given for 130 days

31.11. A strip of second-hand rubber belting, approxi-

before any difference in the two groups was seen (Fig.

mately 1.5 metres wide, has been laid along the centre

31.10). In addition, only 28 per cent of supplemented

of a concrete track which runs from a dirt yard to the

animals required a repeat treatment in the same digit,

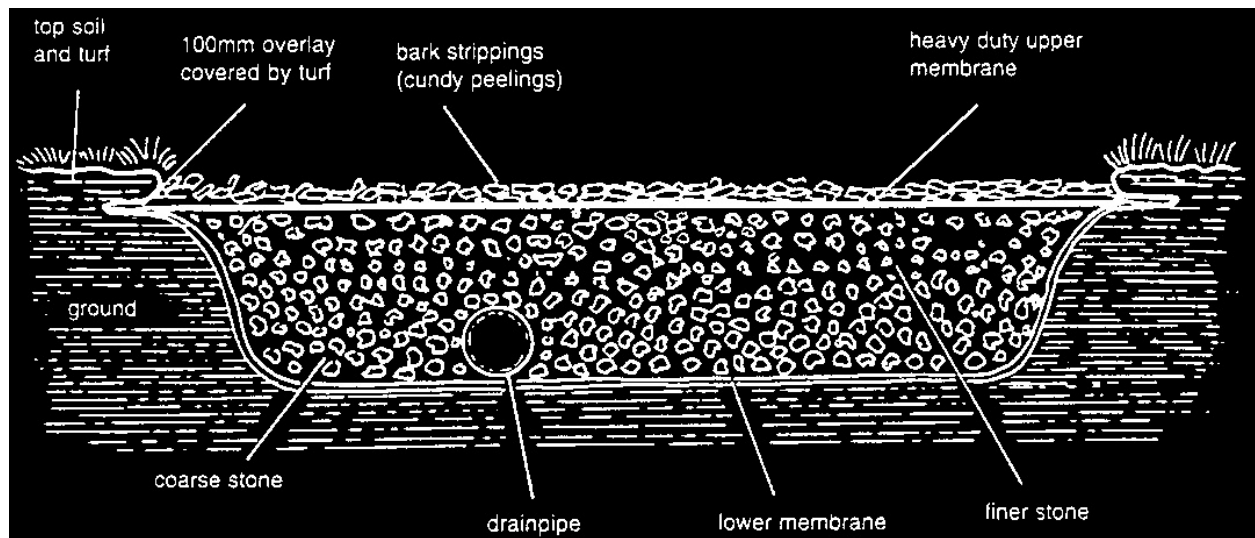
milking parlour at a dairy in California. Although the compared with 72 per cent of unsupplemented controls cows could walk anywhere they wished on the track, (Blowey et al., 2000c).

note how they all prefer to walk on the rubber belt. In Hagemester (1996) showed that animals receiving the UK, if cows are allowed to amble out to a field at 10 mg biotin daily had significantly fewer sole ulcers their own speed they will usually choose to do so by and less heel erosion than unsupplemented controls, walking on the soft earth of a grass verge, rather than and Lischer et al. (1996) reported a correlation between on stones (Plate 31.12). They even place their feet in

biotin supplementation and the rate of healing of exactly the same spot each time, making holes in the claw lesions. New horn formed more rapidly in those ground. This preference for a softer surface has led to cows supplemented with 20 mg biotin/day. A two-year the development of specific cow tracks. The ground is Canadian field study of 265 Hereford beef cows with excavated to 0.3 cm deep and 1.0–1.5 metres wide and

a high prevalence of vertical fissures (sandcracks) lined with a permeable geotextile road construction demonstrated that supplementation with 20 mg biotin/membrane which prevents sinkage of the track. A cow per day significantly reduced the prevalence of drainage pipe runs along the base, surrounded by a vertical fissures from 29.4 per cent to 14.3 per cent large aggregate, perhaps having fine stone on the top ($P < 0.05$; Campbell et al., 1996).

(Fig. 31.11). This is covered with a second special toughened membrane, which allows water to drain through but will not allow mud to rise up through it. Finally a General management layer of bark strippings, sometimes known as Cundy Many aspects of management have already been discussed in the housing and feeding sections. This section upper membrane to provide a comfortable walking will cover a few miscellaneous points relating to lame-surface for the cows. Tractors and other vehicles must



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Fig. 31.11

Cross-section of a cow track.

not use the track. Similar tracks may be constructed

Hoof wear: Both inadequate and excessive hoof wear

in gateways, around water troughs and in other

can cause problems. Heifers reared and housed in

areas where the ground gets badly poached. On very

totally bedded areas (straw, shavings or sand) may not

well drained land some farmers have constructed a very

receive sufficient hoof wear. The toes become over-

simple track by scraping away the topsoil and then

grown, the foot rotates caudally and the corium may

unrolling a large round straw bale onto the underlying

become damaged at the sole ulcer site. The provision of stone. Wet bales, too badly soiled for straw yards, could a lightly abrasive concrete feeding area is essential, both be used. The straw needs replacing approximately every two or three weeks, depending on the weather, but it horn production to produce the thick sole that is makes a good track and is certainly cheaper.

needed in the postpartum animal. At the other extreme, The influence of floor surface on white line disease is cows or heifers (and especially fresh calvers) that are interesting. It is commonly stated that cows become made to walk long distances on gravel or even concrete lame because of a specific type of stone or gravel in a roads can wear their soles so thin that they are easily track, particularly if sharp flints are present. However, compressed by thumb pressure. This syndrome is beef cattle could probably walk along the same track common in the grazing systems of Australia, New Zealand and Uruguay. Heifers reared on pasture are

gests that it is the weakening of the white line which is often introduced into the dairy herd immediately after the critical factor and not the sharpness of the stones. calving and expected to walk long distances to and from grazing. This produces maximal wear at a time when

Rough handling: This has also been shown to have an hoof growth is minimal, and soft soles with subsequent effect. A survey of farms showed that cows which were bruising of the fragile postpartum corium leads to an forcibly rushed along farm tracks by a herdsman, dog increase in white line disease and toe ulcers (see 'neg- or tractor had a far higher incidence of lameness than ative net growth', p. 415). If heifers are exposed to con- farms where the cows were allowed to walk along at crete or some other hard surface for a few weeks before their own speed. This was presumably because in the calving, this can stimulate an increased thickening of latter case they chose their own footing, thus avoiding the sole and the severity of the syndrome is reduced. bruising to the sole and corium. Similarly, if heifers are A similar 'soft sole' syndrome is seen in young bulls

introduced into a highly competitive situation where introduced to work in a dairy herd, particularly if the they are forced to make many sudden turning move- bulls are large and do not use the cubicles. The soles of ments, this both increases hoof wear and forces the wall their hind feet, especially at the toe, can wear down to away from the sole, leading to an increase in white line the corium. Ideally, bulls in cubicle systems should be defects. When walking, cows should be allowed to move rested in a straw yard, for example cubicles by day and at their own pace. Their heads will be down as they look a straw yard by night, or alternate weeks in cubicles and at the ground surface in front of them for a safe and soft straw yards. On a daily basis bulls soon learn which is footing. If cows are being driven too fast, or if they are to be their period of lying and compensate for the too crowded, then their heads will be high, they cannot cubicles by lying in the straw yards for long periods of see their footing and increased lameness may result. time.

Conformation: Conformation affects the incidence of

Slurry heel (heel horn erosion, heel necrosis)

lameness, which is therefore influenced by genetics and

In feet which have been exposed to slurry over a long

breeding. Bulls should be chosen to give a good depth

period of time, the smooth, soft and pliable heel horn

of heel and a good upright angle of the front wall, as in

often becomes black and pitted and in more extreme

Fig. 31.3.

cases totally eroded, especially in the axial area (Plate

31.13). Although perhaps not too serious externally,

Foot trimming: The final management factor that

slurry heel causes important internal changes. Removal

influences the development of lameness is routine foot

of weight bearing at the heel allows the foot to rotate

trimming. If parturition is a major stress period for

backwards, thereby predisposing to sole ulcers. In

the development of coriosis, then feet need to be in

advanced cases the flexor tuberosity no longer has

optimum shape at calving in order to minimize this

adequate support and as a result penetrates the horn

effect. This is achieved by hoof trimming at drying off, of the sole. The corium will be pinched as the cow and at any other time when they are overgrown. walks and sole ulcers develop.

Deep slurry heel fissures may develop into an under-run sole, which may be further complicated by a secondary digital dermatitis. Erosion of the heel also predisposes to white line defects, and by predisposing to a caudal rotation of the claw, to sole ulcers.

Other causes of foot lameness

There is no one single cause of heel horn erosion.

As it is almost exclusively a condition of housed cattle,

Hoof disorders:

simple erosion by slurry seems to be the most probable factor. Others have suggested the involvement of

- Foreign body penetration

specific bacterial infections such as *Dichelobacter nodosus*

- Slurry heel

(Toussaint-Raven, 1985) or *Prevotella* (*Bacteroides*)-

- Vertical fissure (sandcracks)

melaninogenicus (Greenough, 1987). Coriosis/laminitis

- *Hardship lines and laminitis*

may also be involved, leading to poorer quality heel

- *Horizontal fissure*

horn, which is then more susceptible to the corrosive

- *Broken toe*

effects of slurry.

- *Axial wall fissures*

Skin disorders:

Treatment and control

- *Interdigital necrobacillosis (foul of the foot or footrot)*

Deep fissures may need to be investigated to eliminate

- *Digital dermatitis (hairy warts)*

(and correct) an under-run sole, but if the lesion is

- *Interdigital skin hyperplasia (corns, growths or relatively superficial it is best left and treated topically. tylomas)*

Removal of heel horn is commonly contraindicated,

- *Mud fever*

because to do so must inevitably lead to a caudal rota-

tion of the claw and increase the risk of sole ulcers.

Bone and joint disorders:

Hygiene is the best control measure, maintaining the

- *Pedal bone fracture*

feet in a clean and dry environment, with regular foot-

- *Necrosis of the apex of the pedal bone*

baths. Lime, used in cubicles for mastitis control, also

- *Pedal arthritis*

acts as a disinfectant and drying agent.

Vertical fissures (sandcracks)

Hoof disorders

Vertical fissures occur as a result of damage to a small area of the periople and underlying coronary band.

Foreign body penetration of the sole

Horn formation is disrupted at that point, although the

Typical foreign bodies are stones (especially sharp adjacent horn continues to grow. This leaves a gap (the flints), nails (particularly those with flat heads), fragments of wood, glass or tin and occasionally even the point of disrupted production (Plate 31.14). In North

sharp root of a cast tooth will penetrate the sole. After America vertical fissures are commonly seen in both removal of the foreign body it is essential to provide beef cattle and older dairy cows kept in sand lots where drainage by opening the sole at the point of entry and the combination of age, sand, wind and dry weather removing all under-run horn.

removes the protective periople. Vertical fissures can
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also be a result of a digital dermatitis infection on the the interdigital space. White line lesions running coronary band. A two-year Canadian field study of 265 obliquely along the axial wall are often referred to as Hereford beef cows with a 37 per cent prevalence of axial wall fissures (Plate 31.15). Recently there has been sandcracks demonstrated that supplementation with an apparent increase in incidence (Vermunt, 1998). The 10 mg/day of biotin significantly reduced the incidence cause is unknown, but wet conditions underfoot have of sandcracks from 29.4 per cent to 14.3 per cent been suggested. Due to their position in the interdigi-

(Campbell et al., 1996).

tal space they are quite difficult to pare and lesions
For treatment, the full length of the fissure should be
which damage the coronary band can lead to perma-
opened using the curved tip of the hoof knife. Even a
nent vertical fissures. Digital dermatitis lesions affect-
small abscess can cause intense lameness. If the fissure
ing the coronary band in the interdigital space may be
is large, a block should be applied to the sound claw.
a further factor.

Hardship lines

Skin disorders

Any disruption in horn formation may leave a groove,
sometimes referred to as a 'hardship groove' (Gree-

Interdigital necrobacillosis

nough et al. , 1997), encircling the hoof wall. These are (foul of the foot, foot-rot)
the result of coriosis/laminitis. Inflammation of the
laminae is said to lead to massive pressure under the
This is a bacterial infection of the skin of the interdigi-
hoof wall, causing the wall to push forward and the toe
tal cleft. Two organisms are primarily involved, namely

to lift. The eventual effect is a concave front wall with

Prevotella (Bacteroides) melaninogenicus and *Fusobac*-numerous hardship lines.

terium necrophorum. Cultural studies have suggested

that *P. melaninogenicus* should be further subdivided

into *Porphyromonas asaccharolytica* and *Prevotella*

Horizontal fissures (sandcracks)

species (Berg & Weaver, 1994). *Dichelobacter nodosus*

Animals severely ill, for example with mastitis, metritis

may also be involved. *F. necrophorum* is an obligate

or any toxic condition, may undergo a total cessation

anerobic Gram-negative bacterium found in the intes-

of horn formation. When horn production resumes,

tinal tract of both cattle and sheep and it is widespread

instead of a hardship groove, there may be a horizontal

in the environment, surviving for up to ten months. The

fissure encircling the hoof wall. Initially this may cause

A and AB biotypes produce a potent exotoxin which

no problem, but as the defect moves distally towards

causes a suppurative necrosis and depresses phagocy-

the toe it loses its support and attachment at the heel.

toxis, and a degree of synergism may exist between

The protruding 'thimble' of horn is then able to rotate

B. melaninogenicus and F. necrophorum. Injury to the on the underlying corium, causing pinching, pain and

interdigital skin may be required to allow entry of infec-

lameness. Both claws of all four feet may be affected.

tion. This is usually due to stones, stubble, kale stems,

The date of the illness can be calculated by measuring

hardened dung in the interdigital area, sticks, very dry

the distance from the coronary band to the fissure and

pasture, rough flooring, etc. Damage will be easier when

dividing this by the rate of horn growth, namely 5 mm

the skin is soft due to continued wetting. In some

per month.

instances the damage may be caused by penetration

For treatment, the loose 'thimble' of horn is removed.

of spirochaete organisms. Reservoirs of infection may

If the corium is extensively exposed on one claw, a

exist in wet, dirty areas such as the gateways to fields

block should be applied to the other claw if it is sound.

and around water and feed troughs. It is also possible

However, not all horizontal fissures lead to lameness.

that mild digital dermatitis or interdigital dermatitis

Some simply grow to the toe and are shed naturally. It

might be the cause of the initial damage.

is only necessary to trim the foot if the cow is lame. A

The condition has a worldwide distribution in both

cow with one long claw and one short, due to the hori-

dairy and beef cattle. Usually only one or a few animals

zontal fissure fragment having been shed from one claw

are affected at one time. However, the disease appears

only, is sometimes referred to as having a 'broken toe'.

to be contagious with the incidence increasing in wet

humid weather and interdigital skin damage. Epizootics

can arise when cattle are moved to a new environment

Axial wall fissures

or mixed together. The most probable source of infec-

The white line junction runs along the abaxial wall from

tion is discharge from the feet of infected animals. The

the heel to the toe, caudally along the axial wall at sole

same animal can be affected repeatedly and as suscep-

level and then passes obliquely in a proximal caudal

tibility only changes slightly with age, acquired immu-
direction along the axial wall to the coronary band in
nity to the bacteria appears to be poor.

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Clinical signs

*physical damage to the interdigital cleft. Frosted
ground can also produce damage.*

Typically 'foul' is seen as a sudden onset lameness, more

*Disinfectant footbaths (formalin, copper sulphate,
commonly in the hind foot, and it is characterized by
etc.) provide excellent control and are discussed on p.*

*bilateral swelling above the coronary band of both
432. Antibiotic footbaths have been used in the control
claws, often with the digits forced apart. Pyrexia is
of superfoul where digital dermatitis may be involved.*

*common. Initial erythema of the interdigital skin pro-
gresses to a fissure and subsequent slough of the epi-
dermis to expose the underlying necrotic dermis (Plate*

Digital dermatitis

31.16). Unless treated, lesions may progress to an infec-

This is a bacterial skin infection that primarily affects

tion of the navicular bursa, the flexor tendon sheath or the epidermis. First reported in Italy (Cheli & Mortelaro, 1974), digital dermatitis now has a worldwide distribution. Figures of frequency of occurrence vary virulent condition known as 'superfoul'.

enormously depending on whether the incidence is recorded as a cause of lameness or whether prevalence

Treatment

of lesions is recorded. Many cows show low-grade lesions. For example, Laven (1999) found a lesion

A wide range of parenteral antibiotics including prevalence of 41 per cent in 1810 hind feet examined, penicillin, oxytetracycline, sulphonamides, tylosin, tilmicosin, ceftiofur and cefquinome are all effective. The UK, digital dermatitis now represents approximately 30 per cent of all cases of lameness, which is probably why of the initial lesion. The foot should always be lifted and

the overall incidence of lameness in cattle has not examined to remove necrotic tissue and any predisposed since digital dermatitis was first reported in ing foreign body and a topical antibiotic applied. Local 1987 (Blowey & Sharp, 1988).

treatment must improve the rate of healing and reduce the spread of infection into the environment.

Aetiology

Superfoul

Histological studies were first to implicate spirochaetes

(Blowey et al., 1992; Read et al., 1992). These had a This is a peracute form of interdigital necrobacillo-predilection for keratinized cells and produced a toxin

sis with a poor response to treatment unless aggressive

which is keratolytic. A possible association between

therapy is instigated in the very early stages (David,

Borrelia burgdorferi and other Treponemes has

1993). Cultural examinations have indicated that the

been suggested. Digital dermatitis-infected cows had a

same organisms are present, although most cases are

higher seropositivity to B. burgdorferi strain B31 and seen in herds concurrently infected with digital der-Treponemes 1-9185MED and 2-1498 than control cows

matitis. An 'invasive spirochaete', similar to both digital

(Demirkan et al., 1999). Other workers have suggested dermatitis and the spirochaete identified in peracute the involvement of *Dichelobacter nodosus* and Campy-foot-rot in sheep, has been seen in lesions of superfoul.

lobacter faecalis

The main difference between foul and superfoul is the While spirochaetes can often be found in large speed of onset and the severity of the lesions. Necrosis numbers at the junction of viable and necrotic tissue of the interdigital skin may be seen within 12 hours, with layers, they are often difficult to culture. However, succ-deep necrotic fissures into the dermis within 24 hours. cessful culture techniques have been reported. Two Early and aggressive therapy is therefore essential. The different spirochaetes have now been demonstrated. treatments used are similar to conventional foul, but at One is a long, filamentous organism 12 mm long and 3 a higher dose and for a longer period. Some benefit has mm wide and the other a short spirochaete 5 to 6 mm been reported from the use of local anaerobic therapy long and 0.1 mm wide. They are also different on enzy-

such as clindamycin, spiramycin and metronidazole.

matic analysis (Walker et al., 1995). The most probable cause is an organism with characteristics most consis-Prevention and control: Environmental hygiene and

tent with the genus *Treponema*. Potential isolates sug-footbathing are the main control measures. If outbreaks

gested are *T. phagedenis*, *T. vincentii* and *T. denticola*.

occur, passages should be scraped with increased fre-

quency, more bedding can be used, and if cattle are

Epidemiology

outside, they should be moved to a different paddock.

Attention should be given to gateways, areas around

There is considerable confusion in the literature

water troughs and other gathering points to minimize

between incidence of disease seen as lameness and

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prevalence of lesions, with few studies having examined

digital dermatitis (PDD) or 'hairy warts', although they

large numbers of feet at a single point in time. Laven

are now becoming increasingly common in the UK.

(1999) reported a 41 per cent lesion prevalence but did

Although the heel area is the most common site,

not relate this to lameness incidence. Hedges et al. lesions of digital dermatitis may also be seen in the (2000), in a study of over 1100 cow years in five UK interdigital space, on the sole following a sole ulcer, dairy herds, found that digital dermatitis ranked white line disease or some other lesion exposing the approximately equal to sole ulcers and white line corium, adjacent to the accessory digits or at the ante-disease, and accounted for around 20 per cent of all rior aspect of the interdigital space adjacent to the cases of clinical lameness examined. Lesions are most coronary band. Lesions at this point are particularly commonly seen in housed cattle, especially dairy cows, significant because they can damage the germinal layers but heifers and beef animals can also be affected. of the hoof wall to produce a vertical fissure. In chronic Lesions in hind feet are more common than in front feet cases granulation tissue may protrude from the fissure and there would appear to be relatively little immunity, and because the corium is inflamed exostoses develop because affected cattle are more likely to have lesions

on the anterior wall of the pedal bone. Vertical fissures

on both hind feet (Laven, 1999) and reinfections occur resulting from anterior digital dermatitis are currently

in the same cows year on year, suggesting recrudes-

a common indication for digit amputation.

cence of latent infections. Environmental hygiene is the

main factor influencing disease, with the most severe

Treatment

outbreaks occurring in housed cattle in winter, although

Most lesions regress following cleaning and topical

the increase in lesions and lameness seen in early lac-

application of antibiotics, although repeated treatments

tation (Laven, 1999) could also be a reflection of peri-

may be necessary. Lameness resolves but it is likely that

parturient immune suppression. The highest incidence

lesions persist to recrudesce at a later date. A wide

of disease is seen in the early lactation period and this

range of topical antibiotics have been used including

is associated with animals showing a high prevalence of

lincomycin, combined lincomycin and spectinomycin,

lesions prepartum. Disease incidence is higher in lac-

oxytetracycline, erythromycin, tylosin and tiamulin.

tating than dry cows. Suggested causes include lactating

Concurrent parenteral therapy is indicated for anterior

cows spending longer standing (for milking, moving and

lesions to reduce the risk of vertical fissure develop-

feeding), higher bacterial levels in slurry from higher

ment. Frequency of application varies with lesion sever-

concentrate diets, faeces are not as dry and stocking

ity, but ideally the lesion should be cleaned and treated

density is often higher.

for at least two consecutive days. Severe lesions should

be bandaged with topical antibiotics for 3–4 days.

Treatment of PDD (hairy warts) is more difficult

Clinical signs

because the organism is protected by 10–20 mm of kera-

tinized epidermis. Ideally these lesions should be ampu-

The earliest lesions of digital dermatitis are seen as a

tated at lower epidermal level, under local anaesthesia,

dry, painless, grey encrustation of hyperkeratotic mate-

and an antibiotic dressing applied for three to five days,

rial, often lining the caudal edge of the interdigital

although an antibiotic dressing alone may be sufficient.

pouch. The pouch is most probably the major reservoir

Single topical treatments and footbaths are not effective

of infection in carrier animals, although similar encrus-

against PDD. Antibiotic and antiseptic footbaths are an

tations can be seen along the junction of the heel horn

important aspect of both treatment and control.

and skin. More advanced lesions, and those sufficiently

painful to cause lameness, are typically seen as a moist,

Prevention and control

light greyish-brown area of exudate, 10–20 mm in diam-

eter, often with matted hairs and encircling the inter-

Environmental hygiene is the most important control

digital cleft (Plate 31.13). Cleaning the lesions reveals a

measure and factors which produce a cleaner and/or

red, raw area of epidermitis, very painful when touched

drier foot environment will lower disease incidence.

and having a characteristic fetid odour and a granu-

Cubicle and feed passages should be scraped at least

lar ‘strawberry’ appearance. Advanced lesions create

twice daily and accumulations of stale slurry such as

superficial epidermal erosions and irradiate across may be found in cross passages and around water the heel bulbs, but ulceration into the dermis is rare: troughs should be avoided. Feet should be kept as most lesions retain an epidermal covering. Longstand-dry as possible by good drainage, ample bedding and ing, chronic lesions have a proliferative appearance, the use of lime in cubicle beds (which also helps in with marked epidermal hypertrophy and hyperplasia mastitis control). Footbaths are an important control (Plate 31.17). These lesions are more prevalent in North measure, although antibiotics applied as a foot spray on America where they are referred to as papillomatous a whole-herd basis have also been effective.

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Interdigital dermatitis (IDD)

grade digital dermatitis lesions are so common that it is always worth applying a topical antibiotic. Larger Considerable confusion exists in the literature over lesions require surgical amputation, although many whether IDD is a separate entity or whether it is a com-

recur. Regular footbathing through formalin is also said to resolve many lesions.

with stages of slurry heel. Many of the lesions attributed to IDD contain the spirochaete of digital dermatitis. It is not generally considered to be a cause of

Mud fever

lameness, but large numbers of cattle are said to be affected and this predisposes to other lesions such as and muddy conditions. The lower leg becomes slightly digital dermatitis, foul, slurry heel and interdigital swollen, with hard, dry and flaking skin. There may be hyperplasia. Dichelobacter nodosus is said to be a hair loss and even bleeding if the skin cracks.

common organism involved, but others dispute whether For treatment, the legs should be thoroughly washed and dried followed by application of a greasy antiseptic ointment or a teat spray which contains a high level

Clinical signs

of emollient. As the organism *Dermatophilus* may be involved, three days of injectable antibiotic (penicillin) Many cases are mild, leading to irritation and hyper- may also help (see p. 886; Blowey & Weaver, 2003). aemia of the interdigital skin, with lesions spreading onto the heel bulb. Fissures and necrosis of heel horn can result, with coronary band lesions said to disrupt

Bone and joint disorders

hoof wall formation and lead to vertical fissures.

The latter is also characteristic of anterior digital

Fracture of the pedal bone

dermatitis

Bulling activity, with the mounting cow falling back heavily onto hard or rough concrete, is the most

Control

common cause of a fractured pedal bone. Bones weakened by age, fluorine poisoning or a foreign body penetrating the sole of the hoof may be at greater risk of control.

fracture. Typically it is the inner claw of the front foot that is involved and by adopting a cross-legged stance the cow transfers her weight onto the sound lateral

Interdigital skin hyperplasia (see p. 182)

claw. However, the stance alone is not sufficient to diag-

Also called corns, tylomas, fibromas and just 'growths',

nose fracture of the pedal bone. Cows with ulcers in

interdigital skin hyperplasia is an overgrowth of the

both inner claws will adopt the same position. Most

natural skin fold adjacent to the axial hoof wall. There

animals heal well if a block is applied to the sound claw.

is a fold of interdigital skin adjacent to both the medial

and lateral claw, and the hyperplasia may develop from

Apical necrosis of the pedal bone

either side. It is an overgrowth of skin with gross hyper-

trophy of the epidermis. The term 'fibroma' is therefore

In a few cows, what initially appears to be a standard

technically incorrect. The lesion is hereditary in certain

white line abscess at the toe sometimes fails to heal,

heavier breeds of cows, especially beef breeds. In other

even though it may have been treated thoroughly. At

cases it is secondary to chronic irritation of the inter-
the second examination there will probably be a char-
digital skin, for example due to low-grade foul, digital
acteristic foul odour and even with further extensive
dermatitis or simply impaction with dirt. Cows walking
removal of under-run tissues the toe fails to heal (Plate
over rough surfaces, leading to excessive splaying of
31.18). In such cases the apex of the pedal bone has
the claws, may show an increased incidence. Lameness
become infected (osteomyelitis). The negative net horn
occurs either when the lesion reaches such a size that it
growth (p. 415) which occurs in the periparturient
is compressed by the claws during locomotion or when
heifer, leading to erosion of toe horn, can be an exac-
it becomes secondarily infected, for example by digital
erbating factor, especially if housing coincides with par-
dermatitis or foul.

turition. A similar condition is seen in weaned beef
Mild cases can be treated by removing excess horn
calves that continually run around the pen when intro-
from the axial wall and the axial surface of the sole.

duced into feed lots.

Many lesions regress spontaneously when they are no

Treatment must involve removal of all necrotic bone.

longer traumatized during locomotion, although low-

Some clinicians report success with aggressive debride-

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ment of the bone, but this has not been this author's

generally results in a good response. There is an

experience. Provided the coronary band remains intact,

additional requirement to redress the lesion regu-

removal of the necrotic toe and the adjacent wall and

larly, however, and additional weightbearing on the

sole with an embryotomy wire may result in full reso-

remaining digit may lead to early culling. If ampu-

lution. Ideally, a radiograph is needed to identify the

tation can be performed at drying off, then a single

extent of the necrosis, and as many pedal bones are

dressing change after two to three days may be suf-

already severely eroded (Plate 31.19), total amputation

ficient, with the second dressing fully removed three

of the digit may be the best option (see p. 1119).

to four days later. Most cases heal remarkably well (see also p. 1119).

Deep pedal infections

General treatment and control

Severe or neglected cases of sole ulcers, white line of foot lameness

disease, interdigital necrobacillosis or foreign body penetration can lead to infection tracking into deeper

Footbaths

tissues. Complications must be suspected when severe

Footbaths are an excellent preventive measure for lameness follows treatment of an apparently standard lameness and cows should be walked through once a lesion, when swelling appears above the coronary band of week during the winter housing period. Solutions of the affected digit or when pus can be expressed from a 5–10 per cent formalin or 2.5 per cent copper sulphate sinus when massaging the digit. In any of these instances or zinc sulphate have been used, as have a variety of it is the author's opinion that immediate and aggressive other disinfectants. The main objective of a footbath

parenteral therapy should be instigated, for example oxy-
is both to clean and disinfect the foot and in so doing
tetracycline (2 g twice daily), tylosin or penicillin. If
it should help to reduce the incidence of conditions
swelling of the coronary band was the only apparent
such as:

lesion, this may be sufficient to effect resolution.

Deep infections may produce a retro-articular

- Interdigital necrobacillosis

abscess (namely in the heel caudal to the deep flexor

- Slurry heel and heel erosion

tendon),

an infection of the navicular bursa,

- Interdigital skin hyperplasia ('tylomas' or 'corns')

osteomyelitis of the navicular (distal sesamoid) bone

- Digital dermatitis

or a purulent coronopedal arthritis. A range of treat-

Formalin also has a drying action on the foot.

ment options is available, all of which involve aggres-

However, it is unpleasant to handle and its use is not

sive antibiotic cover, and most will require a block

permitted in some countries. Similarly copper sulphate applied to the sound claw, thus minimizing weightbearing on the affected claw (p. 432). The option chosen by authorities because of the risk of pollution. Often two baths are used, the first containing water to wash and clean the feet. There is then a raised concrete strip to

- Radical fenestration of the sole or wall to provide drain off excess water before the cows walk into the drainage, followed by regular flushing of the lesion

second bath containing the active chemical, as shown in Fig. 31.12. The liquid in the bath needs to be only 80–100 mm deep, as only the claws need to be

- Insertion of a drainage tube, the upper end of which is strapped to the hock to allow ease of flushing at lead to damaged skin on both feet and teats.

milking (Plate 31.20). The lower end is closed. A
For best effects cows should have their heels cleaned
practical method of siting this tube is to use a trocar
by a water spray as they enter the milking parlour.
and cannula to follow the sinus tract into the foot
Excess water then drains off during milking, after which
and then exit above the coronary band.

the cows should exit through a footbath and into a clean

- Permanent arthrodesis can be created by drilling
environment, with the whole herd being bathed on the
through the pedal joint and curettage of the articu-
same day to avoid cross-contamination. If done care-
lar joint surfaces.

fully, a single passage through an antibiotic footbath

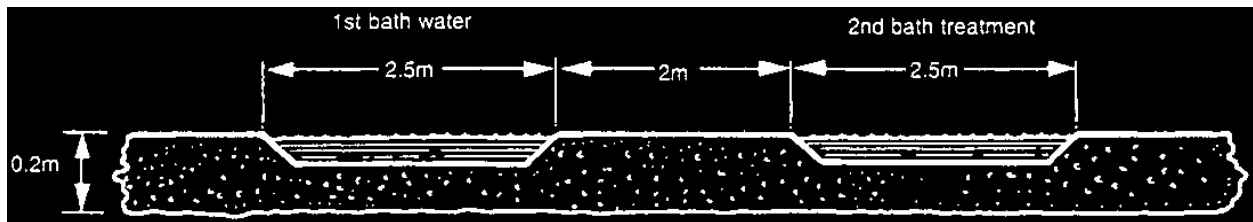
- A surgical approach, opening the joint from the
will dramatically reduce the incidence of lameness due
heel, is also available.

to digital dermatitis in as little as 24 hours. Unfortu-

- Digit amputation is relatively simple. It can be per-
nately it does not eliminate infection from a herd.

formed in the standing animal under regional intra-

A range of antibiotics have been used to treat and venous anaesthesia and provided that there is no control digital dermatitis, and at varying concentrations. major ascending infection of the flexor tendon it In most countries none are licensed for this use. The



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Fig. 31.12

Two footbaths in series.

following are the most common: oxytetracyclines at disinfectants, a beta-ionine terpene compound and a 4–6 g/litre, lincomycin at 1.0 g/litre, combined triplex of copper, a peroxy compound and a cationic lincomycin/spectinomycin at 33 g lincomycin plus 66 g agent. The latter was found to be at least as effective as spectinomycin in 150 litres, erythromycin at 0.6 g/litre, oxytetracycline.

tiamulin at 0.5 g/litre and tylosin at 1.3 g/litre. The dosage and frequency of bathing required depends on

Nursing, dressings and footblocks

the severity of clinical signs, with a single or perhaps two passages through a fresh solution once every four

Nursing

weeks originally being the most common recommen-

Lame cows obviously find walking difficult. They are

dation in the UK. (This was not effective on the hairy

much less able to compete with the rest of the herd,

warts seen in North America, however.) Although this

especially if they are at the lower end of the social

initially appeared reasonably effective, by the late 1990s

dominance scale, which is commonly the case with lame

all antibiotics appeared to be losing their effectiveness

heifers. In addition, they find it difficult to manoeuvre

and revised concepts of footbathing were considered

in and out of the cubicles. Cubicles are not easy to use

(Blowey, 2000). At that stage footbaths were instigated

at the best of times and if a cow is not fully mobile

only when lameness appeared and usually when a

cubicles are even more difficult to negotiate. This small but significant proportion of the herd was lame. results either in lame cows spending longer time standing, Clearly at this stage lesions have progressed to quite an advanced stage and a single treatment is likely to be less effective. they spend a long time lying and do not feed sufficiently,

effective. resulting in marked weight loss. Ideally lame cows If footbaths were instigated at an earlier stage, before should be transferred into a straw yard, where it is lesions appeared and especially if dry cows were treated easier for them to lie down and get up and where there is less competition for food. Many herdsmen have to be more effective. A system of continual daily foot-bathings in 2.5–5 per cent formalin for periods of two straw yards results in an increase in yield in as little as 24 hours, especially in heifers.

in reducing lesion severity, although even this does not totally eliminate infection from a herd and the treatment has to be repeated after intervals of three to four

Foot dressings

weeks. Some herds now use footbaths on a continual

Opinions vary over the need to apply a bandage and daily basis. Although formalin and other disinfectants

dressing to an exposed corium, for example following do not penetrate the epidermis as well as antibiotic

the trimming of sole ulcers or white line lesions with solutions, regular use eventually leads to drying and

under-run sole. There appears to be a minimal risk of sloughing of superficial keratinized tissue. The black

infection from the environment penetrating the corium, debris of hairy warts can be peeled off after two weeks

even if cows with extensively under-run soles are

of treatment, although in a proportion of cases charac- allowed to walk out into the slurry, and it is surprising

teristic granular areas of active dermatitis may still be how quickly the exposed corium becomes covered by a

visible beneath. Individual severely affected cows can

layer of new horn. On the other hand, there are several
be stood in formalin for 20–30 minutes and this also
potential disadvantages in applying a dressing, any of
results in resolution of lesions. The system has also been
which may retard healing. These include the following:
effective against combined outbreaks of foul and digital
dermatitis.

- Unless changed almost daily, the dressing will

A variety of other footbath chemicals have been used
absorb infection from the environment and impede
including acidified ionized copper sulphate, hydrogen
drainage. Impeded drainage may allow infection to
peroxide/peroxyacetic acid or quaternary ammonium
spread, producing further under-run horn.

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- Dressings prevent exposure to air and air often
improved by forcing the claws apart with a small roll of
promotes healing.

paper towel. With the PVC shoe the glue is mixed in

- The presence of a bulky dressing on the sole means
the shoe until it forms a paste. When the paste is just

that the affected sole becomes weightbearing. This starting to set the shoe should be pushed as far back could make sole ulcers worse and cannot be beneficial in the production of new horn.

the shoe or block supports the heel, otherwise the cow

- Astringents, sometimes used to reduce granulation rotates backwards onto the sound claw, leading to dis-tissue from a sole ulcer, may damage the corium comfort and very rapid wearing of the block. Although and hence discourage the formation of the new some authors recommend removing blocks after two or horn, which is so badly needed to cover the sole.*

three weeks to retrim the sound claw, most clinicians consider this unnecessary. On average, PVC shoes have Few clinicians now use dressings on uncomplicated been shown to remain in place for over two months, sole ulcers or white line abscesses and most consider allowing ample time for the new sole to reform (Blowey that this results in improved healing. Dressings are et al., 1999). The PVC shoes can easily be removed by

needed following amputation of a digit, amputation of clipping around their outer wall with hoof clippers.

large interdigital skin hyperplasia lesions, treatment of extensive digital dermatitis and occasionally to control haemorrhage.

References

Footblocks

Arkins, S. (1981) Lameness in dairy cows. Parts I and II. Irish The use of a block applied to the sound claw is an excel-Veterinary Journal, **35**, 135–40; 163–70.

lent practice as it both promotes healing and con-

Bell, E.M. & Miller, A.M. (1977) Lameness in cattle. Interim siderably improves the welfare of the cow. A variety of

report, Edinburgh School of Agriculture.

devices are available, for example:

Berg, J.N. & Weaver, A.D. (1994) Bacterial aetiology of diseases in the foot rot complex: recent research and

- Tie-on shoes and boots

nomenclature changes. In Proceedings of the International

- Nail-on blocks

Symposium on Disorders of the Ruminant Digit, Banff,

- Blocks and shoes which are glued on

Canada, pp. 51–7.

Blowey, R.W. (1990) A simple treatment of deep foot infec-

Tie-on shoes are the least popular, because they are

tions in cattle. *Veterinary Record*, **127**, 515.

difficult to fix and by encasing the whole foot keep it

Blowey, R.W. (1998) In *Cattle Lameness and Hoofcare*,

damp and can retard healing. They also do not reduce

An Illustrated Guide. Old Pond Publishing, Ipswich.

weightbearing on the affected digit as effectively as

Blowey, R.W. (2000) Control of digital dermatitis. *Veterinary other methods*.

Record, **146**, 10.

Nail-on blocks are used successfully in skilled hands,

Blowey, R., Girdler, C. & Thomas, C. (1999) Persistence of although the sole of the claw to be blocked needs to be

foot blocks used in the treatment of lame cows. *Veterinary flat*. They are cheap and fast to apply, but they do not

Record, **14**, 642–3.

remain in situ for as long as the glue-on blocks: they Blowey, R.W., Hedges, V.J., Green, L.E. & Packington, A.J.

(2000c) The effect of biotin supplementation on the treat-

may move slightly and occasionally create sole ulcers

ment of white line lesions in dairy cows. In *Proceedings of under the rear edge of the block*. The nail holes must

the 11th International Symposium on Disorders of the Rumi-

also weaken the sound claw.

nant Digit, Parma, Italy, September 2000.

Wooden blocks, rubber blocks and PVC shoes, all of

Blowey, R.W., Ossent, P., Watson, C.L., Hedges, V.J.,

which can be glued onto the sound hoof, are the most

*Green, L.E. & Packington, A.J. (2000a) Possible distinction popular products.
The PVC shoe (Plates 31.7 and 31.20)*

between sole ulcers and heel ulcers as a cause of bovine

gives the best support because it is glued to both the

lameness. Veterinary Record, 147, 110–12.

wall and the sole, and weightbearing is therefore trans-

*Blowey, R.W., Phipps, R., Jones, A.K. & Barringer, A.J. (2000b) ferred to the wall,
which is the correct weightbearing*

A comparison of the effects of high fibre and high starch

structure. The glue also sets more rapidly, thus facilitat-

diets on hoof lesion score in multiparous dairy cows. In

ing ease of application, especially in colder climates.

Proceedings of the 11th International Symposium on

*Disorders of the Ruminant Digit, Parma, Italy, September However, on cows
with very large feet it may not be*

2000.

possible to push the shoe sufficiently far back towards

Blowey, R.W. & Sharp, M.W. (1988) Digital dermatitis in dairy the heel, and the foot can rotate caudally, leading to

cattle. *Veterinary Record*, **122**, 505–8.

discomfort.

Blowey, R.W. & Weaver, A.D. (2003) In A Color Atlas of Dis-For all glue-on blocks, the sound claw should be

eases and Disorders of Cattle. Mosby, London.

scraped totally clean and dry with a hoof knife, avoid-

Blowey, R.W., Sharp, M.W. & Done, S.H. (1992) Digital der-

ing even finger contact. Access to the inner wall can be

matitis. *Veterinary Record*, **131**, 39.

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Booth, J.M. (1989) Lameness and mastitis losses. Veterinary of biotin on lameness in dairy cows. *Journal of Dairy*

Record, **125**, 161.

Science, **84**, 1969–74.

Brizi, A. (1993) Bovine digital dermatitis. *The Bovine Practi-Hedges*, V.J., Blowey, R.W., Packington, A.J. & Green, L.E.

tioner, **27**, 33–7.

(2000) A longitudinal field study of the incidence and loca-

Campbell, J., Greenough, P.R. & Petrie, L. (1996) The effects tion of specific causes of lameness on claw health in dairy

of biotin on sandcracks in beef cattle. In *Proceedings of the cows and the effect of biotin supplementation*. ISVEE,

9th International Symposium on Disorders of the Ruminant

Colorado, August 2000.

Digit, Jerusalem, p. 29.

Hirst, W.M., French, N.P., Murray, R.D. & Ward, W.R. (2000) Cheli, R. & Mortellaro, C.M. (1974) La Dermatite digitale del The importance of first lactation lameness as a risk factor

bovino. In Proceedings of the 8th International Meeting on for subsequent lameness. In Proceedings of the 11th Inter-Disorders of Cattle, Milan, Italy, pp. 208–13.

national Symposium on Disorders of the Ruminant Digit, Clarkson, M.J., Downham, D.Y., Faull, W.B., Hughes, J.W.,

Parma, Italy, September 2000, pp. 149–51.

Manson, F.J., Merritt, J.B., Murray, R.D., Sutherst, J.E. & Laven, R. (1999) The environment and digital dermatitis.

Ward, W.R. (1996) Incidence and prevalence of lameness in Cattle Practice, 7, 349–56.

dairy cattle. Veterinary Record, 138, 563–7.

Leach, K.A., Logue, D.N., Kempson, S.A., Offer, J.A., Ternent, Collick, D.W., Ward, W.R. & Dobson, H. (1989) Association

H.E. & Randall, J.M. (1997) Claw lesions in dairy cattle:

between type of lameness and fertility. Veterinary Record, development of sole and white line haemorrhages during

125, 103–6.

the first lactation. The Veterinary Journal, 154, 215–25.

Da Costa Gomez, C., Al Masri, M., Steinberg, W. & Abel, H.J.

Leech, F.B., Davies, M.E., Macrae, W.D. & Withers, F.W. (1960) (1998) *Effect of varying hay/barley proportions on micro-Disease, Wastage and Husbandry in the British Dairy Herd.*

bial biotin metabolism in the rumen stimulating fermentor

HMSO, London.

RUSITEC. In *Proceedings of the Society of Nutritional*

Leonard, N., O'Connell, J. & O'Farrell, K. (1996) *Effect of Physiologists*, No. 7, 14–28.

overcrowding on claw health in first calved Friesian heifers.

David, G.P. (1993) *Severe foul-in-the-foot in dairy cattle.*

Veterinary Journal, **152**, 459–72.

Veterinary Record, **132**, 567–8.

Lischer, C., Hunkeler, A., Geyer, A. & Ossent, P. (1996)

Demirkan, I., Walker, R.L., Murray, R.D., Blowey, R.W. &

The effect of biotin in the treatment of uncomplicated claw

Carter, S.D. (1999) *Serological evidence of spirochaetal*

lesions with exposed corium in dairy cows. In Proceedings

infections associated with digital dermatitis in dairy cattle.

of the International Symposium on Disorders of the Rumi-

The Veterinary Journal, **157**, 69–77.

nant Digit, p. 31.

Eddy, R.G. & Scott, C.P. (1980) *Some observations on the*

Livesey, C.T. & Flemming, F.L. (1984) Nutritional influences incidence of lameness in dairy cows in Somerset. *Veterinary on laminitis, sole ulcer and bruised sole in Friesian cows.*

Record, **106**, 140–4.

Veterinary Record, **114**, 510–12.

Esslemont, R.J. & Kossaibati, M.A. (1996) *Incidence of*

Livesey, C.T., Laven, R.A., Marsh, C. & Johnson, A.M. (2000) *production disease and other health problems in a*

Changes in cysteine concentration in sole horn of Holstein

group of dairy herds in England. Veterinary Record, **139**, cattle through pregnancy and lactation. In *Proceedings of*

486–90.

the 11th International Symposium on Disorders of the Rumi-

Esslemont, R.J. & Spincer, I. (1993) *Report No. 2. DAISY –*

nant Digit, Parma, September 2000, pp. 319–21.

The Dairy Information System. University of Reading.

McLennan, M.W. (1988) *Incidence of lameness requiring*

Green, L.E., Hedges, V.J., Blowey, R.W., Packington, A.J. & *veterinary treatment in Queensland. Australian Veterinary*

Schukken, Y.H. (2002) *The impact of clinical lameness on*

Journal, **65**, 144–7.

the milk yield of dairy cows. In Proceedings of the 12th

Midla, L.T., Hoblet, K.H., Weiss, W.P. & Moeschberger, M.L.

International Symposium on Disorders of the Ruminant

(1998) Supplemental dietary biotin for prevention of lesions

Digit, Florida.

associated with aseptic subclinical laminitis (pododermati-

Green, L.E., Hedges, V.J., O'Callaghan, C., Blowey, R.W. & tis aseptica diffusa)
in primiparous cows. American Journal Packington, A.J. (2000) Biotin
supplementation to dairy

of Veterinary Research, 59, 733–8.

cows – multivariate analysis of the prospective longitudinal

Ossent, P. (1995) The pathology of digital disease. Cattle Prac-study. In
Proceedings of the 11th International Symposium

tice, 3, 263.

on Disorders of the Ruminant Digit, Parma, Italy, September Ossent, P. &
Lischer, Ch. (2000) Sole ulcer, what's new about 2000, pp. 305–7.

an old disease? In Proceedings of the 11th International

Greenough, P.R. (1987) An illustrated compendium of bovine

Symposium on Disorders of the Ruminant Digit, Parma,

lameness, part two. Modern Veterinary Practice, February, Italy, September
2000, pp. 46–9.

94–7.

Paulus, N. & Nuss, K. (2002) Claw measures at defined

Greenough, P.R., Weaver, A.D., Brom, D.M., Esslemont, R.J.

sole thicknesses. In Proceedings of the 12th International

& Galindo, F.A. (1997) *Basic concepts of bovine lameness.*

Symposium on Lameness in Ruminants, Florida, pp. 428–

In Lameness in Cattle (ed. by P.R. Greenough & A.D.

30.

Weaver), 3rd edn, p. 10. W.B. Saunders, Philadelphia.

*Prentice, D.E. & Neal, P.A. (1972) Some observations of the Hagemester (1996)
Research Institute of Biology in Farm*

incidence of lameness in dairy cattle in west Cheshire.

Animals, Rostock GDR, Research Report.

Veterinary Record, 91, 1–7.

Hedges, V.J., Blowey, R.W., O’Callaghan, C., Packington, A.J.

Read, D.H., Walker, R.L., Castro, A.E., Sundberg, J.P. &

*& Green, L.E. (2001) A longitudinal field trial of the effect Thurmond, M.C.
(1992) An invasive spirochaete associated*

434 • Chapter 31

*with interdigital papillomatosis of dairy cattle. Veterinary (1995) Spirochaetes
isolated from dairy cattle with papillo-Record, 130, 59–60.*

*matous digital dermatitis and interdigital dermatitis. Veteri-Russell, A.M.,
Rowlands, G.J., Shaw, S.R. & Weaver, A.D.*

nary Microbiology, 47, 343–55.

(1982) Survey of lameness in British cattle. Veterinary

Wells, S.J., Trent, A.M., Marsh, W.E. & Robins, R.A. (1993) Record, 111, 155–60.

Prevalence and severity of lameness in lactating cows in

Toussaint-Raven, E. (1985) Cattle Footcare and Claw Trim-

a sample of Minnesota and Wisconsin herds. Journal of

ming. The Farming Press, Miller Freeman, Ipswich.

the American Veterinary Medicine Association, 202, 78–

Tranter, W.P. & Morris, R.S. (1991) A case study of lameness 82.

in three dairy herds. New Zealand Veterinary Journal, 39, Wells, S.J., Trent, A.M., Marsh, W.E., Williams, N.B. & Robins, 88–96.

R.A. (1995) Some factors associated with clinical lameness

Vermunt, J.J. (1998) Axial wall cracks. In Proceedings of the in dairy herds in Minnesota and Wisconsin. Veterinary

10th International Symposium on Disorders of the Ruminant

Record, 136, 537–40.

Digit, Lucerne, Italy, September 1998, p. 141.

Whitaker, D.A., Kelly, J.M. & Smith, E.J. (1983) Incidence of Walker, R.L., Read, D.H., Loretz, K.J. & Nordhausen, R.W.

lameness in dairy cows. Veterinary Record, 113, 60–2.

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Lameness Above the Foot

A.D. Weaver

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Diseases of skin and subcutis

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Tarsal cellulitis

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Introduction

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Peroneal paralysis

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Sciatic paralysis

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Downer cow syndrome

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Introduction

Fractures

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Introduction

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Lameness above the foot accounts for only 5–15 per

First aid

442

cent of dairy cow lameness, and perhaps twice as great

Fracture repair

442

a proportion in beef cattle, for which data are lacking.

Long bone fractures: external fixation

442

Economic conditions in the UK and elsewhere in

Long bone fractures: internal fixation

444

Western Europe have led to an increase in the size of

Introduction

444

the average dairy herd (see p. 23), but the labour force

Humeral fractures

444

Femoral fractures

444

has been reduced. Additionally, only a minority of

Tibial fractures

444

farmers have been financially secure enough to improve

Growth plate and other injuries

444

older dairy units, or to replace them with new accom-

Vertebral fractures

445

modation. A personal survey of dairy cattle housing

Pelvic fractures

446

(Weaver, 1997) revealed their construction dates to

Dislocations and subluxations

447

be 1955 to 1994 (n = 49) mean 1976, i.e. over 20 years Hip joint

447

old. Seven units had undergone structural improve-

Sacroiliac luxation and subluxation

448

ments 5 to 28 years (mean 19 years) later. In the period

Stifle joint: femoropatellar

448

1997 to 2000 two units were completely rebuilt, while

Stifle joint: femorotibial

449

six stopped operating as dairy farms. The result has

Fetlock joint: metacarpophalangeal and

been an apparent increase in both minor and more

metatarsophalangeal

450

Muscle and tendon injuries

451

severe injuries in dairy cows. In the worst scenario a

Introduction

451

routine (herd fertility) visit to a dairy unit can reveal

Specific muscles

451

two downer cows, both needing urgent nursing care (see

Contracted flexor tendons

451

p. 440), while the labour – two or three experienced

Traumatic flexor tendon injuries

452

persons – is not always available immediately to move

Joint diseases

453

such cows to straw-bedded boxes.

Osteochondrosis

453

Non-digital lameness problems due to poor accom-

Hip dysplasia

453

modation arise from inadequate size and quality of

Degenerative arthritis

454

cubicles, false economy of bedding requirements, over-

Infectious (septic) arthritis

455

crowding of cubicles and feeding areas, inappropriate

Introduction

455

mixing of first lactation heifers with mature cattle

Septic arthritis: joint ill in neonate

455

Septic arthritis: older cattle

457

(leading to bullying, slipping and trampling of younger

Miscellaneous neuromuscular diseases

458

stock), and an increasing tendency to push cattle

Spastic paresis

458

around rather than permit them to move at their own

Spastic syndrome

459

speed.

435

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The results, discussed elsewhere in this chapter,

the forward phase of the stride shortened. After one

include an increased incidence of skin and subcuta-

week muscular atrophy causes increased prominence of

neous problems such as tarsal cellulitis (p. 460), carpal
the scapular spine ('sweeney').

swelling (p. 460), traumatic tendon injuries (p. 451),
pelvic damage (p. 446) and hip dislocation following

Treatment

falls (p. 447). Not one of these conditions is new.

However, as housing systems in the present economic

Treatment is purely palliative. Vitamin B complex may
climate fail to satisfy two of the five freedoms – freedom

be given systemically. The prognosis is usually good.

from thermal and physical discomfort, freedom from

fear and stress – there is a renewed obligation to rec-

Brachial plexus injuries

ognize the deficiencies in specific dairy units and to

advise on both economic and welfare grounds, the need

Aetiology

for radical improvements. The introduction of herd

Trauma, as from sudden caudal and ventral displace-

health assurance schemes (p. 52) and increased welfare

ment of the shoulder and entire forelimb, together with

checks on many farms means that housing conditions

*abduction can lead to separation of any group of the
for dairy herds are likely steadily to improve.*

*nerve roots C6 to T2. The nerves mainly affected are
Economic conditions render many of the treatment
the radial, median and ulnar. Occasionally, the nerves
and management modalities in this chapter hopelessly
are damaged by penetrating injury in the axilla with
non-economic except in valuable pedigree stock. It is
extensive additional damage to the vasculature. More
therefore all the more important that early and accu-
frequently, the plexus is injured during traction in dys-
rate diagnosis (e.g. septic arthritis in neonate, p. 455,
tokia and severe abduction of the forelimb.*

*tibial fracture, p. 444) is sought and the options are dis-
cussed fully with the responsible person on the farm.*

Pathology

Nerve paralyses

*The avulsion close to the spinal cord results in a com-
plete loss of motor supply to the forelimb. Occasionally,*

Introduction

the cranial segments of the network (supplying the

suprascapular and axillary nerves) are spared.

Nerve paralyses of the forelimb include the suprascapular, brachial plexus and radial. The main hindlimb involvement is with the femoral, obturator, tibial, per-

Clinical signs and diagnosis

oneal and sciatic. The usual common aetiological com-

*The forelimb is non-weightbearing and maintained in
ponent is trauma. The typical age of affected individuals
flaccid extension, with a tendency for the digits to be*

*varies from neonate to mature cow. The treatment in all
dragged. Sensation is absent from the level of the elbow
forms involves careful nursing and supportive care (e.g.
distally. Muscular atrophy is evident after seven days.*

oral fluids), and management of the primary lesion (e.g.

Differential diagnosis includes radial paralysis

fracture). Sometimes multiple nerve injuries occur

(dropped elbow, more localized loss of skin sensation)

*as in the downer cow syndrome, which is considered
and humeral shaft fracture with or without radial nerve
separately (p. 439).*

injury (see p. 444), and forelimb dislocation.

Suprascapular paralysis

Treatment and prevention

Aetiology and pathology

Since brachial plexus avulsion has a very poor prog-

The suprascapular nerve originates from cervical 6 and

nosis, treatment is not usually justified. In calves with

7 roots. It may be involved in brachial plexus injuries

partial avulsion, anti-inflammatory drugs and limb

(see below). The nerve supplies the supraspinatus and

support are suggested. Affected cattle should be con-

infrapinatus muscles. It has no sensory component. It

fined to a well-bedded stall and care should be taken

is occasionally injured by blunt trauma in calves butted

to avoid the development of decubital damage to the

by others at mangers and troughs when the vertical bar

analgesic lower limb.

barrier bruises the scapular region. Associated injuries

can include fractures of the scapular neck or acromium.

Radial paralysis

Clinical signs and diagnosis

Aetiology and pathogenesis

Suprascapular paralysis causes few clinical signs initially. The nerve (roots C7–T1) is damaged in its distal portion. The shoulder joint may be slightly abducted and secondarily to humeral shaft fractures, which tend to be



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Cattle maintained in lateral recumbency under general anaesthesia should have soft supporting surfaces and the down forelimb should be extended forwards to reduce the pressure on the vasculature to the lower parts of the limb. Also, the upper foreleg should not be roped tightly down against the table, whereby it would increase pressure on the lower leg.

Femoral paralysis

Aetiology and pathology

The femoral nerve (L4–6 spinal nerve roots) has a short

course, ramifying in the iliopsoas and quadriceps muscles. It is liable to damage in the oversized fetus of large-framed breeds (Simmental, Charolais, Holstein)

Fig. 32.1

Radial paralysis of right foreleg of six-year-old at delivery when presented in the 'hiplock' or 'stifle Friesian cow. Paralysis was present on standing following 120 lock' position, which tends to cause femoral hyper-minutes in right lateral recumbency for surgery of a septic paro-extension as forced traction is exerted. The femoral tiditis under general anaesthesia. Total recovery took 48 hours. nerve, usually unilaterally, is drawn against the pubic The classical signs are present: dropped elbow, flexed carpus brim at the pelvic inlet. The injury can occur in either and inability to extend forelimb for weight bearing. anterior or posterior presentation. The lesion is sometimes a rupture of the nerve, but more frequently spiral, comminuted and to have sharp protruding spikes involves very severe perineural haemorrhage and of bone. Radial nerve paresis frequently occurs follow-oedema. In other cases, the quadriceps femoris muscle

ing a long (one to two hour) period of general anaesthesia is severely stretched and incurs damage to its vascular supply in lateral recumbency when the primary damage and nerve supply. The resulting quadriceps atrophy is a compressed vascular compartment. A dairy cow presents with typical clinical signs.

trapped under the lower horizontal bar of a cubicle division can also, on release, have radial paralysis. While

Clinical signs and diagnosis

partial paralysis is more common, complete radial paralysis can result from brachial plexus injuries or

The animal, usually a neonate, is unable to maintain the severe trauma. Cattle entering a crush fast and banging stifle in extension due to quadriceps dysfunction. The the shoulders against the yoke gate occasionally have a joint is flexed and the digit rests with minimal weight temporary partial radial paralysis.

bearing. Usually, hock and digital joints are also flexed, though the toe is not dragged. A small area of loss of skin sensitivity may be detectable lateral and proximal

Clinical signs and diagnosis

to the stifle joint. Quadriceps atrophy within 10 to 14 days results in a triangular depression in the lateral. The main signs are a dropped elbow due to triceps cranial part of the thigh. Fibrosis of the remaining paralysis, knuckled fetlock and an inability to advance quadriceps muscle mass develops later. The stifle joint the limb (Fig. 32.1). If the limb is placed in its usual may become unusually prominent. Differential diagnosis involves dorsal or lateral patellar luxation, septic weight. Sensory loss can be detected over the elbow and gonitis, femoral fracture or growth plate separation, hip lateral aspect of the forearm.

dislocation and quadriceps rupture.

Differential diagnosis includes humeral fracture with or without radial paralysis.

Treatment and prevention

The prognosis is guarded or poor. Recovery is unlikely

Treatment and prevention

in calves with femoral nerve rupture. The recovery time

The prognosis is good if the nerve has not been sec-

for regeneration was about four months in an experiment by humeral bone fragments. Treatment is supportive, keeping the animal on soft bedding and on non-slip surfaces. The fetlock should be bandaged to prevent iatrogenic trauma. Anti-inflammatory drugs Calves should receive colostrum by bottle within 6 hours of birth if the injury has prevented rising to suck.

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Clean and dry bedding is essential. The calf should be frequently turned. Analgesics, anti-inflammatories, and vitamin E and selenium preparations should be given.

Tibial paralysis

Obturator paralysis

Aetiology and pathology

Aetiology and pathology

Tibial paralysis is rare. It is associated with iatrogenic damage following deep infections in the caudal thigh

Obturator paralysis classically involves unilateral or musculature or lacerating injuries in the medial aspect bilateral damage to the nerve, usually in heifers during of the hock with associated damage to related tendons.

dystokia due to fetal oversize or maternal immaturity, when the nerve (L4–L6 nerve roots) is damaged by fetal pressure on its course along the medial aspect of the

Clinical signs and diagnosis

ileum or along the pelvic floor. Experimental section of Tibial paralysis results in hock flexion from inability pri- the nerve, which innervates the adductor muscles (gra- marily to cause gastrocnemius contraction, which would cilis, pectineus, adductor and external obturator), has result in hock extension. It also causes hyperextension caused mild abduction of one or both hind limbs but of the digits in that the weight is borne more on the experimental animals have been able to rise without

heels. Skin sensation is absent over the caudal aspect of the metatarsus and digits. Differential diagnosis of the metatarsus and digits. Differential diagnosis synonym for obturator paralysis, 'calving paralysis', is includes peroneal paralysis and gastrocnemius and considered a misnomer since the latter is usually associated with the 'downer cow' syndrome (Cox & Onapito, 1998) (see p. 439). Many clinical cases of so-called obturator paralysis have a more complex aetiology involving branches of the sciatic nerve or primary Treatment and prevention

ogy involving branches of the sciatic nerve or primary Treatment is symptomatic, involving confinement and muscle damage.

Treatment and prevention

ogy involving branches of the sciatic nerve or primary Treatment is symptomatic, involving confinement and muscle damage.

vitamin B complex injections.

Clinical signs and diagnosis

Peroneal paralysis

Abduction of the hindlimb (unilateral) or a straddled

Aetiology and pathology

gait (bilateral) are the classical signs. Confusion clinically may result from the stance adopted by a heifer

As with the tibial nerve, the peroneal nerve is a major branch of the sciatic nerve (L6–S2 nerve roots). Its common site of damage is over the lateral aspect of the stifle joint where the nerve lies relatively close to the paralysis. Some cattle are recumbent and such recumbency is rarely due to simple unilateral obturator injury. or chronic pressure (prolonged recumbency).

Cattle are liable to slip with gross hindlimb abduction, giving rise to hip luxation. Skin sensation remains

Clinical signs and diagnosis

unimpaired. Diagnosis is based on the history of possible injury at parturition and on the characteristic

The hock is hyperextended, and fetlock and digits are stance and gait.

flexed (knuckled over) (Fig. 32.2). The foot can be placed in its normal position. Skin sensation is lost dorsally from the fetlock distally to the coronary band.

Treatment and prevention

Diagnosis is easily made on the clinical signs and

The animal should be kept on non-slip surfaces to avoid sensory loss. Differential diagnosis involves tibial par-possible hip luxation or femoral neck fracture. Sand on alysis and painful digital diseases.

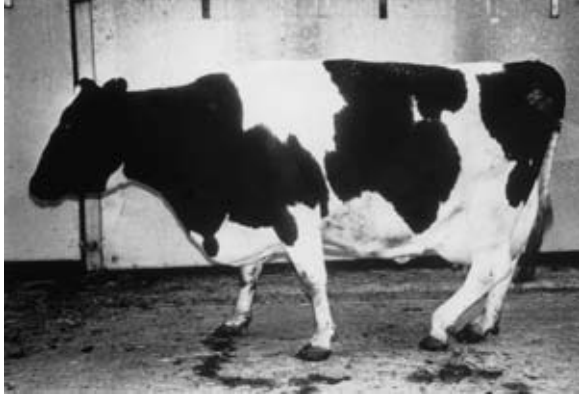
the concrete floor of a calving/isolation box, overlaid with plenty of straw, is ideal. The hocks and hind feet

Treatment and prevention

should be kept close together by a figure of eight

pattern of rope above the hocks or fetlocks. The dis-

The prognosis is good with recovery usually evident in tance between these joints should not exceed 30 cm (12 a few days. Self-induced trauma to the fetlock is mini-inches), which permits the animal to rise. Supportive mized by application of a soft bandage or a light resin care should be continued for several days. Breeding cast to the fetlock.



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Downer cow syndrome (see p. 797)

Defined as ‘a cow down in sternal recumbency for unknown reasons’, the syndrome usually affects high-yielding dairy cows in the first 48 hours postpartum. In the UK the incidence appears to be increasing as a result of overcrowding in cubicle houses (see p. 43). Veterinary attention may not be sought for 24 hours, as a response to calcium borogluconate therapy is awaited. The condition has a multiple aetiology including traumatic, metabolic, neurological and toxic infectious causes. The following are examples:

- *Traumatic: sacroiliac luxation and subluxation,*

Fig. 32.2

*Peroneal paralysis in six-year-old Friesian cow
bilateral or unilateral coxofemoral luxation,*

showing extended hock and knuckling of fetlock. Two days pre-rupture of gastrocnemius muscle, pelvic fracture, viously the digit had also been flexed. Some weight is now borne massive blood loss (profound anaemia). on claw normally. Total recovery took another three days.

- **Metabolic:**

non-responsive hypocalcaemia, hypokalaemia.

- **Neurological:** lymphosarcoma infiltration into tho-

Sciatic paralysis

racic or lumbosacral spinal canal, bilateral peroneal paralysis, complications of obturator paralysis.

Sciatic nerve paralysis involves the nerve that originates

- **Toxic infectious:** septic metritis, acute coliform from three ventral roots (L6, S1–2). Damage to L6 root mastitis (pp. 334, 519).

may be an important component of ‘calving paralysis’ or obturator paralysis (Cox, 1981) (see p. 438). The The primary recumbency, whether due to dystokia sciatic nerve is liable to damage unassociated directly (46 per cent of cases), milk fever (38 per cent), or other

with parturition but in prolonged unilateral recumbences (16 per cent) (Chamberlain, 1987), is followed by bency. The site is usually close to the medial aspect of secondary recumbency. The usual explanation for the the femoral greater trochanter and may be associated development of a downer syndrome in a cow, which is with struggling. Such circumstances can involve partial unsuccessfully treated for clinical signs diagnostic of recovery from milk fever or postparturient hypocalcaemia, as the cow struggles to rise on a slippery surface the hypocalcaemia has been treated too late and that (e.g. concrete) (Cox & Onapito, 1998). Rarely, sciatic unrecognized traumatic injury has already taken place. paralysis is secondary to femoral neck or pelvic fracture Half of all 'downer cows' develop within 24 hours of (pubis, ischium), or iatrogenic in origin from septic parturition. They tend to be high producers. The incidence is higher in winter. Survey data indicate a considerable range (3.8–28.2 per cent) in the incidence of

downer cows as a percentage of cases of milk fever. One

Clinical signs and diagnosis

study revealed that 33 per cent of downer cows recov-

The limb is entirely non-weightbearing. Sensation is

ered, 23 per cent were slaughtered, and 44 per cent died

lost from the limb distal to the stifle except for a strip

or were euthanized.

on the medial aspect of the thigh distal to the mid-

Pressure damage causes ischaemia of muscles and

metatarsal region, which is supplied by the saphenous

nerves. Following a variable period of struggling,

branch of the femoral nerve. Differential diagnosis

further skeletal injury such as gastrocnemius muscle

should include femoral fracture, the signs of which will

rupture may occur. The differential diagnosis is often

include crepitus, possibly swelling and abnormal mobil-

difficult since frequently a non-responsive milk fever

ity, and septic gonitis and tarsitis.

cow does not appear to have any further specific

abnormality.

The aetiology of this pressure damage involves

Treatment and prevention

effects resulting from the 'compartment syndrome'

The prognosis is usually hopeless. Many cases are pre-sented as 'downer cows'. It is vital that this paralysis be within an osteofascial compartment following external recognized early, so that long-term nursing measures pressure or internal filling of the compartment with will not be instituted. Early slaughter is advisable.

blood or oedematous fluid, or both. External pressure

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results in ischaemia, 'leaky' vessels, and a postcompres-sion swelling. The effects of the crush syndrome, which onset of recumbency in cows that recovered to stand, refer to the systemic results or muscle damage, involve and at 48 hours in cows that became permanent largely the absorption of muscle breakdown products.

'downers'. Plasma or serum CK values reflect the total

One specific cause of the downer cow syndrome is mass of injured muscle, but CK half-life is such that it

pressure damage to the hindquarters following a period is of doubtful value as a prognostic indicator several of prolonged recumbency (3–6 hours or more), result-days after initial recumbency, when little further active ing both in extensive muscle damage and reversible or muscle damage may be occurring.

irreversible changes in the sciatic nerve caudal to the Plasma aspartate aminotransferase (AST) is elevated proximal end of the femur and to the peroneal nerve.

in animals with muscle damage. This enzyme is also

Consideration of the aetiology and pathology of

released in cardiac myopathies, but postpartum

traumatic factors is reviewed in other sections (see

increases in downer cows are almost invariably related

Fractures, p. 441; Dislocations and subluxations, p. 447;

to the muscle component.

Muscle and tendon injuries, p. 451).

In cows recumbent for over 48 hours (at the time of second clinical examination), clinical pathology can be reassessed. Urinalysis may now reveal proteinuria

Clinical signs and diagnosis

indicative of myoglobinuria (from muscle breakdown),

By definition the animal is recumbent. The cow is

ketonuria and bilirubinuria (from partial anorexia).

usually alert, eats, and can defecate and urinate.

If the aetiology of the persistent recumbency remains

Affected cows make little or no attempt to rise in the

in doubt, a repeat complete clinical examination should

hindlimbs. Some alert cows move around using the

bear in mind less common causes (e.g. infiltration of

forelimbs and have been termed 'creeper cows'. Cows

lymphosarcoma into the spinal canal). A careful

confined to a stanchion do not have facilities to demon-

recheck of the history may aid diagnosis at this stage.

strate this movement, which may appear after the

animal is transferred to a well-bedded stall or box. Dull

Treatment and prevention

or comatose and anorectic cows may be 'downers' due

to systemic toxic factors (coliform mastitis, severe

The downer cow should be moved to a well-bedded

metritis). Occasionally, downer cows are hyperexcitable

loose box not later than 24 hours after the onset of

due to hypomagnesaemia or hypocalcaemia (see recumbency. Ample food and water must be provided Chapter 46).

in low, wide-based containers.

Rectal palpation is crucial to evaluate various

The primary aim is to raise the cow. Any attempt

traumatic aetiologies: sacroiliac (sub)luxation, pelvic

should be made on a relatively non-slip surface. Per-

fracture, severe, intrapelvic, soft tissue injury and

sonnel should be ready to assist with tail support fol-

coxo-femoral luxation, as well as to check the state of

lowing provocative stimulation (e.g. electric goad). Hip

the uterus (puerperium). External assessment includes

clamps, e.g. Bagshaw hoist, may be applied to the wings

palpation of the spine for abnormal angulation,

of the ilium for a maximum of 20 minutes. Usually, the

swelling, or crepitus (fracture), musculature of the

ability of the cow to bear weight is obvious. While slung

hindlimbs, especially the adductor group medially

in this manner, the hindquarters should be checked for

(muscle rupture), manipulation of the upper hindlimb

symmetry and the opportunity taken to flex and extend and pelvis for evidence of fracture or dislocation, and each hindlimb to check for fractures. Other devices for determination of possible loss of skin sensation. The elevation include air cushions or bags, webbing slings possibility of acute coliform mastitis should be investigated. The cow should be turned to the opposite side cows that can stand following hoisting require to be for similar examination of the other hindlimb. Cows hobbled above the hocks or hind fetlocks to prevent usually prefer to lie with the more painful hindlimb abduction of the hindlimbs.

uppermost.

Hip clamps may be repeatedly applied for a few

Downer cows have variable changes in blood mineral minutes of forced elevation of the hindquarters to levels, some showing a persistent hypocalcaemia, improve circulation. When lowered, the cow should hypophosphataemia, and possible hypomagnesaemia. be placed with the previously dependent side

The role and significance of hypokalaemia is disputed.

uppermost.

The release of potassium from damaged muscle cells

The most important component of treatment is good

does not necessarily cause hyperkalaemia because the

nursing. The cow must be made comfortable. Owners

usual coexisting hypocalcaemia has a tendency to cause

should be observant for the possible development of a

hypokalaemia. All cases show an elevated creatine

toxic (e.g. coliform) mastitis during recumbency. Rapid

kinase (CK), which is specific for muscle injury. In one

loss of weight, signs of systemic toxicity, and anorexia

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are grave prognostic indicators. Soft bedding is essen-

Prevention of the downer cow syndrome is particu-

tial for effective nursing in all downer cows, which

larly vital today since treatment is very labour-intensive

should be turned several times daily.

and repeated veterinary involvement is too costly for

Fresh water should be available alongside a bucket

the minimal profit margins of many dairy farms.

containing a commercial electrolyte solution. Parenteral treatment includes repeated injection of solutions containing calcium–phosphorus–magnesium with

Fractures

glucose and potassium. Additional potassium should only be given by stomach tube (e.g. 50 g KCl daily).

Introduction

Tripelennamine hydrochloride given as 12 ml solution

In the assessment of bovine fractures, several factors

i.v. is an effective stimulant and antihistaminic that

require consideration in each clinical situation.

makes a downer cow appear more alert (Chamberlain,

1987). Analgesics such as phenylbutazone, flunixin

meoglumine and ketoprofen should be restricted to cows

Economics

with obvious signs of pain.

The prognosis is good in well-nursed ‘creeper cows’

With some exceptions, economic consideration is a vital

with some voluntary hindlimb movement and cows that

factor. Many cattle not used for breeding, e.g. steers,

are capable of a short period of weight bearing when

are best slaughtered at once, since treatment would be hoisted or slung. The prognosis is poor in animals with costly. Young potentially valuable breeding stock fall no hindlimb movement. Proprioceptive reflexes should into a different category, where slaughter value is neg- be repeatedly checked in such cows.

ligible in comparison with the potential value following Generally, excellent nursing, a consistently good fracture healing.

appetite, and some spontaneous movement should encourage owners to persist and practise patience.

Size, age and sex

Valuable prognostic indicators for survival in a limited British survey of 64 downer cows were as Young cattle respond well to restrictions imposed follows.

during fracture immobilization. They tend to rise easily despite external fixation and to be less liable to develop

- On day 2 of recumbency: body condition score, decubital lesions. The soft thin cortices of long bones in quality of nursing care, absence of hypocalcaemia,*

calves make plate and screws generally an unsuitable relative hypomagnesaemia, and lower AST and CK means of internal fixation. Calves have a remarkable enzyme levels.

ability rapidly to produce a fibrous response at a frac-

- On day 4 of recumbency: survival was associated ture site and early callus formation.*

with little further rise of CK and with continued good nursing.

Disposition

A US survey of 15 veterinarians' experience with slings used on 145 cows showed a recovery rate of 52 An advantage of fracture healing in cattle compared per cent.

with horses is their tolerant disposition making han-

Prevention depends primarily on the avoidance of dling easier.

parturient paresis associated with milk fever (see Chapter 46). Calving should take place in properly

Location and character

designed areas with good footing. Many farms will

benefit from stalls with 30 cm (12 inches) clean sand as bedding. Generally, the more proximal the fracture, the more difficult is its anatomical reduction and rigid fixation. Thus the incidence of downer cows has been primarily attributed to humeral and femoral fractures have notoriously low recovery rates, while metatarsal and metacarpal fractures and its prompt treatment.

Fractures usually heal well. Fracture of the distal phalanx, Periparturient cows should be placed onto a minimal slip surface and sand is excellent for this purpose if in which displacement is minimal, is discussed elsewhere (see Fracture of distal phalanx, p. 429).

stall-bedded calving boxes are not available.

Comminuted fractures are the rule in cattle. Many A high incidence of downer cows in first-calf heifers, fractures are closed initially but become open following where hypocalcaemia can be ruled out, may be due to inappropriate handling such as movement to a stall. dystokia. Breeding policies may require modification

The prognosis for open fractures, especially proximal such as use of a smaller breed bull or later first service fractures (humerus, tibia, femur), is guarded. Soft tissue dates for heifers.

damage is often limited to haematoma and oedema

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formation resulting from the original insult and movement of the fractured bone ends. In certain instances, extensive experience is unnecessary. External fixation significant nerve damage (e.g. to radial nerve in a spiral humeral shaft fracture) may be an important consideration in the prognosis.

in two limbs simultaneously. Young cattle develop

Most fractures tend to be oblique or spiral in cattle.

osteomyelitis following open long bone fractures more

Maintenance of reduction while external or internal

frequently than older cattle. Careful examination of the

fixation is applied tends to be more difficult than with

fracture site, preferably after clipping of the skin, is transverse fractures.

essential to avoid missing an open fracture. External fixation alone may be contraindicated in very heavy animals.

Duration of fracture

Unlike small animals and horses, the duration of a

Choice of materials and technique for

bovine fracture before presentation can often be 12–24 external fixation

hours, a consequence of the relatively casual methods of husbandry practised on some farms. The result is

Immobilization techniques include the splint or cast, invariably a greater degree of soft tissue injury than the modified Thomas splint, and both methods together.

in a freshly presented fracture. One effect of a longer

Casting materials available today include plaster of duration of movement at a fracture site is an open

Paris (one form of which is rapid-setting), thermoplastic fracture.

tic polyester polymers (Hexcelite), and polyurethane

resin incorporated into woven material of polyester (Cuttercast, Baycast) or fibreglass (Deltalite/Scotch-

First aid

cast). Casts should always be applied over a layer of padding. The depth of padding should be considerably greater if soft tissue swelling is anticipated at the fracture site and also should be generous over bone prominences. The padding should extend beyond the intended proximal point of the cast. The cast should be applied at 90° to each other (e.g. cranial and lateral surfaces of the forelimb to stabilize metacarpal fracture) and fixed with old towels or sheeting, and splinting with polyvinyl chloride tubing, or stiff rods such as broomsticks placed in position with duct tape. The joints above and below the fracture should be immobilized. This technique is least 8 cm (3 inches) proximal to the radiocarpal joint

impossible in femoral, tibial and humeral fractures. In and similarly distal to the fetlock.

tibial fractures, a Robert Jones bandage comprising

Positioning is very important in applying a cast.

layers of cotton wool firmly bound by cotton bandage

General anaesthesia is rarely necessary; xylazine is

can achieve adequate immobility if sufficient bulk is

usually adequate for restraint and analgesia. The limb applied.

must be forcibly extended for fracture reduction and

then maintained immobile until the cast has set. Trac-

Fracture repair

tion on the distal part of the limb may be aided by

inserting wire through holes drilled in the toes.

In certain fractures (e.g. rib) spontaneous healing is the

The advantages of a lightweight cast include

rule. In others (e.g. external angle of ilium) repair

increased comfort and ease of rising, and are achieved

usually takes place but a sequestrum may form. The

by fibreglass (e.g. Vetcast, Deltalite, Scotchcast), which

sequestrum generally is only a cosmetic blemish but

has a strength five times greater than plaster of Paris.

sometimes becomes infected and then requires removal.

Weight reduction is also possible using several layers of

Most fractures require treatment by methods of

plaster overlaid by fibreglass casting tape, once the

external or internal fixation, or both, if recovery is to

plaster has hardened. Strength combined with lightness

take place.

is the result.

Some fractures require radiography before reduction

and immobilization to determine the extent and sever-

Long bone fractures:

ity of bone damage. Fractures may also be radi-

external fixation

ographed two to three weeks after immobilization to

assess the degree of healing. Removal of the cast at this

External fixation of fractures is usually satisfactory if

time is often unnecessary. The radiolucency of

the site is distal to the humerus or proximal tibial meta-

cast materials varies, plaster of Paris being poor,



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Deltalite/Scotchcast fair with some mottling, and Cuttercast/Baycast is excellent.

Extension bars have sometimes been incorporated into external casts. These U-shaped metal stirrups have the curved part of the U distal to the toes, so that weight is borne by the bar and transferred proximally to the cast, into which the two arms of the extension bar are buried during cast application. Such a device is only useful if there is a specific indication to avoid weight bearing by the digits. The effective increase in limb

length makes standing up and ambulation more difficult. A modification is the hanging cast. This device comprises one or more intramedullary pins that are drilled through the proximal fracture fragment, and onto which a U-shaped bar is fitted after the usual external immobilization has been applied. When weight is borne, it is transferred proximally to load bearing by the proximal fracture site, so that the fracture site is not stressed.

Thomas extension splints may be put on either forelimb or hindlimb. Good padding is essential both at the points (axilla, inguinum) where maximal pressure will be applied, as well as at any contact points more distally. The Thomas extension splint must be 'made to measure' for the individual case (e.g. circumference of proximal and distal rings, length of cranial and caudal

Fig. 32.3

Bilateral distal metacarpal fractures in a one-day-old longitudinal bars). In growing stock the length should

Salter calf. Circular lesions result from pressure of obstetrical be slightly greater (e.g. 5 cm or 2 inches) than necessary,

chains applied during forced extraction. In the absence of to permit growth. Some devices have expansion clamps percutaneous infection (seen here) external immobilization from or rings on the longitudinal bars to allow continual mid metacarpal to coronary level is indicated. modification.

The bottom ring should be wired to the foot to maintain the leg in extension. Bandages and padding should be placed around the limb at potentially exposed areas Humeral fractures in cattle aged two and a half before they are taped to the longitudinal bars. In the months to three years can heal four to six weeks following Thomas splintage (Wintzer, 1961). In other the tibial (gaskin) area to the cranial bar. The extension cases, immobility has been unsatisfactory and non-splint should be checked carefully for fit and effective union has resulted despite massive callus formation. immobilization of the fracture site before release of the Open fractures, at sites where external fixation is patient. Many cases will benefit from combination of an

practical, require particular attention. The presence of external cast (e.g. fibreglass) and a Thomas extension skin perforation should carry the presumption of an splint.

open fracture even if bone cannot be seen or palpated
Cattle up to 350 kg (800 lb) will generally adapt well through the injury. The wound is cleansed and carefully to Thomas splintage. Patients should be confined to a stall with relatively little straw bedding. Assistance to explore. Any free bone fragments should be removed. Larger wounds require debridement. It may be useful to take tissue samples or a blood clot for microbiological culture in the case of valuable cattle. Wounds should be copiously irrigated with a polyionic solution
Neonatal and other calves less than a month old with (isotonic balanced electrolyte with neutral pH). The simple fractures and rapid callus formation may require value of adding a 0.1 per cent povidone-iodine solution
slackness.

should be copiously irrigated with a polyionic solution
Neonatal and other calves less than a month old with (isotonic balanced electrolyte with neutral pH). The simple fractures and rapid callus formation may require value of adding a 0.1 per cent povidone-iodine solution

external support for two weeks only (Fig. 32.3). Other (1 : 100 dilution of povidone-iodine 10 per cent solution) young cattle should be checked for progress at three weeks. This has not been established in cattle.

weeks by removing the cast with an oscillating saw.

Antibiotics should be given parenterally in all open

Sometimes the same splint may be reapplied (e.g. with fractures in cattle. Natural and synthetic penicillins Deltalite, Cuttercast).

are the first choice. Systemic antibiotics should be

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continued for seven to ten days. Antibacterial sensitiv-

Humeral fractures are difficult to plate due to their

ity patterns may be checked via samples taken at an

short length and spiral configuration. Multiple bone

early stage and before antibiotics are given. Samples

fragments often necessitate the use of lag screws.

should preferably be taken from infected bone at

In older and heavier cattle, internal fixation is less

surgery rather than from discharging sinus tracts where

successful due to greater mechanical forces. Often

contamination with surface pathogens will complicate long-term Thomas splintage is adopted.

interpretation of laboratory results. Antibiotic therapy should be instituted as soon as possible following iden-

Femoral fractures

tification of an open fracture. Small wounds may be covered with a sterile protective dressing, but any external support of the fracture site should be removed after Most have a history of forced extraction. The existence three to five days to permit quick assessment of the of concurrent disease should be evaluated (e.g. wound. Young cattle develop osteomyelitis following colostrum intake). No form of external immobilization open fractures more frequently than adults. The reason is helpful. Internal pinning (Steinmann) rarely achieves may be a richer blood supply in the damaged area. sufficient immobility of the fracture site. The technique of choice is usually plate and screws, possibly double plating, with two plates placed at about 90° to each other (lateral and cranial).

Long bone fractures: internal fixation

Similar techniques may be attempted in older cattle.

Reduction may present major problems. The huge

Introduction

mechanical force acting at femoral fracture sites makes

Economic factors are often important in considering

bending of the plates or loss of screws common. Some

internal fixation of bovine long bone fractures. Exper-

femoral shaft fractures eventually heal with some

tise in the discipline is also essential for successful

months of stall rest alone.

reduction and rigid support under aseptic surgical

conditions.

Tibial fractures

Anatomical factors mean that long bone repair by

internal fixation is most frequently required in humeral,

Tibial shaft fractures are amenable to a variety of

femoral and tibial fractures. Young calves have very

fixation techniques since exposure is relatively easy.

thin cortical bone and screws are liable to loosen or be

Problems arise with severely comminuted or multiple

stripped during insertion. Failure is the common result. fractures extending into the proximal and distal metaphyses. Both access to the fracture site and reduction are much simpler in the calf than adult.

tional support by a Thomas splint, some tibial fractures In adult cattle, where the cortex is thick and dense, may be immobilized by cerclage wire with external the major problems are associated with general anaesthesia (e.g. regurgitation, rumen tympany, compromised pinning. In this method two or three 6 mm Steinmann pins are placed through both cortices of both proximal and distal segments under strict asepsis, the protruding ends are cut to protrude about 2.5 –5 cm (1–2 inches) the degree of overriding of the fragment ends.

from the skin, and a resin (methylmethacrylate) bridge Specialist texts should be consulted for details of

is placed over these protrusions to immobilize the fracture site. The sites of skin puncture should be carefully covered with dressings to reduce the chance of infection tracking along the pins and causing osteomyelitis, which is the primary complication of this technique. Conservative treatment often fails to result in acceptable fracture resolution due to insufficient stabilization

Animals up to yearling size (e.g. 250 kg, 550 lb) are of the break. This often results in the development of amenable to internal fixation unless the fracture is severe lateral deformity of the limb with associated severely comminuted. Such fractures are usually mid-weight loss.

shaft oblique and spiral. The radial nerve is often involved (p. 436). There is often severe overriding,

Humeral fractures

Growth plate and other injuries

making reduction difficult. Intramedullary pinning (or stack-multiple-pinning) combined with cerclage

Separation of the growth plate is a common complication of forced traction in dystokia. The usual site is the

distal femur.



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fracture involving the acetabulum. The simplest method of treatment is confinement, which has a low success rate. Alternatively, the femoral head may be resected to permit development of a pseudoarthrosis, but lameness usually persists along with obvious muscle atrophy and compensatory conformational changes in the contralateral limb. Finally, internal fixation by Knowles pins

or lag screws has been successful but requires intraoperative radiography to ensure the correct direction of insertion from the lateral aspect of the greater femoral trochanter.

Vertebral fractures

Aetiology and pathology

Elsewhere in this chapter, reference is made to long bone fractures and growth plate separation (p. 444) resulting from excessive traction during dystokia. But vertebral fractures due to forced traction, usually in an exotic beef breed such as Charolais, can lead to fracture and/or luxations in the caudal thoracic or cranial lumbar region (preferentially T12–L3). The common form is a fracture of the caudal physis of T13 (Schub & Killeen, 1988).

Postmortem changes include perirenal and

Fig. 32.4

*Separation and displacement of distal metacarpal
perivertebral haemorrhage,
fractured ribs,
cord*

*growth plate in 15-month-old Limousin bull. Complete recovery
compression, severed cord, subdural haemorrhage,
followed manipulation and traction reduction (over 1000 kg pull) and
myelomalacia.
and four weeks immobilization in resin cast.*

Clinical signs and diagnosis

*distal metacarpus or metatarsus (Fig. 32.4). Non-weight
The calf is alive before traction is applied in dystokia,
bearing swelling and crepitus are major clinical
but following delivery the calf is weak, usually recum-
signs.*

*Cases must be distinguished from distal
bent, with a swollen head, dyspnoea, and spinal devia-
metacarpal/metatarsal shaft fractures and fetlock dislo-
tion. These calves usually die within 24–48 hours from
cation or subluxation. Most cases respond to manual
neurogenic shock (pain, trauma, hypoxia).*

reduction and some weeks' immobilization in a cast.

The normal rigidity of the spine in the affected tho-

*Other sites of growth plate separation include the
racolumbar region invariably cannot tolerate the spinal*

distal femur, distal tibia and distal radius. Apart from curvature induced during typical traction on forelimbs external support, cross and Rush pinning are suitable of a fetus in anterior presentation when these dorsal methods of immobilization. The pins in the first method structures become stretched and convex. When fetal traverse the epiphysis obliquely to emerge through the hips lock on entering the maternal pelvis, the thoraco-opposite metaphysis. In Rush pinning, the pin is lumbar region is approximately level with the maternal directed from the epiphysis to slide in a sledge-runner vulva. If the fetal body is then twisted and bent from fashion proximally along the internal surface of the side to side, acting as a pivot, a considerable risk of opposing diaphysis. Although such pins are liable to excessive shearing or traction forces can develop. displace rather easily, healing is rapid and the success rate is high.

Treatment and prevention

A specific and difficult orthopaedic problem is fracture of the femoral neck and separation of the proxi-

The magnitude of traction forces in dystokia should be appreciated. Two men can exert a 350 kg pull for 30 seconds. A calf-puller device can produce over 500 kg traction. Adequate lubrication of the birth canal and an includes proximal femoral shaft fracture and pelvic instinctive reluctance to use more than manual pressure



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are the two most important preventive measures, since

Osteoporosis is a predisposing factor to pelvic treatment is useless. A greater willingness to adopt fracture in some high-yielding dairy herds.

caesarean section in oversized viable fetuses will

Other pelvic fractures involve the wing or shaft of the help prevent further accidental injuries (see p. 1115).

ilium, tuber ischii, and the acetabular margin. The ilial fractures usually involve massive trauma, such as falls down cliffs. Tuber ischii damage may result from road

Pelvic fractures

traffic accidents in which the impact is caudal. Acetabular margin fracture chips often involve traumatic hip

Aetiology and pathology

luxations.

Severe pelvic fractures are rare in cattle. Trauma is almost always involved. The most common pelvic frac-

Clinical signs and diagnosis

ture involves the tuber coxae, which is damaged during

The degree of lameness varies considerably from nil passage through a narrow doorway or in a sudden fall (tuber coxae) to severe and even to recumbency (pubic

laterally onto hard ground (Fig. 32.5). Unlike other and ilial shaft fractures). The external bony landmarks pelvic fractures, the tuber coxae fragment may be are frequently asymmetrical. Crepitus may be obvious pulled ventrally by the fascia lata, becoming a as the animal walks. Rectal palpation will often permit sequestrum from an inadequate blood supply and may localization of the crepitus and an appreciation of develop a septic draining tract to the exterior. surrounding soft tissue injury. One useful technique is Another specific condition is separation of the pubic palpation during lateral rocking of the hindquarters. symphysis, resulting from excessive traction on a fetus Fractures of the ilial shaft, pubis and cranial ischium in the pelvic canal of an immature heifer. can be appreciated on such rectal exploration. Vaginal palpation should be performed in mature cows and recently calved heifers. Clinical diagnosis of acetabular fractures, which tend to involve the dorsal rim, is difficult as the localized crepitus must be distinguished from coxofemoral dislo-

cation (in which the greater femoral trochanteric position is abnormal) and femoral neck or physeal fractures (which may be suspected from the increased mobility of the hip region in the affected limb). Radiography is essential for diagnosis in some cases, but, due to size and equipment constraints, is rarely feasible.

Other differential diagnoses include pelvic bruising leading to haematoma formation and pelvic abscessation.

Haematomas rarely cause lameness, while abscesses tend to have a more gradual onset and a protracted course. Several sites of pelvic fractures can lead to nerve trunk (sciatic, femoral, obturator) and blood vascular damage (middle uterine artery, internal pudendal, internal iliac arteries and veins).

Fractures of the bodies of the ischium and pubis invariably involve the obturator foramen, usually also the obturator nerve, and are serious in that marked pain and discomfort tend to cause prolonged periods of recumbency and a reluctance to stand. Commonly, both bones are fractured, making the pelvis unstable, and

healing is then unlikely. In some cases, healing is liable to be accompanied by excessive callus formation. This sometimes extends to produce hip osteoarthritis and a persistent lameness.

Fig. 32.5

Fracture of right wing of ilium in adult cow, showing

Degenerative osteoarthritis of the hip in the older

asymmetry relative to the greater femoral trochanter and ischial bulbs and cows leads to considerable peripheral new

tuberosity.

bone formation. Sometimes, following minor trauma,

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small portions of the new acetabular dorsal rim

appreciated on limb abduction and flexion. Rectal

are chipped off (pathological fractures), leading to a

palpation aids localization of crepitus to the hip. It will

sudden increase in severity of the lameness. Often such

also help to differentiate the less common directions of

fracture chips will heal by fibrous tissue, but lameness

luxation. In cranioventral luxation, abnormal move-

persists.

ment may be appreciable cranial and lateral to the pelvic inlet when the cranial border of the pubis is followed. In caudoventral luxation into the region of the

Treatment and prevention

obturator foramen, movement may be noted as the leg. Surgery is indicated in few cases of pelvic fracture, e.g. is abducted, flexed and extended, released, taking the tuber coxae sequestrum. The only useful treatment is femoral head out of and back into the margin of the generally rest in a well-bedded stall. Use of hip clamps foramen.

(e.g. Bagshaw hoist) to raise animals is contraindicated.

Diagnosis may be confirmed in smaller cattle on ven-

Few cattle become acclimatized to slings, which may trodorsal radiographs. Differential diagnosis includes increase the discomfort caused by the fracture. Cases of femoral neck fractures, physeal separation, pelvic pelvic fracture should be reassessed after three to four (acetabular) fractures, and greater femoral trochanteric weeks of good nursing, and, in the absence of obvious fractures.

improvement, most cases should be salvaged.

Treatment

Dislocations and subluxations

Manipulative reduction should be attempted in all uncomplicated cases of craniodorsal luxation seen

Hip joint

within 24 hours of the onset. The chance of successful manual reduction decreases markedly thereafter. Cases

Aetiology

of caudoventral dislocation, which are often recumbent, Coxofemoral luxation is sporadic, occurring mostly in perhaps due to additional obturator damage, have a two to five-year-old cattle. It has an association with poorer prognosis.

parturition and the early postpartum period. At this

The common craniodorsal luxation is treated by time, ligamentous relaxation is maximal and obturator careful positioning of the patient so that the body is or other nerve injury during dystokia may predispose fixed while the affected upper leg can be extended in cattle to abduct the hindlimbs and to slip the feet

various directions. Deep sedation (xylazine) is advisable, resulting in a splayed-leg collapse.

The leg is forcibly circumrotated in ever-increasing

Hip dislocation can occur in various directions,

moving circles by an assistant to loosen up the periarticular

craniodorsal being most frequent (about 80 per cent

soft tissues, which may be in spasm. A large, firm block

of total), and cranioventral and caudoventral being less

is placed between the ground and the medial femoral

common.

region to act as a fulcrum when inward pressure is

exerted on the stifle, tending to cause hip abduction.

The limb is subject to controlled traction along the

Pathology

longitudinal axis of the limb. The veterinarian should

The gross pathological changes in luxation include

concentrate on distal pressure on the greater tro-

rupture of the joint capsule and of the ligament of

chanter. The amount of femoral head movement

the femoral head (teres ligament), loss of articular car-

usually increases with time, and manipulation should

tilage and surrounding soft tissue, haemorrhage and not be quickly abandoned. Reduction is usually very oedema.

obvious, and traction should be stopped at once, otherwise a caudoventral luxation may be produced. Once replaced, the limb is circumrotated slowly and carefully

Clinical signs and diagnosis

in an attempt to remove blood and other debris from

A sudden onset of lameness is the only consistent sign.

the acetabulum. The animal should not be permitted to

Other so-called typical signs refer only to the common

stand at once, since spontaneous relaxation is liable.

craniodorsal luxation, when the leg is held rotated out-

The hindlimbs should be shackled above the hocks or

wards, the hock medial, and stifle more lateral than

fetlocks for 24–48 hours. A good non-slip surface should

normal. The leg may appear shortened and the greater

be beneath the cow when standing up and, if available,

femoral trochanters are asymmetrical, the affected side

a hip hoist is a useful aid.

being relatively more dorsal. Movement of the limb is

A high success rate has followed open surgery for somewhat restricted and painful. Crepitus may be reduction of hip dislocations (Tulleners et al., 1987). In

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this series few cases responded to manipulation. As region. The dropped spine may develop over two to experienced by others, caudoventral and cranioventral three days and be preceded by evidence of localized dislocations present special problems since limb traction cannot be exerted in the correct direction. The promontory has been pushed caudally and depressed, standard surgical approach is medial and cranial to the so reducing the dorso-ventral diameter of the pelvic greater trochanter through the gluteal musculature. inlet. Very severe cases may be unable to stand as a Care should be taken to avoid the sciatic nerve. The result of spinal nerve trauma.

femoral head is mobilized and moved appropriately In cases of subluxation and distortion, the signs are with a combination of manipulation by a non-sterile

relatively mild. Some hindlimb ataxia and weakness
assistant handling the hock, which can be easily rotated
may appear. Crepitus may be noted during rectal pal-
slightly or, at will, abducted, and moved by appropriate
pation as the cow is walked forwards, and localized
instrumental leverage. Toggling through the femoral
swelling over the ventral part of the joint is diagnostic
head and acetabulum to produce an artificial
when the history indicates a sudden onset.

intra-articular ligament was unsuccessful in a long-
Differential diagnosis includes coxofemoral osteo-
term study.

arthritis, pelvic fractures, obturator or other nerve
Recumbency following hip dislocation and for
damage, lumbar spondylitis and progressive hindlimb
a period after successful reduction necessitates a careful
paralysis (spinal abscess, lymphoma).

watch for the possible development of acute severe
mastitis, since many cases have recently calved. Excel-

Treatment and prevention

lent nursing in good comfortable surroundings is essen-

tial for recovery.

The prognosis is favourable for cases of distortion and

Apart from a surgical series (Tulleners et al., 1987) in subluxation that are confined to a well-bedded stall for

which many animals were immature and, therefore,

a few days. More severe, recumbent cases of luxation or

better surgical candidates, most cases have a guarded

subluxation have a guarded or poor prognosis. A

prognosis for craniodorsal dislocation and a poor prog-

sacroiliac luxation never undergoes spontaneous repo-

nosis for any other direction. Recumbency at presenta-

sition and manual reduction is impossible. However,

tion makes recovery less likely.

some cows can survive for years with sacroiliac luxation

Hip dislocation is the clinical diagnosis in some

despite the bizarre appearance of the hindquarters.

‘downer cows’ (see p. 439).

In nursing affected cows, no forcible attempt (hip

clamp, electric goad) should be made to make them

stand. Analgesics (phenylbutazone) every second day

Sacroiliac luxation and subluxation

for one week may increase comfort.

Aetiology

Sacroiliac displacement involves a partial or complete separation of the fibrocartilaginous joint surfaces. The

Stifle joint: femoropatellar

aetiology usually involves excessive ligamentous flaccidity around parturition, when most cases occur. Some

Aetiology and pathology

cases involve a degree of dystokia. A condition of Patellar luxation may occur dorsally, medially and sacroiliac distortion is recognized when there is no displacement. Dorsal displacement (fixation) is seen in placement, but excessive mobility of the joint surfaces mature cattle as an intermittent condition, unassociated is detectable. In true luxation considerable haemorrhage and soft tissue damage occurs in the joint space is rare and is usually congenital. Anatomical defects and peripherally (haematoma formation).

predisposing to medial luxation have not been clearly identified, though in newly calved heifers with large

udders the abducted hindlimb posture may lead to tem-

Clinical signs and diagnosis

porary medial patellar luxation.

Signs of sacroiliac luxation are characteristic. The

Lateral patellar luxation is occasionally seen as a

lumbosacral spine is dropped relative to the sacral

specific entity in mature cattle and young calves. This

tuberosities of the ilium, which are correspondingly

luxation in calves may sometimes be secondary to

prominent. Cows initially prefer recumbency and show

quadriceps atrophy resulting from femoral paralysis

slight ataxia, hindquarter weakness, and some knuck-

(see p. 437). In calves that first show signs at three

ling of the hind fetlocks. These signs are attributed to

months (Weaver & Campbell, 1972) without a history

associated bruising of the ventral nerve roots of L5–S2.

of femoral paralysis, no predisposing factors have been

Crepitus can be easily elicited from the sacroiliac

identified.



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week. Surgery is preferably carried out in the standing case as an aseptic procedure following sedation and local analgesia. A large udder or a difficult temperament makes recumbency (affected side down) necessary in some cases. The surgical site is superficial to the most distal palpable point of the medial patellar ligament, close to its insertion into the tibial tuberosity.

A 4 cm (1 1/2 inch) long vertical skin incision is made cranial to the ligament. A tenotome is inserted just deep to the ligament with the blade in a vertical position. A Hey Groves knife, with a blunt rounded point, is ideal for this surgery. The conventional disposable scalpel blade and (Bard) handle are unsuitable since slight movement easily results in breakage and loss of the

blade. The cutting edge is rotated through 90° towards the skin and the medial patellar ligament is sectioned

Fig. 32.6

Upward patellar fixation in 15-month-old Canadian Holstein heifer. The degree and duration of caudal extension without damaging or entering the joint cavity. A single were unusual. Heifer recovered following medial patellar non-absorbable skin suture achieves adequate wound desmotomy.

apposition. The operated cow should walk normally at once. No deleterious long-term pathology has been reported in operated cattle.

Both medial and lateral patellar luxation are treated

Clinical signs and diagnosis

by a joint overlap procedure. In lateral luxation the

In dorsal patellar luxation or fixation, the first sign may

femoropatellar joint is incised longitudinally about 1 cm

be initial hindlimb stiffness followed by a jerky action

(1/2 inch) medial to the patella, which is replaced in the

in which the limb remains extended caudally longer

trochlea. The capsule is closed by vertical mattress

than usual, and is then pulled forwards and upwards in sutures in a joint capsular overlap procedure. Sometimes a movement resembling equine stringhalt (Fig. 32.6). This action may be intermittent and separated by position. The fascia of the thigh may then be split dorsal to the femur, and the animal is pushed backwards, the hindlimb may become operated with a one week interval (Husband & Weaver, 1995). In medial patellar luxation, the overlap procedure is carried out lateral to the patella.

(permanent upward fixation). Palpation reveals the patella to be more prominent and dorsal than usual, and the patellar ligaments are unusually easily felt. The

Stifle joint: femorotibial

patella is now resting partly in the supratrochlear fovea,

Aetiology and pathology

while the medial patellar ligament is felt to be fixed

around the medial trochlear ridge.

Complete femorotibial luxation is rare and incurable.

Both medial and lateral patellar fixation are associ-

*Subluxation is relatively common and is typically seen
ated with a characteristic posture. The stifle is markedly
in the mature cow or bull as a result of primary cranial
flexed, and the limb collapses on weight bearing, exactly
cruciate injury (see p. 455). Predisposing factors include
as in femoral paralysis (p. 437). Bilaterally affected calves
heavy weight and sudden twists and falls as when
prefer recumbency. The patellar position is obvious visu-
mounting or being mounted by cows in oestrus. The
ally and on palpation. It can usually be replaced on to the
cranial cruciate ligament is partially or completely rup-
femoral trochlea but may immediately relaxate. Radi-
tured (Fig. 32.7). Damage to the caudal cruciate is less
ography can confirm the abnormal position but is a
likely. One or both tibial menisci are often torn and dis-
superfluous procedure. Differential diagnosis includes
placed at the time of rupture (Fig. 32.8). Secondary
femoral nerve paralysis, quadriceps muscle rupture,*

*osteoarthritic changes quickly develop with initial loss
gonitis and distal femoral epiphyseal separation.*

*of articular cartilage from the femoral condyles,
followed by exposure and erosion of subchondral
bone and peripheral osteophyte proliferation at the*

Treatment and prevention

joint margins.

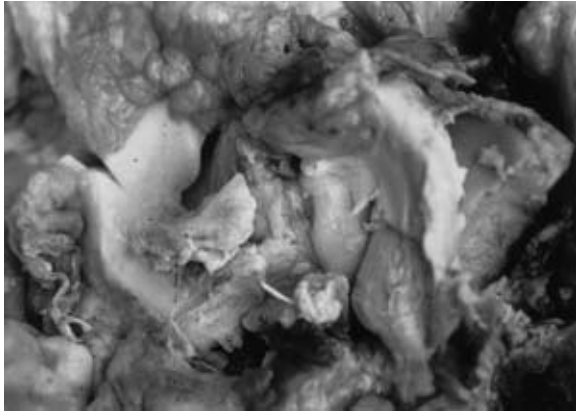
Dorsal patellar luxation should be treated by medial

Detachment of the medial meniscus has been

patellar desmotomy if signs persist for more than a

reported in young (1 month to 1 year) dairy cattle





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Fig. 32.8

Degenerative osteoarthritis of stifle joint of aged cow

exposing damaged menisci and tibial condyles. Menisci are

eroded and partially detached, allowing partial eburnation of the underlying tibial condyles. Surrounding musculature is pale due to extensive fibrosis.

and if radiographic facilities are available, lateral views

of the stifle may demonstrate significant (approximately

2.5 cm (1 inch)) cranial displacement of the tibial

plateau relative to the femoral condyles. In some cattle,

arthrocentesis is advisable to rule out septic gonitis and

to demonstrate the presence of cartilaginous debris and

blood in the femorotibial joint. Other differential diag-

Fig. 32.7

Degenerative osteoarthritis of stifle of aged beef cow

notes include collateral ligamentous injuries, patellar

showing ruptured cranial cruciate ligament, massive osteophyte proliferation along margins of femoral trochlea and cartilaginous fracture, and, in young cattle, proximal tibial epiphyseal

loss on femoral condyles.

separation.

Treatment and prevention

following injury to the medial collateral ligament.

Radical surgery involving replacement of the ruptured

Mounting activity has been postulated as a major

cranial cruciate ligament by skin or synthetic material

aetiological factor (Nelson et al., 1990).

has occasionally been successful. These cases have

either been experimental animals in which the ligament

has been sectioned, or smaller animals in which surgery

Clinical signs and diagnosis

has been done very soon after the original injury.

A sudden onset of a medium degree lameness is char-

Most cases have lacked adequate long-term follow-up

acteristic of femorotibial subluxation due to cruciate

studies.

injury. There is generalized soft tissue swelling and

crepitus may be spontaneous or may be induced by

rotating the point of hock laterally and medially to

Fetlock joint: metacarpophalangeal

produce some rotation of the tibial joint surfaces. The

and metatarsophalangeal

tibial tuberosity may be unusually pronounced and a

Aetiology and pathology

‘drawer-forward’ sign may be demonstrable initially.

The animal, placed in lateral recumbency with the

Dislocation is rare. It invariably involves rupture of

affected limb uppermost, has the stifle put into moder-

both collateral ligaments as well as the intra-articular

ate extension. The clinician then attempts to move the

structures. Trauma is usually so severe that massive soft

proximal tibial region cranially, relative to the femoral

tissue injury exposes the joint surfaces. Subluxation

condyles. Sometimes this movement is even apprecia-

either in a medial – lateral or cranial – caudal direction

ble as the animal walks. If not diagnostic on palpation,

is seen sporadically.

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Clinical signs and diagnosis

screws can be drilled from the caudal surface of the calcaneus into the distal portion of the tibial shaft to

Clinical signs include a non-weight bearing stance, immobilize the tarsus for some weeks so that fibrous swelling, obvious gross displacement and crepitus on repair at the rupture site can take place.

manipulation of the distal extremity. Diagnosis is Cranial tibial (peroneus tertius) rupture is also traumatically easy. Differential diagnoses include epiphyseal separation in calves, and distal metacarpal, metatarsal upwards of the hindlimb by ropes strung over an overhead beam and followed by severe struggling. The animals with similar signs of gross swelling but crepitus may animal has a characteristic gait. While the hock is be hard to elicit. Subluxations can be successfully abnormally extended, the stifle remains flexed, in other reduced following sedation and extension. A support words the reciprocal stifle–hock action is lost. When bandage or cast should be applied subsequently.

standing, any abnormality may be hard to detect, but the limb can be extended caudally so that the metatar-

Treatment and prevention

sus and tibia form a straight line while the stifle remains flexed. The gastrocnemius tendon is then slack. The site

Treatment is usually hopeless. If the joint surfaces can

of muscle rupture varies from its origin in the extensor

be replaced to be congruent, a synthetic collateral liga-

fossa of the femur to the insertion and to the muscle

ment may be inserted by utilizing steel wire anchored

belly itself. Some area of painful swelling is apparent

to screws drilled into each epiphysis. Alternatively, the

and most cases respond to stall rest over a period of one

ligaments may be replaced with polypropylene mate-

to four weeks.

rial. It is usually impossible effectively to suture

Adductor muscle damage has been briefly discussed

together the ruptured ends. In young calves the syn-

under downer cow syndrome (see p. 439).

thetic material may require later removal to permit

Ventral serrate rupture is spectacular due to loss of

normal growth of the epiphysis, otherwise the material

its normal anatomical function of supporting the chest

is left in situ. Meniscopexy was successful in treatment in the form of a sling between the forelegs. The scapu-of 20 out of 27 young cattle with medial collateral liga-

lar cartilage projects above the level of the thoracic

mentous and medial meniscal injury (Nelson et al.,

spine and is readily palpated subcutaneously. The

1990).

prominence varies in degree, being more obvious when

weight is borne and less obvious in the forward swing

Muscle and tendon injuries

of the limb. Colloquially termed 'loose shoulder',

Channel Island breeds, especially the Jersey, appear

to be predisposed. This feature may reflect a smaller

Introduction

muscle mass relative to body weight. The aetiology in

The muscles most liable to rupture include the gas-

young cattle is possibly related to muscular dystrophy

trocnemius, the adductor group and the cranial tibial

(vitamin E and Se deficiency; see page 258). Cases may

(peroneus tertius). All three are particularly liable to

be associated with turn-out to pasture. Pathological damage when a heavy cow is struggling to rise postpartum examination of chronic cases in adult cattle reveals tum (see Downer cow syndrome, p. 439).

severe muscle degeneration, fibrous tissue proliferation and serous infiltration. While mild cases may recover completely, complete rupture is incurable, but animals

Specific muscles

move around well, remain productive and emergency

The gastrocnemius is particularly exposed to damage as slaughter is rarely necessary.

a primary extensor of the hock. Prolonged recumbency, excessive weight, and possibly mineral imbalance leading to a degree of osteomalacia can predispose to

Contracted flexor tendons

gastrocnemius rupture.

The common site is the tendon–muscle junction. The

Aetiology and pathology

rupture is usually complete and leads to considerable swelling due to extravasation of blood and the devel-

Contracted digital flexor tendons are the commonest

opment of oedema. The hock is dropped and weight
congenital abnormality in cattle. The name 'contracted
bearing is minimal. Diagnosis is rarely in doubt since
tendons' is a misnomer, as the primary abnormality is
the clinical picture is typical.

an arthrogryposis or articular rigidity, usually in flexion
Gastrocnemius muscle rupture is treated by stall rest
(Fig. 32.9). Most cases are mild and self-correcting as
and maintenance of the hock in extension by means
the calf moves around to an increasing extent. Some
of a Thomas splint. In yearling animals, compression
are more severe and sometimes associated with



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diameter polyvinyl drainpipe may be placed on a well-
padded limb to immobilize the flexed joints in

maximum extension. Such a splint in a neonatal calf must be checked by weekly removal and replacement. The toes should be left exposed to encourage weight bearing and walking. If the stance is not corrected in four weeks, surgery is indicated.

More severe cases will only respond to desmotomy, which should be undertaken at two to three weeks old, after the immediate neonatal stress period. Correction of carpal flexion is possible at the mediopalmar side of the carpus by an initial longitudinal incision through the retinaculum flexorum, exposure of the superficial part of the superficial flexor, and by complete transection of the retinaculum flexorum, which also involves section of the radial carpal flexor. If normal extension is not

Fig. 32.9

Contracted flexor tendons (forelimb) and flexed hocks achievable, the deep flexor and deep part of the superficial flexor are transected, preserving carefully the neurovascular bundle of median artery, vein and nerves.

Finally, the palmar carpal ligament may be sectioned.

*Exceptionally, with a radius–metacarpus angle $<100^\circ$, it
other congenital defects such as cleft palate and
may be preferable to perform radiocarpal arthrodesis
arthrogryposis (see p. 177).*

with resection of one or both carpal rows. The wound

*Contracted tendons usually affect both deep and
is closed by sutures and then bandaged and cast for five
superficial flexors, and sometimes the suspensory liga-
weeks.*

ments (see p. 178). The condition is bilaterally sym-

*Correction of fetlock flexion is similarly a multistep
metrical in the forelimbs. The hindlimbs may have
procedure. The skin incision is at the mediopalmar
abnormal hock extensor rigidity and fetlock flexion.*

aspect of the metacarpus, and the superficial portion of

Some breeds (e.g. Belgian Blue) have a very high

the flexor tendon is transected. The deep flexor and

incidence in which a relationship is suspected between

deep part of the superficial tendon may be sectioned

relatively excessive fetal size and abnormal intra-

similarly for adequate extension. Small stab incisions
uterine posture. Dystokia is almost invariable in such
may be made in the suspensory ligament. Bandaging
cases. Akabane virus and the ingestion of various toxic
and cast immobilization of the limb are again necessary.
plants (e.g. *Lupinus* species and locoweed) have been
Surgery is likely to be unsuccessful in very severe
alleged in other high incidence outbreaks.
cases of contracted tendons where abnormal flexion
creates an angle of $<90^\circ$
between radius and
metacarpus.

Clinical signs and diagnosis

Prevention of the primary aetiological stimulus is

*Mild cases show about 10–20° excessive flexion of the
difficult as arthrogryposis leading to contracted tendons
carpus and fetlocks. Forced extension discloses the taut-
may result from infectious agents (bovine virus diar-
ness in the flexor tendons and suspensory ligament. The
rhoea (BVDV), akabane virus) or toxic plant material.
abnormality is generally symmetrical. Some calves may*

have a split palate and arthrogryposis of the carpi and

Traumatic flexor tendon injuries

tarsi. Joints are not swollen and extension is not unduly

painful. When moved, mild degrees of abnormal flexion

Sporadic traumatic injuries occasionally affect the

will result in calves moving on the pastern, but, in severe

flexor tendons and often result from contact with farm

cases, the calf is totally recumbent or will walk on the

machinery. Adult cows may injure both deep and super-

distal skin of the fetlock and rapidly develop abrasions

ficial flexor tendons in the metatarsal region, and an

and cellulitis, perhaps allowing secondary infection to

open infected wound results. Careful debridement

establish itself and bring the risk of a septic arthritis.

under IVRA is indicated. A septic tenosynovitis neces-

sitates pressure lavage with sterile isotonic saline b.i.d.

for several days and systemic antibiotic cover. Selected

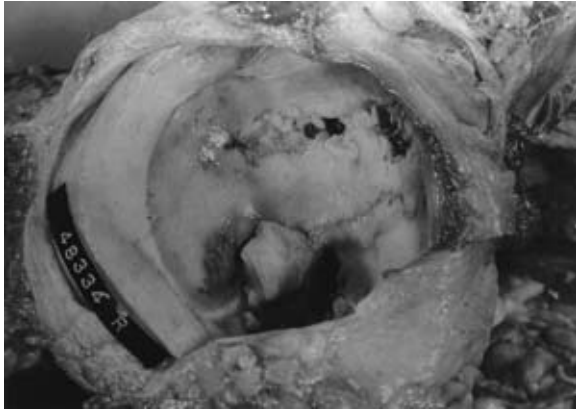
Treatment and prevention

cases of complete transection of both flexor tendons

Some cases resolve but in others splinting of the leg is

may heal following tenorrhaphy (e.g. carbon fibre or

normally sufficient. A half-section of a 5 cm (2 inch) polyamide) and application of a limb cast. Despite



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prolonged convalescence most cases continue to be lame

Treatment and prevention

and the prognosis is guarded (Anderson et al., 1996).

Theoretically, as in the horse and dog, removal of the cartilaginous flap and curettage of the underlying bone is indicated. Surgical treatment has, however, not been

Joint diseases

adopted in cattle for economic and anatomical reasons.

Management measures in high-incidence situations in-

Osteochondrosis

clude a more restricted calorie intake and transfer to a softer bearing surface to reduce concussion. Lam

Aetiology and pathology

steers should be confined. Osteochondrosis in a bull should stimulate consideration of its conformation as a Osteochondrosis (OCD) is seen in young fast-growing possible predisposing factor.

beef cattle on a high calorie intake. Recently an American series of 29 cattle with osteochondrosis was dominated by dairy breeds, primarily mature males (Trostle et al., 1997). Normal endochondral ossification

Hip dysplasia

is disturbed at the cartilaginous endplate of the epiph-

Aetiology and pathology

ysis and at the metaphyseal growth plate, and is associated with a failure of vascular invasion. The thickness

Hip dysplasia is seen in bulls of various fast-growing of articular cartilage increases as a result of continued beef breeds,

predominantly the Hereford and

growth and lack of wear, whereupon the failure of ade-

Aberdeen Angus, though isolated cases have been

quate nutrient diffusion causes a degeneration of the

reported in numerous other breeds. In the Hereford chondrocytes. The result is a characteristic cleft for-breed, particular families have been alleged to have a mation, producing cartilaginous flaps that undergo high incidence. The condition is probably a sex-linked endochondral ossification (osteochondrosis dissecans). heritable characteristic. Although some cases are Another process is the formation of subchondral cyst-observed in the neonate, most develop in calves four to like lesions.

twelve months old with erosion of the acetabular carti-It is not yet known to what extent the changes are lage close to the attachment of the accessory cartilage, generalized, as most surveys have only examined spe-which normally functions to extend the effective artic-cific joints (e.g. stifle, carpus or atlanto-occipital). ulating surface (Fig. 32.10). The femoral articular carti-Changes tend to be symmetrical. Sometimes the joint lage usually undergoes a shallower but more extensive surfaces of several forelimb bones are normal, while sig-erosion.

nificant changes occur in their physes. Osteochondrosis
Later changes include traumatic synovitis, fraying of
has been reported in the coxofemoral, femoropatellar,
the intra-articular ligament, and a tendency to subluxa-
femorotibial and tibiotarsal joints of the hindlimbs, and
tion. Secondary degenerative joint disease follows in a
in the scapulohumeral, humororadial, radiocarpal, and
few weeks.

metacarpophalangeal joints of the forelimbs.

Clinical signs and diagnosis

Steers and young bulls are predominantly affected.

Some lesions cause no clinical signs, while others
produce a mild progressive lameness. Some lesions heal
following production of fibrocartilage. This accounts for
the larger percentage of lesions in autopsies than in
clinical series of lame cattle. Other lesions progress to
become a secondary degenerative osteoarthritis or
degenerative joint disease (DJD). Exacerbations of a
mild slight lameness have been attributed to additional
joint trauma. If warranted, a doubtful case may be
radiographed, but cartilaginous defects are unlikely to

be demonstrated unless pneumoarthography is performed. Suspicious evidence of osteochondrosis is the

Fig. 32.10

Severe hip dysplasia seen in right acetabulum of presence of a free calcified body within the joint ('joint nine-month-old Hereford bull. Severe fissuring and loss of cartilage and erosions in subchondral bone.



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Degenerative arthritis

Aetiology and pathology

Degenerative arthritis (degenerative joint disease, DJD) is attributed to a 'wear-and-tear' phenomenon. This chronic non-infectious disease involves primary degeneration of articular cartilage, generally accompanied by secondary sclerosis and eburnation of sub-

chondral bone, peripheral osteophyte formation and surrounding soft tissue proliferation.

Though a common incidental finding at slaughter, its primary clinical significance is as a cause of progressive debilitating lameness involving major weight-bearing joints. In one study of the stifle and hip joints of cattle

Fig. 32.11

Severe hip dysplasia evident in ventrodorsal disease, atrophy frequently led to partial condemnation radiograph of pelvis of five-month-old Angus bull that had at slaughter (Weaver, 1977). A recent US study in 21 experienced progressive hindlimb lameness for two months. processing plants revealed that 11.9–16.5 per cent of Both femoral heads are subluxated and severe secondary beef and dairy cows and bulls had stifle arthritis osteoarthritis affects the acetabular rims.

(‘stifled’) or a broken leg. The stifle injury resulted in the loss of 17.9 kg tissue from trimming (\$9.72 per carcass) (Roeder et al., 2000). Other studies have shown the frequent involvement of the hock and carpal joints.

Clinical signs and diagnosis

Overweight and straightlegged cattle may be predisposed to early DJD but control studies are lacking. Lameness develops slowly and is preceded by a period in which abnormal lateral swinging of the hindquarters is seen. An inherited DJD of the stifles of Holstein cattle has been reported (Kendrick & Sittmann, 1966). Nutritional imbalance leading to osteochondrosis starts. Unilateral or bilateral crepitus is detectable in 453), which can lead to secondary DJD, is another aetiological possibility. rectal palpation is feasible. The development of subluxation is recognizable from demonstration of the Ortolani sign: with the hand resting on the skin over the greater femoral trochanter, when weight is taken off affected, followed by the opposing femoral condyle.

the hind leg, a distinct 'plop' is felt as the femoral head

The age range was six to eighteen months.

drops into the acetabulum from its previous position on

Early fibrillation of articular cartilage leads to

the dorsal acetabular rim.

necrotic and degenerative chondrocytes, which results

In smaller calves, ventrodorsal pelvic radiographs

in decreased proteoglycan production in the cartilagi-

may demonstrate secondary joint changes and a ten-

nous matrix. The cartilage becomes soft and somewhat

dency to subluxation (Fig. 32.11). Any radiographic

yellowish. Chondrocyte destruction causes release of

suggestion of shallowness of the acetabulum is usually

lysosomal enzymes, especially cathepsin D. Fibrillated

the result of the secondary changes and not a primary

cartilage withstands stress relatively poorly, and fissur-

anatomical feature.

ing, thinning, or cartilaginous erosion follows.

Differential diagnosis is important in apparently uni-

Pain in DJD originates from the exposed subchondral

lateral cases. Stifle disease, acetabular and other pelvic

bone and from the associated capsulitis and synovitis.

fractures, hip luxation and slipped femoral epiphysis are

Clinically, the joint enlarges as a result of the increased

all to be considered. A confirmed bilateral hip lameness

volume of synovia and soft tissue proliferation.

in young bulls is usually diagnostic of hip dysplasia.

Clinical signs and diagnosis

Treatment and prevention

A progressive lameness with gradual muscle atrophy

No successful treatment has been reported. Affected

and weight loss is commonly seen. The affected limb,

bull calves and yearlings should be slaughtered. The hip

usually hind, tends to be dragged as flexion of the

joints should be retained for pathological confirmation

affected joint is reduced. Movement of the stifle or hock

of the clinical diagnosis.

results in palpable joint enlargement. In the stifle,

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proliferative changes (osteophyte formation, periartic-

tides and is considerably more expensive. Prolonged

ular fibrosis) affect both femoropatellar and femorotib-

low-dosage therapy with phenylbutazone in cattle with
ial compartments, and crepitus may be noted. Some
painful arthritides has not led to any demonstrable
cases of stifle DJD reflect a secondary response to
deleterious effects such as gastrointestinal ulceration or
cranial cruciate rupture or meniscal injury. In the hock,
renal papillary necrosis. Corticosteroids and dimethyl
major changes are usually seen craniomedially, affect-
sulphoxide (DMSO) have also been advocated. The
ing the intertarsal and tarsometatarsal joint space
majority of young cattle with stifle lameness due to sub-
(similar to equine 'spavin'). Relatively rarely do
chondral bone cysts formation recovered following con-
changes extend proximally to the tibiotarsal joint.
servative treatment (Ducharme et al., 1985). Preventive Degenerative joint
disease of the hip can be suspected
measures include attention to the conformation of
in older cows when the more distal parts of the limb
further breeding stock, especially bulls, to avoid a
are normal clinically and where crepitus can be
hindlimb conformation that is liable to lead to exces-

*detected over the gluteal region and confirmed on
sive concussive forces in progeny.*

rectal palpation.

*Subchondral bone cysts can only be diagnosed on
radiographic examination. Cases of DJD, ligamentous*

Infectious (septic) arthritis

*rupture and septic arthritis show ranging radiographic
features. Degenerative joint disease of the stifle and*

Introduction

hock shows the extent of the proliferative reaction.

*Infectious arthritis in this context is synonymous with
Lateral radiographs of the stifle in rupture of the cranial
septic arthritis. The rare primary septic arthritis devel-
cruciate ligament have the 'drawer-forward' features
ops from direct joint penetration by a contaminated
seen in dogs. Septic arthritic cases on radiography
foreign body. This section considers secondary septic
demonstrate soft tissue swelling and distension of the
arthritis resulting from spread of pathogens from an
joint capsule in early cases and, when more advanced,
adjacent localized focus, and tertiary septic arthritis*

*the radiographic changes include loss of subchondral
resulting from systemic or haematogenous spread from
bone.*

a focus elsewhere in the animal.

*Synovial fluid analysis is only justified when the dif-
ferential diagnosis includes infectious arthritis or when
fluid is withdrawn prior to injection of a local analgesic*

Septic arthritis: joint ill in neonate (see p. 249) *solution to assess whether
lameness is lessened.*

Aetiology and pathology

*Synovia in DJD is clear or slightly turbid, increased
in volume, does not clot, has a low (<15 per cent)*

*Joint ill in calves is classified as a tertiary septic arthri-
polymorphonuclear lymphocyte count,
a normal*

*tis. The primary infection is in the umbilicus, often as an
protein (<2 g/dl), and a near-normal mucin precipitate
umbilical abscess with extension along the umbilical
(Weaver, 1997).*

*veins towards the liver. Otherwise, spread is from a
Differential diagnosis, apart from infectious forms of*

primary enteric infection. Common pathogens isolated arthritis, includes fluorosis as a primary cause of DJD, are *Escherichia coli*, *Streptococcus* and *Staphylococcus* osteoporosis, rickets and osteoarthritis deformans, all spp., *Arcanobacterium pyogenes*, *Erysipelothrix* in- three of which occur in young growing animals, and *sidiosa* and *Salmonella* spp. Usually, more than one osteomalacia. A sudden onset of lameness sometimes joint is affected, commonly the carpus, tarsus and represents an exacerbation of a non-clinical DJD following a periarticular fracture (e.g. caudal surface of femorotibial joints. The route of haematogenous spread is through metaphyseal or epiphyseal vessels or, alternatively, the synovial membrane. Establishment of foci of infection is naturally favoured by the slowed flow rate of blood through a network of venous sinusoids in the metaphyseal vessels.

Treatment and prevention

Treatment is purely palliative. Affected cattle should be Pathological changes start with an intense polymor- rested. Ketoprofen (10 per cent solution iv or im) has

phonuclear inflammatory response in the synovial recently been shown to have analgesic properties in membrane, the permeability of which is increased, per-lame cattle. Phenylbutazone may be given (initially mitting protein leakage into the synovial fluid. Later 5 mg/kg orally daily, then 5 mg/kg every second day) release of lysosomal and other enzymes into the for its analgesic and anti-inflammatory properties. synovia causes degeneration of the articular cartilage, Aspirin (100 mg/kg orally b.i.d.) is an alternative which is weakened by defective nutrition due to an regimen. Flunixin meglumine has few obvious advantages over either phenylbutazone or aspirin for arthritic articular cartilage, acute synovitis, thickening of the



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colostral intake) are at higher risk. Lameness becomes severe after a few days.

Radiology is not a great aid to diagnosis, as the only initial abnormality is the soft tissue swelling. After two to three weeks, changes suggestive of joint destruction are apparent.

Arthrocentesis is a more valuable aid to diagnosis in early cases. Apart from an increased volume, synovial

fluid in a calf with joint ill is turbid, clots, and has a loose flaky character in the mucin precipitate. The protein

content is >5 g/dl and a Giemsa smear reveals an almost pure (98 per cent) and massive population of polymorphonuclear lymphocytes. Taken aseptically, synovial fluid may be submitted for culture, but is often problematical with this particular material (see p. 457). Differential diagnosis must be made from physeal and intra-articular fractures.

Treatment and prevention

Early and vigorous treatment is essential in joint ill. High therapeutic levels of systemic antibiotics should be given for two to three weeks. Intra-articular antibiotic medication is usually contraindicated, as the blood supply ensures adequate concentrations are obtained in synovia following systemic injection. Suitable antibiotics include penicillin, ampicillin and tetracyclines. Joint lavage with a sterile polyionic solution or

Fig. 32.12

Extensive soft tissue swelling of lateral aspect of left physiological saline is helpful. Generally, an in-out, carpal region in a six-month-old Hereford calf. Bone lysis is through-and-through system using two 14 gauge

evident in the distal part of the radial carpal and fused second needles is effective. Open arthrotomy with removal of

and third carpal bones. Lysis resulted from transverse fracture the fibrin clots and purulent material along with curet-of the proximal surface of the distal component of the joint.

tage of the articular surfaces has been successful in

Recovery followed removal of the bone fragment, curettage

some series. It appears more useful in less complex

and irrigation.

joints such as the stifle and fetlock rather than in carpus

or tarsus.

Recently intra-articular therapy with gentamycin-

impregnated polymethylmethacrylate beads has proved

joint capsule, and sepsis in the growth plate and

useful in neonatal septic arthritis with concurrent

metaphysis (particularly associated with salmonella

osteomyelitis. Such beads must be removed at a second

infection).

surgery. Use of gentamicin-impregnated collagen

sponges (Garamycin,

Essex Chemie,

Lucerne,

Switzerland) which do not need later removal appear

Clinical signs and diagnosis

more promising, and further larger studies are awaited

The first sign is lameness. Later, joint swelling (Fig.

(Steiner & Zulauf, 2000).

32.12) and pain are apparent. The joint capsule may be

Supportive care is important. Casting for three weeks

distended from an increased synovial volume. The pres-

of joint immobility (e.g. in fetlock) followed by band-

ence and physical nature (serous, fibrinous or purulent)

aging for support reduces discomfort and so maintains

of such synovial effusions can be readily detected on

the appetite. Low level oral dosage with phenylbuta-

ultrasonography (7.5 mHz linear transducer) in carpal,

zone can improve the general attitude (p. 1050). Some

stifle and hock disorders (Kofler & Martinek, 2000).

calves also require management of the enteritis by fluid

The polyarthrititis is rarely symmetrical. Not all cases are

therapy (see pp. 195, 209) and others require drainage

associated with either gross umbilical sepsis or with

of an umbilical abscess. Inadequate levels of gamma

severe enteritis. Calves that are immune depressed as globulins may, with difficulty due to the large volumes, a result of low gamma globulin levels (inadequate be corrected by giving intravenous plasma.



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Septic arthritis: older cattle

coccus and Streptococcus spp. Fusobacterium necrophorum and Bacteroides melaninogenicus (anaerobe) Aetiology and pathology

have occasionally been reported. The major problem

The main site of septic (infectious) arthritis in mature

is A. pyogenes. Once infection is in the synovial

cattle is the distal interphalangeal joint resulting from fluid, joint destruction involves the same processes as secondary spread from a focus in the sensitive solar described for DJD. The difference lies in the speed of laminae or from complications of interdigital necrodestruction. The synovia is usually turbid or frankly bacillosis (see pp. 420 and 426).

purulent.

Septic digital arthritis is fully discussed elsewhere (p.

Some reports from the UK (Bracewell & Corbell,

455). Other joints liable to be affected by septic arthri-

1979; Wyn-Jones et al., 1980) and USA (Madison et al. , tis include the fetlock, either by extension along the

1989) noted an idiopathic, acute suppurative gonitis

digital flexor sheath from a digital focus, or by direct

with severe lameness and synovitis. Articular erosions

trauma from a deep wound (Fig. 32.13). The major

were evident radiographically on the lateral tibial

hindlimb weight-bearing joints, the tarsus, stifle and hip

plateau. Much evidence indicates the likelihood that

are occasionally involved as a result of haematogenous

Brucella abortus vaccination may have been the cause.

spread such as pyaemic vegetative endocarditis (e.g.

from primary reticuloperitonitis) or in the tarsus

Clinical signs and diagnosis

from contiguous spread from an infected subcutaneous

bursitis.

Septic arthritis in a major weight-bearing joint causes

Organisms commonly recovered from septic arthritis

a rapidly progressive disease with severe lameness,

in adult cattle include *A. pyogenes*, *E. coli*, *Staphylo-swelling* and localized pain.

In contrast to DJD, the

animal may be anorexic and milk yield can drop

abruptly in dairy cattle. Crepitus is not usually appar-

ent. Arthrocentesis reveals a flocculent or purulent fluid

that clots. It has a high leucocyte count of which over

90 per cent are polymorphs. The protein content often

exceeds 8 g/dl and the mucin precipitate is abnormally

flocculent.

A category 'suspect septic' refers to an early septic

process associated with obvious lameness but appar-

ently normal yellow synovia. The presence again of a

high percentage of neutrophils in a dense leucocyte population is confirmation of early septic arthritis.

Radiography in early (<10 days duration) septic arthritis is usually unhelpful. Ultrasound, on the other hand, can delineate the limits and physical nature of the fluid. From 14 days onwards, subchondral bone destruction and a peripheral periostitis are suggestive of joint sepsis.

Culture of synovial fluid is frequently negative in terms of isolation of pathogens, especially if antimicrobial therapy has already started. It is helpful if synovia is immediately inoculated into a diphasic culture bottle with sodium polyanetholsulphonate to enhance recovery of the organism. Culture of synovial biopsy material is usually more rewarding but is frequently impractical.

Treatment and prevention

The prognosis for septic arthritis is guarded or poor.

Early aggressive treatment is essential. Parenteral

Fig. 32.13

Massive periosteal proliferation of fetlock region

(metatarsophalangeal joint) of a four-year-old Holstein cow

antibiotic therapy should be initiated as soon as pos-

resulting from sepsis of the joint originating from an ascending sible. Until sensitivity, based on synovial cultures,

septic tenosynovitis.

is available 48 hours after sampling, penicillin or

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ampicillin is the drug of choice for most pathogens.

This spasm leads to severe extension of the hock. The

Penicillin is most likely to be effective in vivo against condition has been observed in numerous breeds, but

all common pathogens except *Staphylococcus aureus*

the highest incidence in dairy and beef cattle respec-

(where erythromycin, lincomycin, gentamicin, or

tively is probably in the German and Dutch Friesian

cephalosporin may be better) and coliforms (gentam-

and the Aberdeen Angus.

icin or trimethoprim–sulphadiazine).

It is hypothesized that an overactive stretch reflex is

The joint should be irrigated and drained (through-

the major mechanism (De Ley & De Moor, 1977).

and-through lavage) through two wide-bore

Selective deafferentation of the gastrocnemius muscle inflow–outflow portals produced by 14 gauge needles. by resection of the dorsal root fibres containing affer- Alternatively, small arthrotomy incisions may be made ent nerve fibres (L5–6) abolished clinical signs. Plasma for the same purpose. Irrigation should be done with SGOT is reduced, and alkaline phosphatase is increased large volumes (>5 l) of polyionic solutions, physiologi- in affected calves, which also show a decreased P, Ca, cal saline, or with dilute (0.1 per cent) povidone-iodine and homovanillic acid concentration in CSF. The last- solution.

named effect indicates a lowered metabolic rate of CNS Radical surgery also has a place if suitable facilities dopamine (De Ley & De Moor, 1975).

exist for the maintenance of asepsis and for general Gross and histopathological CNS lesions have gen- anaesthesia. Radical surgery comprises opening the erally been absent.

joint following section of collateral ligaments and inci- sion of joint capsule, evacuation of purulent material,

Clinical signs and diagnosis

curettage of infected cartilaginous surfaces, restoration of joint stability by suturing capsule and ligaments, and

The typical clinical signs are characteristic, and difficult immobilization of the area, if in the distal limb, to

culty arises only in very early cases. Signs may start at prevent ankylosis. Such surgery appears more success-

six weeks to six months, rarely later. Cases encountered ful in the fetlock, carpus and tarsus than in the stifle,

in adult cattle are best considered to be forms of pro-elbow, or shoulder joints.

gressive hindlimb paralysis, likely to involve spinal cord

Idiopathic gonitis (p. 1014) carries a good prognosis pressure resulting from vertebral exostoses.

for recovery, regardless of the method of treatment.

The affected hindlimb is extended so that the hock

Heifers were treated successfully with a variety of

angle is approximately 180° (normal is about 140°). The

antimicrobials, including procaine penicillin G, neomycin

calf walks with a tendency to drag the toes. The limb may

and ampicillin, and, in some cases, with through-and-

jerk intermittently at rest. It is advanced in a pendulum-through needle lavage with a polyionic solution. Lateral like style. Later, less weight is taken and the calf may hop patellar arthrotomy with curettage of lytic areas on the forward. Palpation reveals a very firm gastrocnemius lateral tibial plateau may be useful adjunct therapy. muscle and tendon. The hock can be flexed manually There is no place for intra-articular antibiotics. without causing pain. On release the hock immediately Drainage of infective material from a poorly vascular- adopts the original extended position (Fig. 32.14). ized area is important, whereas systemic antibiotics ade- Lateral radiographs of the hock of calves affected for quately penetrate the well-vascularized joint capsule. several weeks show several features indicative of the Antibiotics should be given for at least two weeks. chronic overextension. The distal part of the tibial Preferred antibiotics are usually penicillin G, ampi- metaphysis is abnormally curved caudally; the distal cillin, ceftiofur and gentamicin. Non-steroidal anti- tibial and tuber calcanei growth plates are widened;

inflammatory drugs should be given if pain is severe and exostoses are present around the distal tibial growth persistent. The dosage is as for cases of DJD (see p. 455). plate, along with some osteoporosis. Often the most striking feature is a cranial curvature of the proximal part of the calcaneus.

In early cases, differential diagnosis includes dorsal

Miscellaneous neuromuscular

patellar luxation, septic or non-infectious gonitis or tar-
diseases

sitis, fracture dislocation of the calcaneus, joint ill and luxation of the biceps femoris muscle.

Spastic paresis (see also p. 179)

Aetiology and pathology

Treatment and prevention

Spastic paresis is a progressive condition of the hind-

Since spastic paresis has a hereditary predisposition, limbs of unknown origin. It is characterized by con-
breeding animals should be salvaged. Surgery can suc-
traction of the gastrocnemius muscle and tendon and of
cessfully alleviate the condition by either tenotomy or

other associated calcanean tendons and muscle bellies.

neurectomy.



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muscle is awkward, complete tibial neurectomy has been advocated (Boyd & Weaver, 1967). Surgery is performed under sedation and epidural block, or general anaesthesia. The site is the lateral thigh, between the two heads of the biceps femoris muscle. The tibial and peroneal nerves are identified by stimulation by forceps or by electrical means. Tibial nerve stimulation causes hock extension and fetlock and digital flexion. The tibial nerve should have a 2 cm (1 inch) portion removed at its most proximal point. Complications following this surgery are uncommon.

Spastic syndrome

Aetiology and pathogenesis

Fig. 32.14

Severe spastic paresis of left hindlimb of a four-month-old Friesian heifer, showing overextension of hock

This chronic progressive disease, which is initially characterized by clonic–tonic spasms of the hindlimb as calf attempts to put more weight on the forelimbs.

musculature, is also known as ‘crampy’, ‘stretches’ or progressive hindlimb paralysis. The aetiology is unknown despite several attempts to find significant pathological lesions in the brain or spinal cord. Idiopathic muscular cramps is one current proposed explanation. Tenotomy has traditionally involved section of the gastrocnemius tendon and partial section of the superficial flexor tendon a little proximal to the point of the hock in the standing animal under local anaesthetic

The condition occurs in mature cattle of many breeds, both dairy (e.g.

Holstein, Friesian and

hock in the standing animal under local anaesthetic

Guernsey) and beef (Hereford, Angus). The incidence of infiltration. A 6 cm (2 1/2 inch) vertical skin incision is made caudally over the Achilles tendon to expose the higher in bulls than cows. Many affected cattle have two tendons. After section, the skin is sutured and the straight rear legs and poor hock conformation. Many immediate surgical effect is usually pronounced hock flexion, with the joint initially close to the ground.

single recessive gene with incomplete penetrance may Initial results have been consistently favourable, but a be the mode of inheritance.

gradual recurrence of the abnormal posture is frequently seen, especially in older (six to ten-month-old) calves. Bilaterally affected calves should have the

Clinical signs and diagnosis

second leg operated four weeks following initial surgery.

Sudden episodic spasmodic contractions affect the

It has been claimed that this classical operation

muscles of both hindlegs, and sometimes also the back, ignores the fact that a second, deeply situated, tendon neck and forelegs. The animal appears to be normal of insertion of the gastrocnemius muscle is left during recumbency. Signs are evident soon after rising. untouched. A modified operation, therefore, involves a The onset of the syndrome is slow, and signs may lateral approach just cranial to the Achilles tendon, appear for a few months only to disappear for several which is dissected to isolate the caudal component of weeks. The hindlimbs are hyperextended caudally in a the gastrocnemius tendon, from which a 2 cm (1 inch) fixed manner that may persist for several minutes. length is resected. The superficial flexor tendon is left Spasms may extend forward to the forelimbs and neck untouched. The cranial tendon of the gastrocnemius is muscles. The head is extended and the forefeet identified and a similar tenectomy performed. In some advanced with the back arched. If then forced to move, cases, the dense fascia caudal to the distal end of the the gait is stiff and ungainly, and episodic lifting of the

tibia is transected (Pavaux et al., 1985).

hindlimbs may also be seen during forward movement.

Neurectomy of the tibial nerve or of its gastrocne-

Animals generally spend considerable periods

mus branches is the alternative to tenotomy. Clinical

recumbent when the condition is advanced.

results have been better than with tenotomy. Since

Other conditions that may confuse an initial diagno-

identification of the multiple (seven or more) branches

sis are relatively few but include bilateral DJD affect-

of the tibial nerve innervating the gastrocnemius

ing the stifle or hock, and severe spinal spondylosis.



Treatment and prevention

pressure points and poor vascularity, may result in localized abscessation and pyaemic spread to the lungs, The spastic syndrome is incurable. Palliative treatment liver, kidneys and heart. The common organism is A. has included vitamin D, bone meal (not available in pyogenes.

many countries) and sedatives. Phenylbutazone has Affected cattle should be put out on soft bedding, provided temporary relief in some cases. Affected bulls the wounds should be cleansed with chlorhexidine and apparently normal bulls that sire affected progeny hydrochloride, and systemic antibiotics should be should be culled from any breeding programme. injected for five to seven days to prevent systemic spread. The prognosis for healing is good if appetite is

Diseases of skin and subcutis

maintained unless clinical signs of involvement of other organs (e.g. heart) are apparent, and suggestive of septicæmic spread.

Skin wounds are more liable around the limbs than the

trunk and head. Their nature and treatment are adequately discussed in textbooks of general surgery. One

Tarsal cellulitis

unusual bovine problem is the constricting foreign body, usually rubber or wire, which slides over the foot

Aetiology and pathology

to the level of pastern or metacarpus or metatarsus

Almost all cases of hock enlargement result from

where it slowly becomes embedded in a mass of gran-

repeated trauma against concrete surfaces in stanchions

ulation tissue to present as a circumferential wound

or loose housing. Typically, soft tissue swelling over the

(Fig. 32.15). Such wounds require careful investigation

lateral aspect of the tarsus may be a false or acquired

because the band may be very deep and contacting

bursitis. Medial swelling of the joint is minimal unless

bone.

the lateral enlargement is pronounced. Lameness is

Two specific disorders are discussed below. Gener-

rare unless the fluid distension becomes massive, when

ally, damage to skin and subcutis is more liable over

mechanical restriction of joint flexion causes stiffness, pressure points such as the lateral aspect of the stifle, or a low-grade infection arises. Radiographs have elbow and hock. Loss of the integument permits low-repeatedly shown the absence of bone changes. grade infection to become established. In a very con-

The swelling is usually symmetrical. Hair loss and taminated environment such damage, arising from skin excoriation are evident. Usually the skin is not broken. Occasionally, a central area of skin sloughs, permitting a dirty red–brown material to escape. At this time, septic infection can supervene and a purulent discharge may be noted for several days before granulation tissue fills the cavity of the false bursa.

Clinical signs and diagnosis

The distribution of the swelling and absence of lameness are almost characteristic (Fig. 32.16). An infectious tarsitis causes a more diffuse swelling and obvious lameness, as will a tarsal bone fracture. Tarsal hydrarthrosis first causes synovial distension of the joint capsule cranially and medially, then both medial and

lateral to the caudal border of the tibiotarsal bone, and cranial to the base of the calcaneus. Tarsal hydrarthrosis is a cosmetic blemish and causes no lameness.

Treatment and prevention

The seasonal occurrence of tarsal bursitis, becoming increasingly severe during the housed period and disappearing in the summer grazing months, is evidence

Fig. 32.15

Circumferential wound of metatarsal soft tissues.

This two-year-old crossbred Hereford steer had a wire embedded deep to the soft tissues, resulting in mild lameness. Wound Errors and deficiencies usually can be found in the

healed in four weeks following removal of foreign body.

lying-in areas of loose-housed herds. Some cows may





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Fig. 32.16

Right tarsal bursitis in a five-year-old Holstein cow

Fig. 32.17

*Severe bilateral carpal bursitis in an eight-year-old
with digital problems resulting in prolonged recumbency. Left*

*Friesian cow associated with *Br. abortus**

infection

supratarsal skin shows evidence of previous injury.

(Czechoslovakia).

refuse to use cubicles and remain out on the slurry-

cellulitis in that the swelling resolves in cattle when at

covered concrete. They often have problems rising from

*pasture. Some cases have been associated with *Br.**

the slippery surface, during which the wet skin of the

abortus infection (Fig. 32.17; see also p. 580).

lateral part of the hock is repeatedly abraded. Other cows suffer injury when they rise awkwardly in poorly designed or badly bedded cubicles. Standings that are

Clinical signs and diagnosis

up to 30 cm (12 inches) too short for the particular

*A soft and painless swelling develops on the dorsal
breed and neck rails placed 5 cm (2 inches) too low may
aspect of the carpus, usually with evidence of skin con-
force cows to stand with the hind feet in the passage-
tusion. It tends to be symmetrical and no breed or age
way. Careful observation (e.g. during oestrous detec-
predilection is recognized. Minor swellings are merely
tion) will usually disclose the specific problem.*

a cosmetic blemish. The exceptional large grapefruit or

Treatment should be conservative and medical. It

*melon-sized mass may cause slight mechanical lame-
should never involve a long incision to drain fluid from
ness but is painless. The fluid appears to be synovia-like,
the false bursa, neither should one attempt radically to
yellow and clear, but less tenacious. It is produced by*

dissect out this bursa. Most cases are best left untreated the bursal lining. The cavity tends to be multilocular. in the absence of lameness. If lameness eventually Differential diagnosis from a precarpal abscess or septic occurs as a result of a low-grade tarsal infection, synovitis, both of which are painful and cause lameness, systemic antibiotics (oxytetracyclines) rapidly reduce the is simple.

size of the swelling. Corticosteroids are usually ineffective. Infected cases may be bandaged daily and be irrigated with warm water, but the response tends to be

Treatment and prevention

incomplete. A cluster of cases indicates a need to check the loafing areas and cubicles (Hughes, 2000).

Surgical drainage is rarely necessary. Such drainage (as in tarsal bursitis, p. 460) is fraught with the risk of introducing infection into a sterile site. The skin is more

Carpal bursitis

liable to contamination every time the animal lies down. It is hard to protect the area with a bandage.

Aetiology and pathology

Most cases respond if placed in a well-bedded straw yard (winter) or put to pasture (summer), i.e. into subcutaneous precarpal bursa and adjacent soft tissues. Roundings where predisposing trauma can be avoided.

Very rarely does infection extend into the joint.

Drainage is needed only when gait, feed intake and Repeated contusion from contact points of the stanchions, stalls, or floor in housed cattle is the main predisposing factor. The drainage should be done as an aseptic procedure. A mixture of antibiotic and corticosteroid solution should be instilled into various

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sections of the collapsed cavity and such injection feature of mature and older bulls. In one extensive should be repeated at weekly intervals.

series of bulls followed to slaughter, severe signs of tail

Radical surgical removal of the bursa and excessive and hindlimb paralysis were only seen in two cases, skin is possible, but is a haemorrhagic and time-

*though all bulls had varying degrees of ankylosing
consuming process despite the presence of a tourniquet
spondylitis.*

*proximal to the carpus. General anaesthesia is usually
Differential diagnosis includes other causes of spinal
required as adequate local infiltration of the bursal wall
cord compression such as aberrant migrating larvae and
is impractical.*

*infiltrating neoplasms such as lymphosarcoma as well as
Destruction of the bursal lining with an astringent
hindlimb degenerative joint disease.*

*(copper sulphate) or irritant disinfectant (tincture of
iodine) is not advisable, as a severe local reaction is pro-
duced, and dissection, curettage and repeated irrigation*

Treatment and prevention

of the wound are essential steps before secondary

*The prognosis depends on the degree and extent of
healing eventually occurs.*

nerve damage. In the progressive ataxic or paraplegic

*Prevention involves careful attention to the housing
case, it is poor. Mild cases may improve following rest*

system for the existing herd. Problem cows should be and use of corticosteroids and diuretics to reduce the put into straw yards before the swellings cause a clinical oedema. Animals should be bedded on deep straw to cal problem.

prevent the development of decubital lesions. Many mild cases relapse following partial recovery. Slaughter on humane grounds is often the action of choice.

Ankylosing spondylitis

Aetiology and pathology

Mineral imbalance-related lameness:

Generally confined to old bulls and defined as an **rickets, osteomalacia** (see also p. 253)

acquired fusion of the ventral aspects of the bodies of the caudal thoracic and lumbar vertebrae, sufficient

Aetiology and pathology

pressure may be exerted on the ventral and lateral

Mineral, indeed nutrient, imbalance can result in lameness from calfhood through the growing yearling stage some hindlimb weakness, ataxia or paralysis. Exostoses

to maturity and old age. The pattern almost invariably first develop on the ligaments of the caudal 2–3 thoracic involves an imbalance or deficiency of two or more and cranial 2–3 lumbar spinal bodies (T11–L3). Degenerative factors.

erative changes in the intervertebral discs may predis- During growth the important elements in normal pose to exostosis formation. The new bone may extend development of healthy bone collagen, which is the into the spinal canal as ‘replacement bone’. Sometimes, framework for provisional calcification of the zone of possibly resulting from a fall or a violent ejaculation, hypertrophied cartilage, are vitamin D, copper, protein part of the exostosis may fracture, the fracture line and energy. Mineralization of the cartilaginous matrix extending through the vertebral body. Fluid exudate results in deposition of much calcium, phosphorus and (blood, oedema) from the fracture may cause pressure carbonate, with lesser amounts of sodium, magnesium on the spinal cord.

and fluoride. Vitamin D, calcium and phosphorus are

essential to the formation of this bone mineral.

In the prenatal period, identified deficiency diseases

Clinical signs and diagnosis

resulting in abortion or the birth of progeny with

An early sign may be a reluctance or difficulty in mount-

skeletal defects include lupinosis (congenital arthro-

ing cows, attributable to the mechanical effects of a rela-

gryposis), manganese (contracted tendons), and zinc

tively inflexible thoracolumbar spine. Usually there are

and vitamin A deficiencies (multiple defects).

no premonitory signs and the bull suddenly has difficulty

Vitamin D deficiency in calves leads by a complex

in rising and may adopt a 'dog-sitting' position or may be

pathway involving the parathyroid gland, kidney and

completely paraplegic, dragging the digits caudally.

intestine to deficient calcification of long bones and of

Sometimes marked ataxia suddenly develops.

the cartilaginous matrix. The result in calves is rickets.

The site of trauma can rarely be accurately defined

The zone of provisional calcification is widened but

due to problems of size. Crepitus is rarely elicited.

the new osteoid is not mineralized. In adults, the same Swelling of the back musculature is not seen. On rectal deficiency causes osteomalacia, which is rare except in examination, exostoses may be palpable dorsal to the northern Australia and South Africa. Usually a calcium: aortic bifurcation, but such exostoses are a normal phosphorus imbalance is also involved. Vitamin



Lameness Above the Foot • 463

D-deficient diets may be identified on some farms where feed is home-mixed.

Clinical signs and diagnosis of rickets

Calves have enlarged metaphyses and growth plates of the long bones, especially the distal metatarsus and metacarpus, fetlock and pastern. The swellings are rather painful to pressure. The costochondral junction exhibits the classical 'rickety rosary' in calves. Radio-

graphic changes include a widened area of radiolucency at the growth plate with flaring of the distal metaphyses, usually in the carpus, pastern and fetlock.

The diagnosis rests on the soft tissue swelling at

Fig. 32.18

Distal right radial septic physitis in two-year-old characteristic sites, lameness, and in cases of doubt, Holstein heifer. Weight bearing is minimal due to severe pain feed analysis. Serum or plasma may reveal depressed and soft tissue swelling. The aetiology, in the absence of levels of calcium, phosphorus and an elevated alkaline penetration, was probably haematogenous.

phosphatase.

Differential diagnosis includes physeal dysplasia and joint ill (polyarthritis).

Clinical signs and diagnosis of osteomalacia

A haematogenous form is seen in calves six to twelve months old as a result of Salmonella infection, which The relative or absolute phosphorus deficiency, usually localizes in the metaphysis, physis and epiphysis of long acquired from low phosphorus pastures, is classically

bones such as the metacarpus as well as the upper limb associated with the development of pica and osteophagia where an embolic process may be responsible (Kersjes et al., 1966; Gitter et al., 1978). Blood flow through the fractures of long bones, weight loss, stiffness and recum-metaphyseal sinusoids is slow, facilitating easy bacterial

bency. Deficiency tends to be exaggerated in cows due to the demands of pregnancy and lactation. Fertility multifocal and cause rapid destruction of bone. Other may be severely reduced. Hypophosphataemia is

organisms in calves include A. pyogenes and E. coli. In accompanied by normal blood levels of calcium. The older, growing cattle, less than two years old, a growth condition must be distinguished from DJD. plate osteomyelitis may involve the distal metatarsus or Vitamin D-deficient osteomalacia in grazing animals

distal tibia. Common bacterial isolates are A. pyogenes is seen in temperate climates with a shortage of sun-

and Salmonella spp. (De Kessel et al., 1982). Periartic-cured hay and affects cattle stressed by the demands

ular soft tissue swelling is prominent (Fig. 32.18). The

ular soft tissue swelling is prominent (Fig. 32.18). The

ular soft tissue swelling is prominent (Fig. 32.18). The

ular soft tissue swelling is prominent (Fig. 32.18). The

of pregnancy and lactation. The clinical manifestation radiographic extent of bone destruction is very variable resembles the phosphorus-deficient form.

and appears to be unrelated to the involvement of epiphysis and metaphysis, which are equally likely to be affected. Many cases have infected sequestra in

Treatment and prevention

the osteomyelitic focus.

In rickets, increased vitamin D in the ration along with

In adult cattle, osteomyelitis of long bones is a sporadic isolated phenomenon. The aetiology may again brings a rapid response.

involve haematogenous spread, or may arise following

Osteomalacia in adult cattle is treated by dietary

direct trauma. Open long bone fractures are an obvious supplementation with rations high in phosphorus.

example (see p. 441). Less obviously, saucer fractures of the diaphysis may lead to formation of a sequestrum.

Sequestra in long bones of cattle are related to common

Osteomyelitis

sites of lacerating wounds, notably the proximal half of the dorsal aspect of the metatarsus (Firth, 1987). This author hypothesizes that severe contusion alone may

Aetiology and pathogenesis

produce sufficient subcutaneous soft tissue damage to

Osteomyelitis of the limb bones in cattle occurs in two render the area susceptible to infection and subsequent forms: one in growing cattle, the other in adults.

adjacent bone sequestration.

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A chronic sequestrum in a long bone may develop a sation, septic myositis or extensive intramuscular fistulous tract, which permits easier delineation of the abscessation. Open wounds should, therefore, be bone by insertion of a positive contrast agent for a explored with a sterile probe.

fistulogram.

Osteomyelitis of long bones without evidence of skin

A specific form of osteomyelitis is Brodie's abscess, trauma must be differentiated from fissure fractures, which is a circumscribed abscess lined with a granular

subperiosteal haematoma, subcutaneous abscesses and membrane surrounded by sclerotic bone. Synonyms are rare bone tumours. In calves, haematogenous bacterial chronic fibrous osteomyelitis and chronic bone abscess. infection of metaphysis, growth plate and epiphysis Though the lining is histologically a definite pyogenic must be distinguished from joint ill, epiphyseal separation membrane, bacteriological culture may be sterile. Both tation and metaphyseal fractures.

F. necrophorum and A. pyogenes have been involved in cases of Brodie's abscess in three and six-months-old calves (Weaver, 1972).

Treatment and prognosis

Cattle with systemic involvement (pyrexia, anorexia) should be given parenteral antibiotics for three to five

Clinical signs and diagnosis

days. Many cases respond to this therapy. Non-responsive cases should be considered for surgery. This Osteomyelitis usually causes severe, continuing pain applies particularly to cases of Brodie's abscess and with local swelling. These signs relate to the active

incision should be made over the metaphysis to attempt process such as long bone infection following an open aspiration of the contents of the abscess if subperiosteal fracture when, if left untreated, purulent material, in position. In other cases a sinus tract through the sometimes with bone spicules, is discharged to the cortex may be enlarged to evacuate the contents and to exterior. Cellulitis may be severe and additional permit curettage of the adjacent necrotic bone and sinuses may form.

pyogenic membrane.

Osteomyelitis in calves where infection is within and Cattle with open long bone fractures and surrounding the epiphysis presents differently. Localized osteomyelitis should have sequestra removed and zation of the lameness may be difficult due to the check radiographic films taken. Treatment then absence of local swelling but pain is usually evident. depends on the type of fracture and extent of Chronic osteomyelitis may not cause lameness when osteomyelitis. The options are discussed elsewhere

the infected focus is effectively walled off by dense (p. 442).

sclerosed bone (involucrum). Sometimes, in such cases, Calves with bacterial infection of the metaphysis lameness suddenly develops as infection flares up once sometimes respond to parenteral antibiotics alone, more.

otherwise local debridement and irrigation of the focus Osteomyelitis rarely extends to involve joints except is justified.

in the digit and any resentment to joint flexion is usually due to extension from the diaphyseal or metaphyseal focus.

Physeal dysplasia

Systemic effects of osteomyelitis include lassitude, mild fever and a reduced appetite. Radiology is indi-

Aetiology and pathology

cated in most cases to confirm the diagnosis, to determine the extent of the pathological process, and to

Physeal dysplasia, also loosely termed epiphysitis assess the possibility of useful surgical intervention.

(though the epiphysis is not involved in an inflammatory process), is primarily a defective development of and its replacement by infected granulation tissue.

one or more growth plates, which later may become Periosteal new bone is often evident and is variable in necrotic and inflammatory and later still may be purulent amount. Initially, the periosteum is elevated over an lent. The problem usually affects the lower limb, especially osteomyelitic focus with an underlying loss of cortical density. Later, in animals where a sequestrum forms, apparently involves a reduced blood supply to the new subperiosteal bone is deposited while sclerosis is growth plate as a result of uneven or excessive mechanical pressure evident around the sequestrum.

ical pressure. The metaphyseal side, dependent on an Differential diagnosis of osteomyelitis with an open adequate blood supply for calcification and ossification, wound involves determining the primary lesion. Some is more affected than the epiphyseal surface. The patho-

cases of wounds may not involve the periosteum and logical process has not been thoroughly investigated in lameness may reflect pain due to subcutaneous abscesses in cattle, but necrosis of the ground substance, resulting



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from folding of the cell columns and loss of continuity, has been noted histologically in housed fattening cattle. Poor-quality, uncomfortable floor surfaces predispose rapidly fattening cattle with limited exercise space to physeal dysplasia.

Gross pathology reveals some subcutaneous oedema and red or red–grey discoloration around the growth plate, which is either necrotic or, in severe cases, puru-

lent. In its most severe form the growth plate undergoes separation.

Copper deficiency or a combined copper–molybdenum deficiency (see pp. 254, 298) has also been associated with physeal dysplasia as a result of defective collagen formation. The abnormal collagen does not permit normal mineralization. The result is stiffness and lameness in calves with a characteristic distal metatarsal swelling. Another result is an increased incidence of long bone fractures.

Clinical signs and diagnosis

Fig. 32.19

Massive septic myositis in right hindquarters of two-year-old Saler bull. The light area (shaved) shows the site of exploratory needle puncture. Abscess cavity contained 12 litres of pus. *Actinomyces pyogenes* was recovered in pure culture. Severe oedema extends down to the hock. Recovery followed drainage and irrigation.

several animals in a group of cattle are affected simultaneously following violent exercise, for example at lameness should be salvaged before there is further turning out to pasture from confined housing. Isolated weight loss.

cases may result from trauma on slats.

In the copper-related form the limbs may be bowed either laterally or medially, the gait is stilted, and the

Lameness of iatrogenic origin

swellings tend to be symmetrical. However, other signs of copper deficiency are present, including unthriftiness, Some forms of upper limb lameness may be the result a rough discoloured hair coat and diarrhoea.

of treatment for disease or injury. Various entities

Radiographic features include rarefying lesions with have been discussed elsewhere: radial paralysis from an increased width of the growth plate with irregular-prolonged lateral recumbency during anaesthesia (p. ity and fragmentation, but with an absence of reactive 436), neonatal long bone fractures and physeal separation change around the metaphyseal margin. The radi-

tions resulting from excessive traction in dystokia
ographic changes are more severe in cattle with physeal
(p. 444), and traumatic injury and transection of
separation and secondary repair. Milder radiographic
the gastrocnemius tendon (p. 451). Another form of
changes are evident in many cattle of the same group
iatrogenic damage is that following intramuscular injection
that do not show lameness.

tions. Occasionally, particularly in calves, the sciatic
The list of differential diagnoses includes rickets,
nerve may be injured. More frequent is the development
traumatic separation of the growth plate, bacterial
ment of an abscess following a subcutaneous injection,
ostitis, septic arthritis and fractures. Radiography is
which rarely causes lameness but remains a cosmetic
necessary to permit this differentiation.

blemish. When a non-sterile preparation, or a dirty
syringe and needle are used for an intramuscular
injection, the results may be more serious. As a rule,
Treatment and prevention
infection is sealed off by fibrous tissue with no ill-

Only the copper-related syndrome is responsive to effects. Sometimes the result is a massive abscess, treatment by an appropriate diet change. Mild non- which causes lameness and requires prompt surgical copper-related cases respond to rest and external drainage and daily irrigation to prevent recurrence support by lightweight casting. Most cases of such (Fig. 32.19).

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References

Veterinary Surgery, 3rd edn, pp. 261–2. Blackwell Science, Oxford.

Hughes, J. (2000) Cows and cubicles. In Practice, 22, 231–9.

Anderson, D.E., St.-Jean, G., Morin, D.E., Ducharme, N.G.,

Kendrick, J.W. & Sittmann, K. (1966) Inherited osteoarthritis Nelson, D.R. & Desrochers, A. (1996) Traumatic flexor

of dairy cattle. Journal of the American Veterinary Medical tendon injuries in 27 cattle. Veterinary Surgery, 25, 320–6.

Association, 149, 17–21.

Boyd, J.S. & Weaver, A.D. (1967) Spastic paresis in cattle.

Kersjes, A.W., Frik, Y.F. & Van de Watering, C.G. (1966)

Veterinary Record, 80, 529–30.

Bacteriele osteitis bij rundereen. Tijd. Diergeneesk. , 91, Bracewell, C.D. &

Corbell, J. (1979) *An association between 1537–47.*

arthritis and persistent serological reactions to Brucella Kofler, J. & Martinek, B. (2000) *Ultrasonographic imaging*

abortus in cattle from apparently brucellosis-free herds.

of disorders of the carpal region in 34 cattle – arthritis,

Veterinary Record, **106**, 99–101.

tenosynovitis, carpal hygroma, periarticular abscess. Pro-

Buchta, F., Rosenfalk, A. & Behse, F. (1984) Sensory poten-ceedings of the International Symposium on Disorders of

tial of normal and diseased nerves. In Peripheral Neuropa-

the Ruminant Digit, Parma, Italy, September 2000, pp.

thy (ed. by P.J. Dyck), p. 995. W.B. Saunders, Philadelphia.

246–9.

Chamberlain, A.T. (1986) Prognostic indicators in the

Madison, J.B., Tulleners, E.P., Ducharme, N.G. et al. (1989) downer cow. In Proceedings of the British Cattle Veterinary Idiopathic gonitis in heifers: 34 cases (1976–1986). Journal Association, pp. 57–68.

of the American Veterinary Medical Association, **194**, 273–7.

Chamberlain, A.T. (1987) The management and prevention of

Nelson, D.R., Huhn, J.C. & Kneller, S.K. (1990) Peripheral the downer cow syndrome. In Proceedings of the British

detachment of the medial meniscus with injury to the

Cattle Veterinary Association, 20–30.

medial collateral ligament in 50 cattle. Veterinary Record, Cox, V.S. (1981) Understanding the downer cow syndrome.

127, 59–66.

Compendium of Continuing Education for the Practising

Pavaux, C., Sautet, J. & Lignereux, J.Y. (1985) Anatomie du Veterinarian, 3, 45–53.

muscle gastrocnemien des bovins appliquée à la cure

Cox, V.S. & Onapito, J.S. (1998) The many causes of downer chirurgicale de la paresie spastique. Vlaams Diergeneesk.

cows. AABP Bovine Proceedings, 31, 164–6.

Tijdschr. , 54, 296–312.

Cox, V.S. & Farmsworth, R.J. (1998) Prevention and treatment Roeber, D.L., Smith, G.C., Floyd, J.G. & Cowman, G.L. (2000) of down cows: a continuum. AABP Bovine Proceedings, 31, Lameness in breeding cattle in the United States: the

167–9.

National Market Cow and Bull Beef Quality Audit 1999.

De Kessel, A., Verschooten, F., De Moor, A., Steenhaut, M. & Proceedings of the 11th International Symposium on

Wouters, L. (1982) Bacteriele Osteitis–Osteomyelitis ter

Disorders of the Ruminant Digit, Parma, Italy, September Hoogte van Groeiplaten bij het Rund. Vlaams Diergeneesk.

2000, pp. 152–6.

Tijdschr. , 51, 397–422.

Schub, J.C.L. & Killeen, J.R. (1988) A retrospective study De Ley, G. & De

Moor, A. (1975) Bovine spastic paresis:

of dystokia-related vertebral fractures in neonatal calves.

cerebrospinal fluid concentrations of homovanillic acid

Canadian Veterinary Journal, **29**, 830–3.

and 5-hydroxyindolacetic acid in normal and spastic

Smith, B.P., Angelos, J., George, L.W., Fecteau, G., Angelos, S., calves.

American Journal of Veterinary Research, **36**, VanMetre, D., House, J.K. &

Hullinger, P. (1997) Down

227–8.

cows: causes and treatments. Proceedings of the American

De Ley, G. & De Moor, A. (1977) Bovine spastic paresis:

Association of Bovine Practitioners, **30**, 43–5.

results of selective desafferation of the gastrocnemius

Steiner, A. & Zulauf, M. (2000) Fenestration of the abaxial muscle by means of
spinal dorsal root resection. *American*

hoof wall and implantation of gentamicin-impregnated

Journal of Veterinary Research, **38**, 1899–1900.

collagen sponges for treatment of septic arthritis of the

Ducharme, N.G., Stanton, M.E. & Ducharme, G.R. (1985)

distal interphalangeal joint in 7 cattle. Proceedings of

Stifle lameness in cattle at two veterinary teaching hospitals the 11th
International Symposium on Disorders of the

(42 cases). Canadian Veterinary Journal, **26**, 212–17.

Ruminant Digit, Parma, Italy, September 2000, pp. 271–2.

Ferguson, J.G. (1982) Management and repair of bovine

Trostle, S.S., Nicoll, R.G., Forrest, L.J. & Markel, M.D. (1997) fractures. Compendium of Continuing Education for the

Clinical and radiographic findings, treatment, and outcome

Practicing Veterinarian, 4, S128–S136.

in cattle with osteochondrosis: 29 cases (1986–1996). Journal Ferguson, J.G. (1985) Special considerations in bovine

of the American Veterinary Medical Association, 211, orthopedics and lameness. Veterinary Clinics of North

1566–70.

America. Food Animal Practice, 1, 131–52.

Tulleners, E.P. (1986) Metacarpal and metatarsal fractures in

Ferguson, J.G. (1997) Surgical conditions of the proximal limb.

dairy cattle: 33 cases (1979–1985). Journal of the American In Lameness in Cattle, 3rd edn (ed. by P.R. Greenough & Veterinary Medical Association, 189, 463–8.

A.D. Weaver), p. 262. W.B. Saunders, Philadelphia.

Tulleners, E.P., Nunamaker, D.M. & Richardson, D.W. (1987) Firth, E.C. (1987) Bone sequestration in horses and cattle.

Coxofemoral luxations in cattle: 22 cases (1980–1985).

Australian Veterinary Journal, 64, 65–9.

Journal of the American Veterinary Medical Association, 191, Gitter, M., Gray, C., Richardson, C. & Pepper, R.T. (1978) 569–74.

Chronic salmonella infection in calves. British Veterinary Journal,
L.C.

(1964) *Peripheral nerve injuries:*

an

Journal, **134**, 113–21.

experimental study in cattle. Veterinary Record, **76**, 1293–

Hickman, J., Houlton, J. & Edwards, B. (1997) *Atlas of* 1301.

Lameness Above the Foot • 467

Weaver, A.D. (1972) *Chronic localised osteomyelitis of the*

Wells, G.A.H., Hawkins, S.A.C., O'Toole, D.T., Done, S.H.,

bovine limb. British Veterinary Journal, **128**, 470–6.

Duffell, S.J., Bradley, R. & Herbert, C.N. (1987) *Spastic syn-*Weaver, A.D.

(1977) *An investigation into condemnations*

drome in a Holstein bull: a histologic study. Veterinary

for hind limb disease in slaughtered cattle with particular

Pathology, **24**, 345–53.

reference to the stifle and hip joints. Veterinary Record, Wintzer, H.J. (1961) *A possible method of treating long*

100, 172–5.

bone fractures in cattle. Dtsch. Tierarztl. Wschr. , **68**, 226–

Weaver, A.D. (1997) *Joint conditions. In Lameness in Cattle*, 8.

3rd edn, (ed. by P.R. Greenough & A.D. Weaver), p. 163.

Wyn-Jones, G., Baker, J.R. & Johnson, P.M. (1980) *A clinical* W.B. Saunders, Philadelphia.

and immunopathological study of Brucella abortus

Weaver, A.D. & Campbell, J.R. (1972) *Surgical correction of strain 19-induced arthritis in cattle. Veterinary Record, 107, medial and lateral patellar luxation in calves. Veterinary* 5–9.

Record, 90, 567–9.

Adult Cattle

Fertility

Chapter 33

Reproductive Physiology in Cattle

P.J. Hartigan

Introduction

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sidered necessary to do this in a text addressed prima-

The hypothalamus

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rily to clinicians? Essentially, because we believe that it

The anterior pituitary gland

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will enhance the reader's understanding of the patho-

Neuroendocrine links

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genesis of many of the reproductive problems encour-

Feedback

474

tered in practice and, as a consequence, it will foster a

Endocrine signals: generation and reception

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more methodical and perceptive approach to diagnosis

Systemic constraints on reproduction

477

and treatment. Underpinning that belief is the knowl-

Puberty

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Male physiology

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edge that efficient reproductive performance in the

Morphology of the testis

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female is dependent on an integrated and

The spermatozoon

485

precisely timed sequence of hormonal changes that are

Neuroendocrine control

485

regulated by the hypothalamus in response to changes

The epididymis

487

in the external and internal environments. Of course,

Seminal plasma

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this means that the sequence and timing of the hor-

Female physiology

488

monal changes are vulnerable to a great variety of stres-

Oestrous cycle

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sors and noxious agents. Many of these deleterious

Pregnancy

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factors have been recognized – and will be mentioned

Physiology of the postpartum period

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later in the relevant sections – but it is likely that many

Take-home concepts for the clinician

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more remain to be discovered, in particular subtle but

significant stressors associated with modern develop-

ments in cattle husbandry. The individual best placed

Introduction

to identify ‘new causes’ of reproductive inefficiency is

the informed clinician working with problem herds;

Reproductive efficiency in a cattle enterprise is a func-

however, such insights are less likely if the investigator

tion of good management. Therefore, it is important

is not fully alert to the central role of the neuro-

that the help and advice provided to management by

endocrine mechanisms and their sensitivity to the effects

the veterinarian should be based upon a sound appre-

of dietary and metabolic factors, hormone imbalances,

ciation of the physiological mechanisms that control the

stressors, drugs, age and many other influences.

principal reproductive events.

The essential numerical data on reproduction in

cattle are well known: puberty in bulls and heifers at 10–15 months of age; normal oestrous cycles of 18–24

The hypothalamus

days (mean = 21 days); normal oestrus lasts 12–24 hours (mean = 18 hours); ovulation occurs about 24 hours

In the neuroendocrine system the dominant role is after the LH peak at the beginning of oestrus; normal played by the hypothalamus, which acts as a relay gestation lasts 278–293 days; the interval from parturition to first ovulation can be as short as 15 days. The decoded and translated into appropriate signals physiological control of each of these events is exerted to ensure the cooperation of the endocrine system with by a system of interdependent endocrine organs the nervous system in regulatory activities. It is able to that form the hypothalamic–pituitary–gonadal (HPG) do so because it contains many neurones that are axis, an important limb of the neuroendocrine capable of forming and releasing peptide hormones system.

(neurohormones) that regulate the functions of various

The primary purpose of this chapter is to provide a

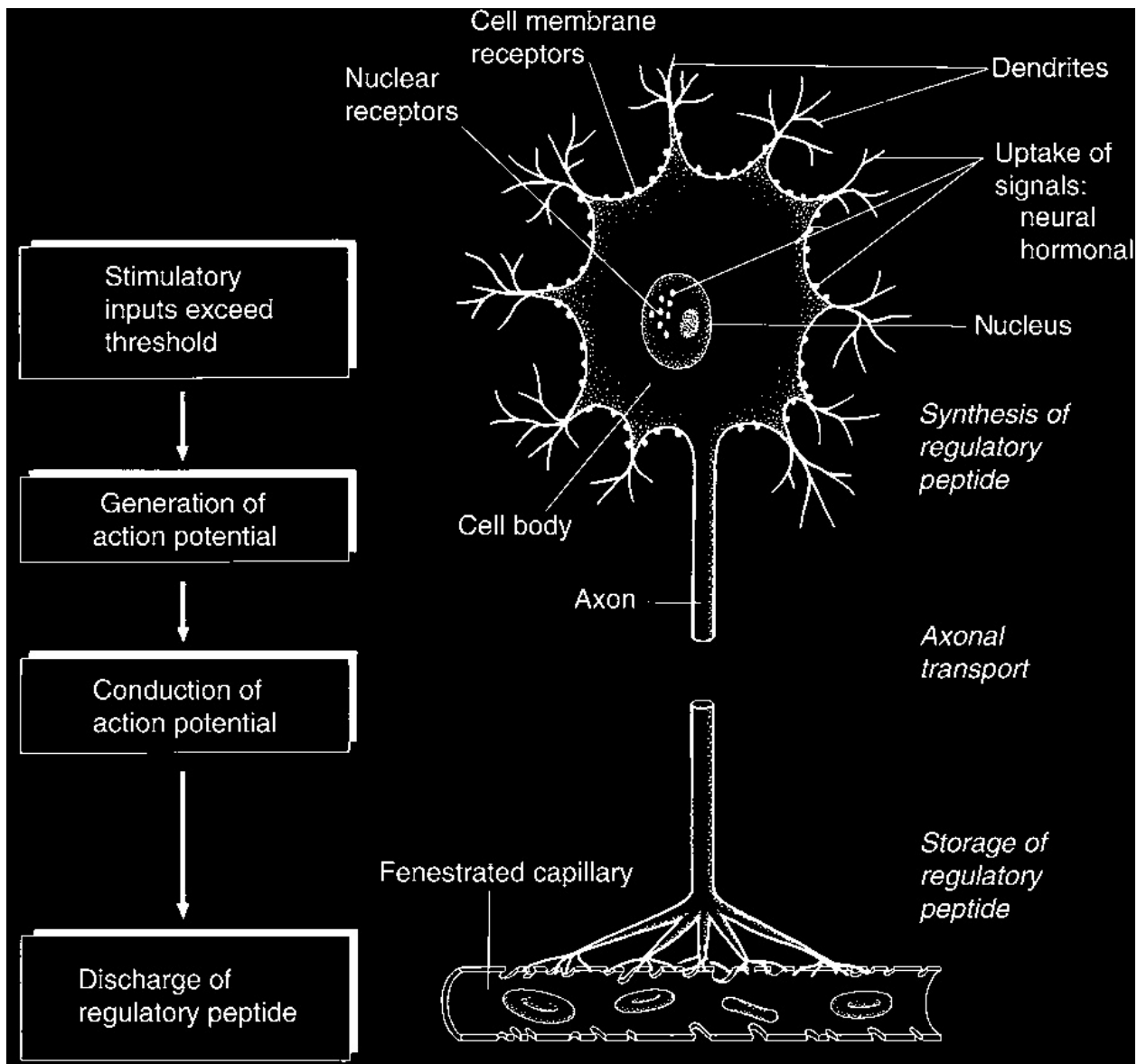
organs, principally via the pituitary gland.

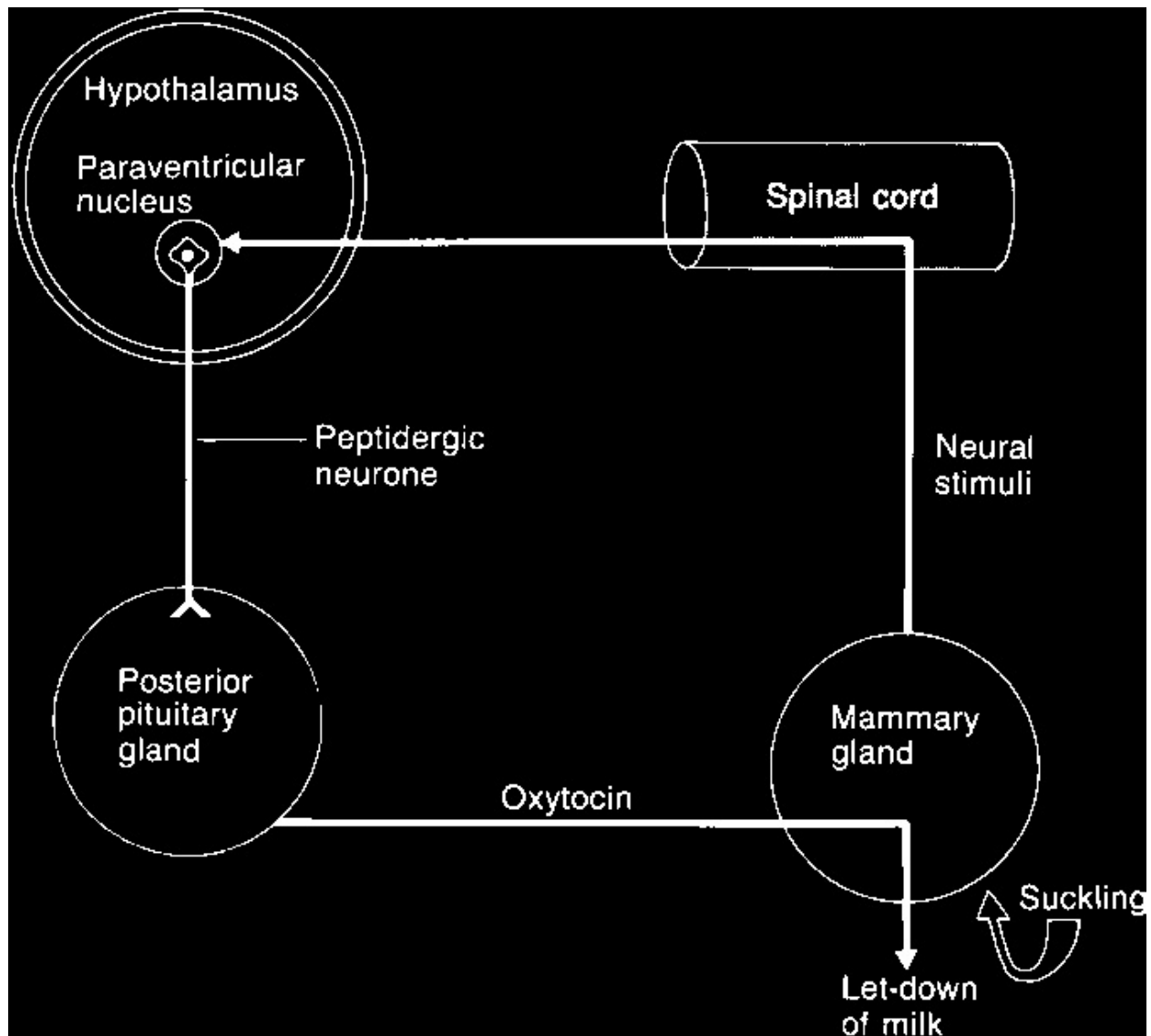
succinct outline of the salient features of the neuroen-

These regulatory peptides are synthesized and pack-

docrine system as it affects reproduction. Why is it con-

aged into granules in the cell body of the neurone





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Fig. 33.1

This diagrammatic representation of a typical peptidergic neurone depicts: (i) the uptake of neural signals by dendrites; (ii) the uptake of endocrine signals by specific

receptors located either in the cell membrane (for water-soluble hormones: peptides, proteins) or in the nucleus (for lipid-soluble hormones: steroids, thyroid hormones); (iii) the endocrine response to signals (synthesis, transport and storage of regulatory peptide); (iv) the neural response to signals (generation and conduction of action potential, which is responsible for the discharge of the regulatory peptide).

before they are transported down the axon to the nerve terminals where they are stored pending release. The peptidergic neurones retain the electrophysiological properties of nervous tissue and they use their electrical activity to release the peptides.

A typical peptidergic neurone (Fig. 33.1) has many hundreds of synapses and through these it receives neural inputs (information) from most parts of the brain. (It also receives information from humoral

inputs; we shall return to this topic later.) The neural and humoral inputs may be stimulatory or inhibitory; at any one moment the response of the peptidergic neurone will reflect its assessment of the current interplay between the synergistic and conflicting stimuli.

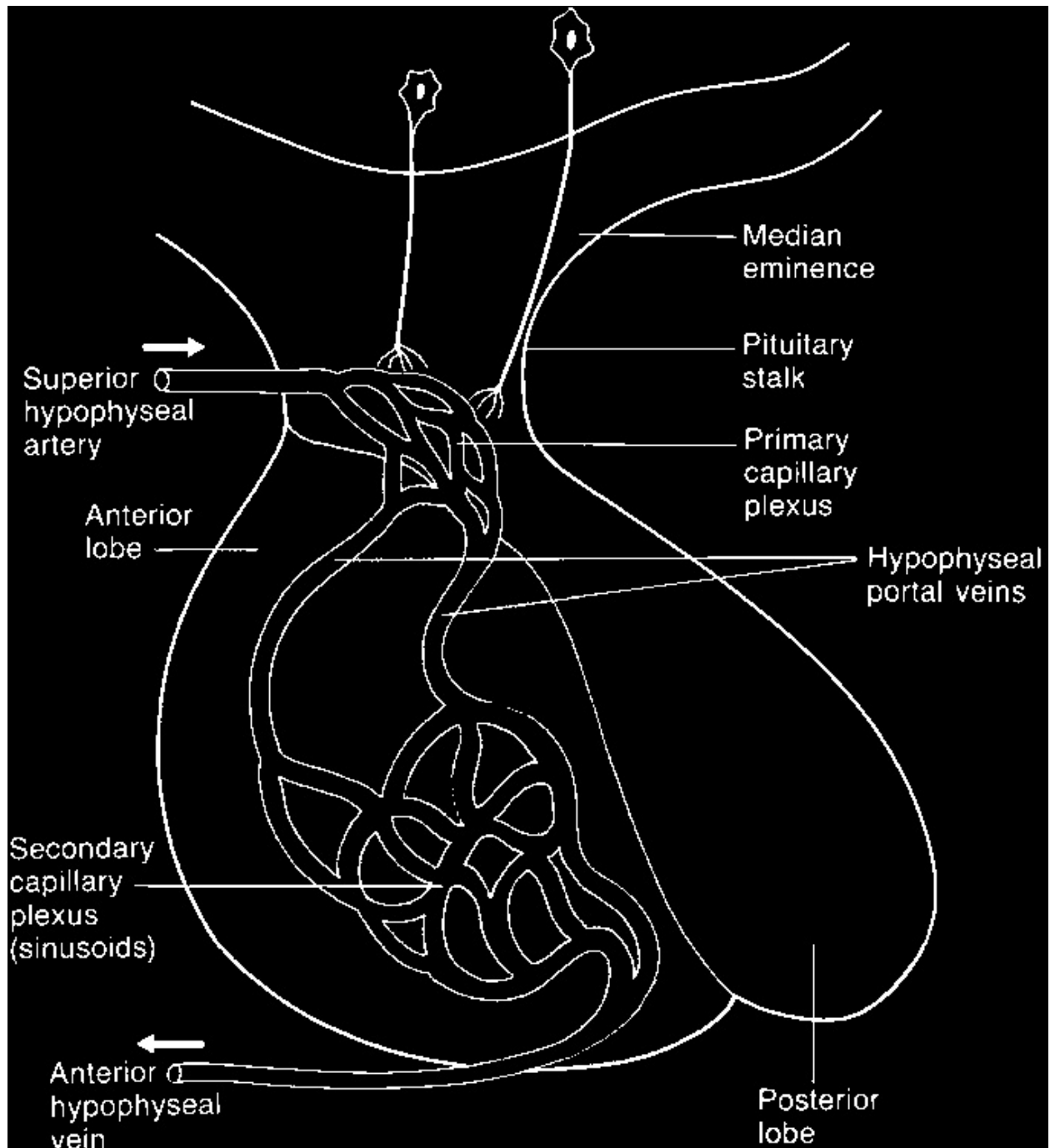
When the stimulatory inputs exceed a critical threshold an action potential is generated, the nerve impulse passes down the axon and it causes the release of the (stored) peptide at the nerve terminals. Usually, the nerve terminals are in close apposition to capillaries with highly fenestrated walls that allow the peptide

Fig. 33.2

Diagrammatic representation of the suckling reflex, molecules to enter the vascular circulation promptly on in which neural stimuli elicit an endocrine response. release.

A simple example to illustrate the physiological phenomena just described is the milk ejection reflex (Fig. 33.2). This reflex depends on a suckling-induced release

and whose axons pass through the pituitary stalk into
of oxytocin. Oxytocin is produced by peptidergic
the posterior pituitary gland. Suckling evokes sensory



impulses that travel via the spinal cord to the brain where they converge on the paraventricular nuclei and generate the impulses in the peptidergic neurones that lead to the release of oxytocin from the nerve endings in the posterior pituitary gland. The reflex is completed when the oxytocin in the blood perfusing the mammary gland induces 'let down' of milk. Conceptually, this is neuroendocrine regulation at its simplest: the neurone that 'reads' the afferent sensory message also produces and releases the effector hormone. This is possible only because the posterior pituitary gland, which arises as a downgrowth of neural tissue from the floor of the third ventricle, retains a direct neural link with the hypothalamus.

The anterior pituitary gland

The anterior pituitary gland (adenohypophysis) arises as an upgrowth from the roof of the primitive mouth and, therefore, it does not have a direct neural link with the hypothalamus. A vascular route has to be used to bring the regulatory peptides from the hypothalamus to the adenohypophysis. A specialized arrangement of

Fig. 33.3

The primary capillary plexus, the hypophyseal the vasculature, the hypothalamo–hypophyseal portal portal veins and the secondary capillary plexus form the system, has evolved for that purpose. In contrast to the hypothalamo–hypophyseal portal system that intervenes normal sequence of arterioles, capillaries and venules, a between the superior hypophyseal artery and the anterior portal system begins and ends with capillaries. In this hypophyseal vein. This is the route taken by GnRH which is syn-instance, the superior hypophyseal artery gives rise to a thesized by neurones whose cell bodies lie within the arcuate plexus of capillaries (the primary plexus) in the region nucleus of the hypothalamus and whose axons abut onto the of the median eminence at the top of the pituitary stalk. external walls of the capillaries in the primary plexus. From this the blood is drained by the hypophyseal portal veins, which pass down the pituitary stalk to end in a secondary plexus (the sinusoidal capillaries) within the anterior pituitary gland (Fig. 33.3). From there the plexus, the hypophyseal portal veins and the secondary

blood returns to the general circulation via the anterior plexus to the secretory cells of the anterior lobe. It hypophyseal vein.

stimulates the release of the gonadotrophins, which

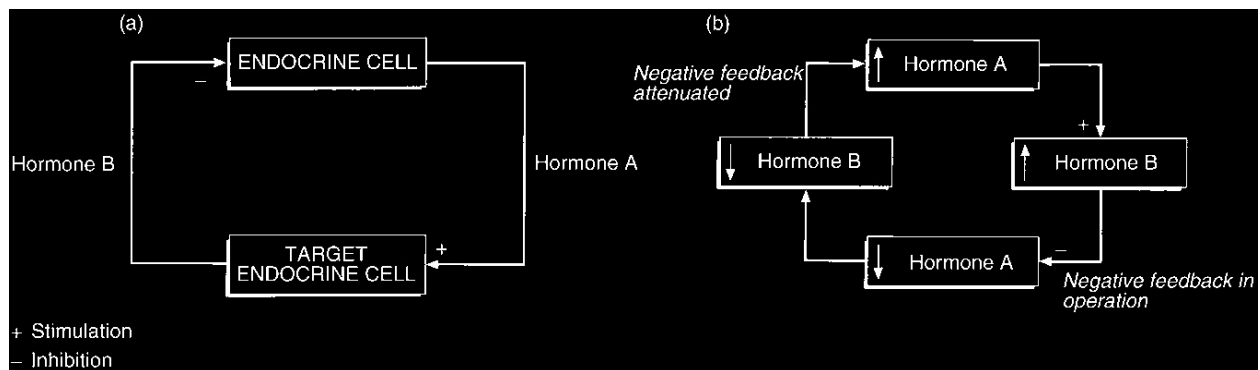
In contrast to the posterior pituitary gland, which enter the general circulation via the draining anterior does not produce the hormones it releases, the anterior hypophyseal vein. Other hypothalamic factors may lobe actually synthesizes the trophic hormones it exert inhibitory actions in the anterior pituitary gland, releases into the circulation, including the gona- e.g. prolactin inhibitory factor (dopamine).

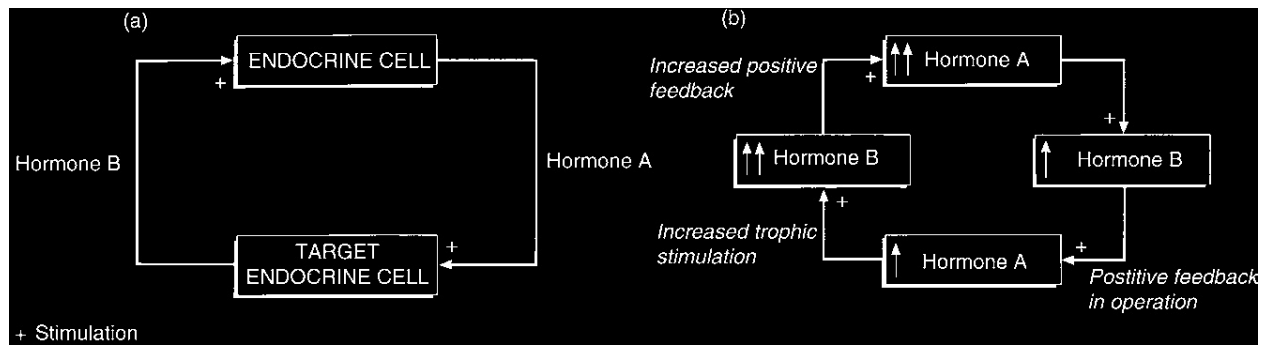
dotrophins: follicle stimulating hormone (FSH) and luteinizing hormone (LH). However, the release of the trophic hormones is governed by stimulatory or inhibitory peptides produced in the hypothalamus. For

Neuroendocrine links

instance, the secretion of FSH and LH is controlled by a gonadotrophin-releasing hormone (GnRH), which is Since the peptidergic neurones form the functional synthesized by peptidergic neurones in the hypothala-

links between the neural and endocrine systems, they must be able to receive, decipher and react to signals via the hypothalamo–hypophyseal portal system. The signals generated by either system in response to changes in the internal and external environments. Neural signals from the median eminence, in close apposition to the fenestrated capillaries of the primary plexus. When a nerve impulse from other regions of the brain converge on the hypothalamus bringing important cues from the special senses (sight, sound, smell), various exteroceptors this hypothalamic factor is transported via the primary plexus (suckling, mating, pain), interoceptors (stimulation of





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Fig. 33.4

Negative feedback. Example: hormone A, FSH; hormone B, oestrogen. FSH stimulates the production of oestrogen by the granulosa cells of the ovarian follicle. The oestrogen exerts an inhibitory effect on the release of FSH by the pituitary gland. When the oestrogen declines the negative feedback is attenuated and secretion of FSH is increased.

Fig. 33.5

Positive feedback. Example: the gonadotrophins FSH and LH (hormone A) stimulate the secretion of oestrogen (hormone B) by the granulosa cells of the preovulatory ovarian follicle (see Fig. 33.24). The rapidly rising concentration of oestrogen (the 'oestrogen surge') stimulates the hypothalamus and the pituitary gland to release a surge of gonadotrophins that is responsible for ovulation.

Dilution abolishes the positive feedback at source.

the cervix and uterus) and stressors (physical, emo-

within stable normal limits while retaining their respon-

tional, overcrowding). The peptidergic neurones also

siveness to the immediate needs of the body. For

respond to humoral signals, especially to hormones

instance, when the concentration of hormone B in

acting through feedback loops. Again, the input may be the circulation rises towards its upper normal limit it stimulatory (positive feedback) or inhibitory (negative feedback). Although there are some important positive feedbacks, e.g. oestrogen prior to ovulation, the major- hormone A is prevented from exceeding its upper normal limit; the concentration of A in the circulation of feedback loops are negative.

declines and, of course, so does the activity of its target cells. The resultant decline in hormone B attenuates the negative feedback inhibition (Fig. 33.4b) and the secretion of hormone A is enhanced before it falls below its

Feedback

lower normal limit.

In a positive feedback loop (Fig. 33.5a) hormone B

In its simplest form, a negative feedback loop (Fig. 33.4a) is a closed system in which hormone A stimulates hormone A. This is an inherently unstable system in the production and release of hormone B which, in

that it is liable to generate ever-increasing quantities turn, has an inhibitory effect on the cells that produce of each of the hormones (Fig. 33.5b); in other words, it hormone A. Such a loop provides a very efficient mechanism lacks the checks and balances of a negative feedback nism by which circulating hormones are maintained system. However, it is ideal for situations that call for

Hypothalamus

Peptidergic
neurone

GnRH

GnRH

Median eminence

Gonadotrope

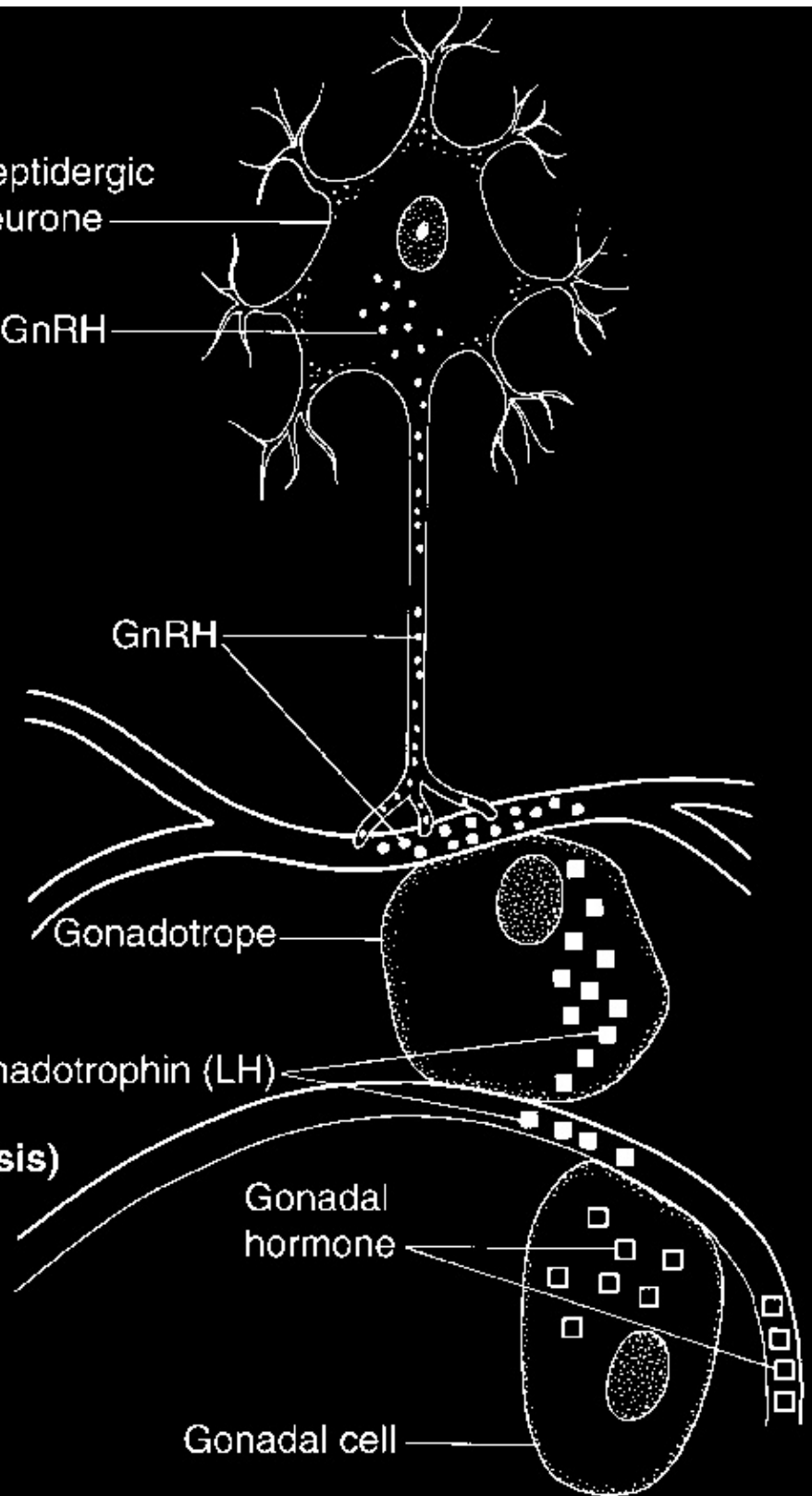
Anterior pituitary gland (adenohypophysis)

Gonadotrophin (LH)

Gonadal
hormone

Gonad

Gonadal cell



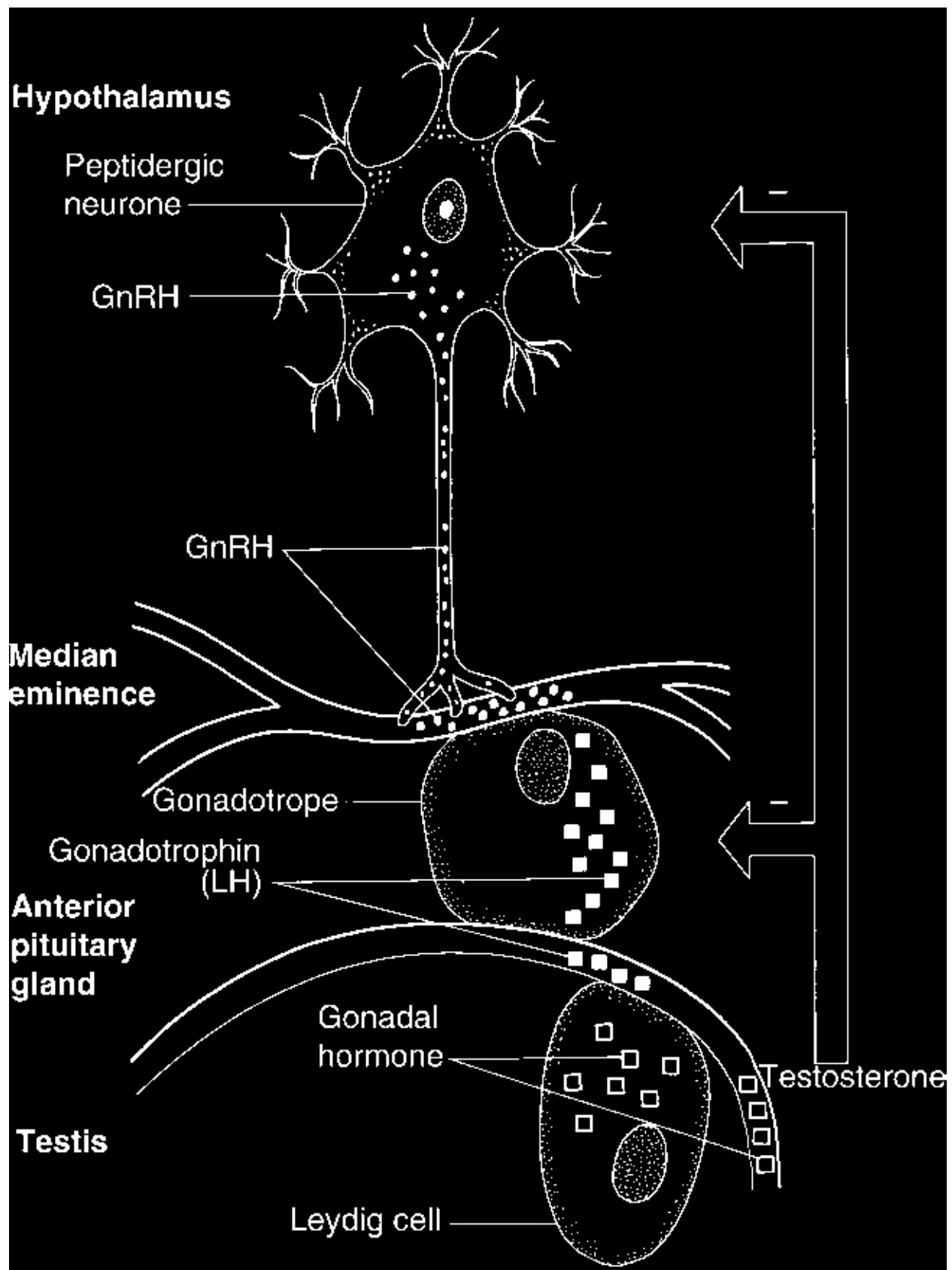


Fig. 33.6

Secretory cells in series: peptidergic neurone

secretes GnRH; gonadotrope secretes LH, FSH; gonadal cell

Fig. 33.7

Long loop negative feedback. LH induces Leydig

secretes testosterone, progesterone or oestrogen.

cells to secrete testosterone; testosterone exerts so-called long loop feedback (i) on adrenohypophysis (suppressing secretion

of LH) and (ii) on hypothalamus (suppressing secretion of GnRH).

short-term but self-limiting responses. The positive

• *Short loop: for example, pituitary gonadotrophins feedback loop may be a purely hormonal phenomenon; acting at the level of the hypothalamus (Fig. for example, the brief but highly significant period of 33.8).*

positive feedback by oestrogen that elicits the preovu-

• *Ultra-short loop: hypothalamic peptides acting at latory surge of LH. Other important positive feedback the level of the hypothalamus (Fig. 33.9).*

mechanisms are initiated by neural stimuli from exteroceptors, e.g. the suckling reflex, or interoceptors, e.g. stretch receptors in the cervix and vagina during the second stage of parturition, that induce reflex release of oxytocin. These two reflexes are excellent examples of feedback), during most of the oestrous cycle it exercises self-limiting responses; removal of the physical stimulus terminates the reflex response almost at once. feedback) shortly before ovulation. Clearly, a single hormone can vary the message it conveys to the reproductive tract are complex and involve several hypothalamus. This raises a crucial question: how do sets of secretory cells in series (Fig. 33.6). For descriptive purposes, three types of complex feedback loops and the hypothalamic peptides encode the signals that

are recognized:

constitute the feedback messages? By definition,

feedback depends on the ability of the target tissues

- *Long loop: for example, gonadal hormones acting*

to detect changes in hormone concentrations in the

at the level of the brain or pituitary gland (Fig.

circulation, and to induce the appropriate re-

33.7).

adjustments (further changes). Hence, the system

Hypothalamus

Peptidergic neurone

GnRH

GnRH

Median eminence

Gonadotrope

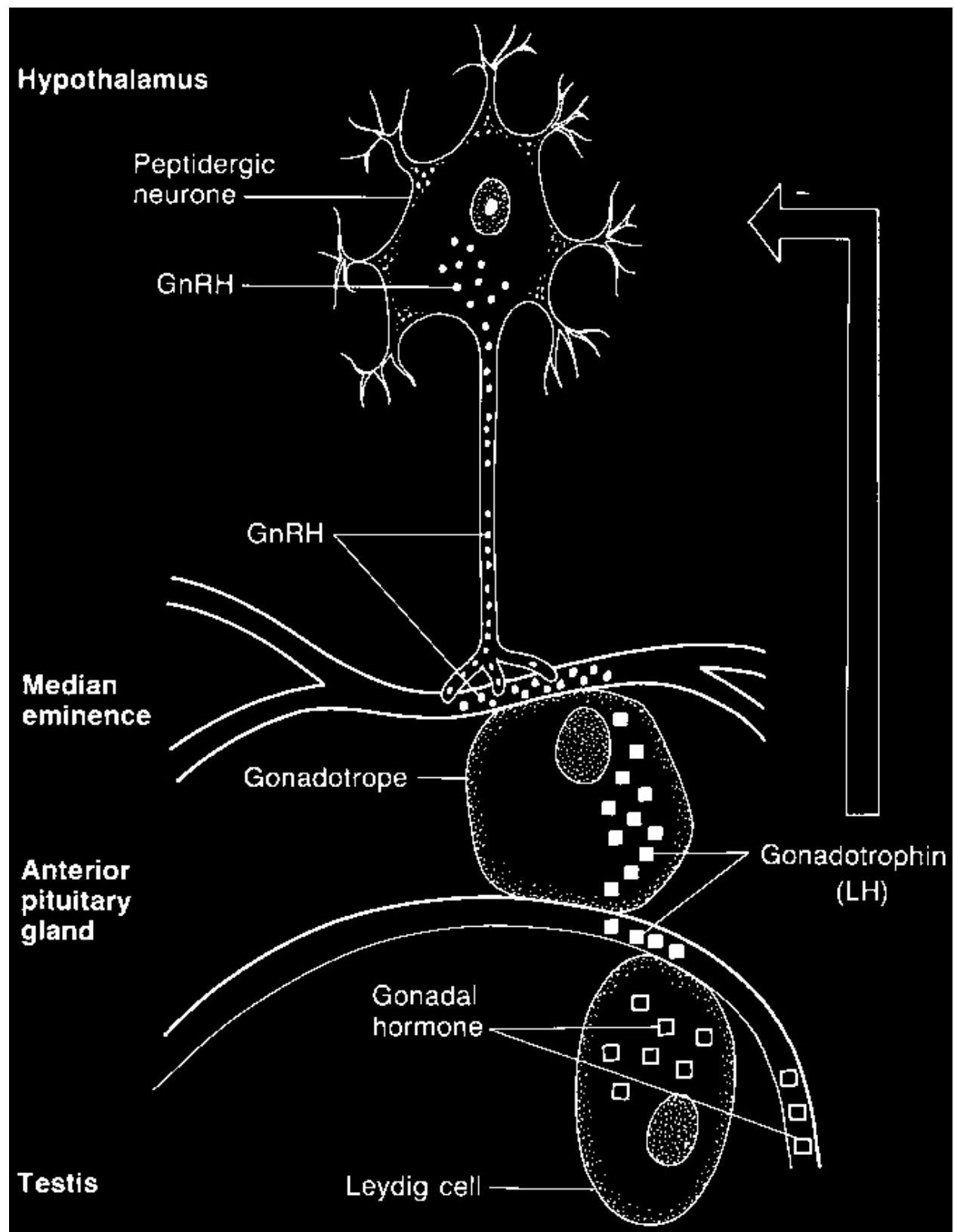
Anterior pituitary gland

Gonadotrophin (LH)

Gonadal hormone

Testis

Leydig cell



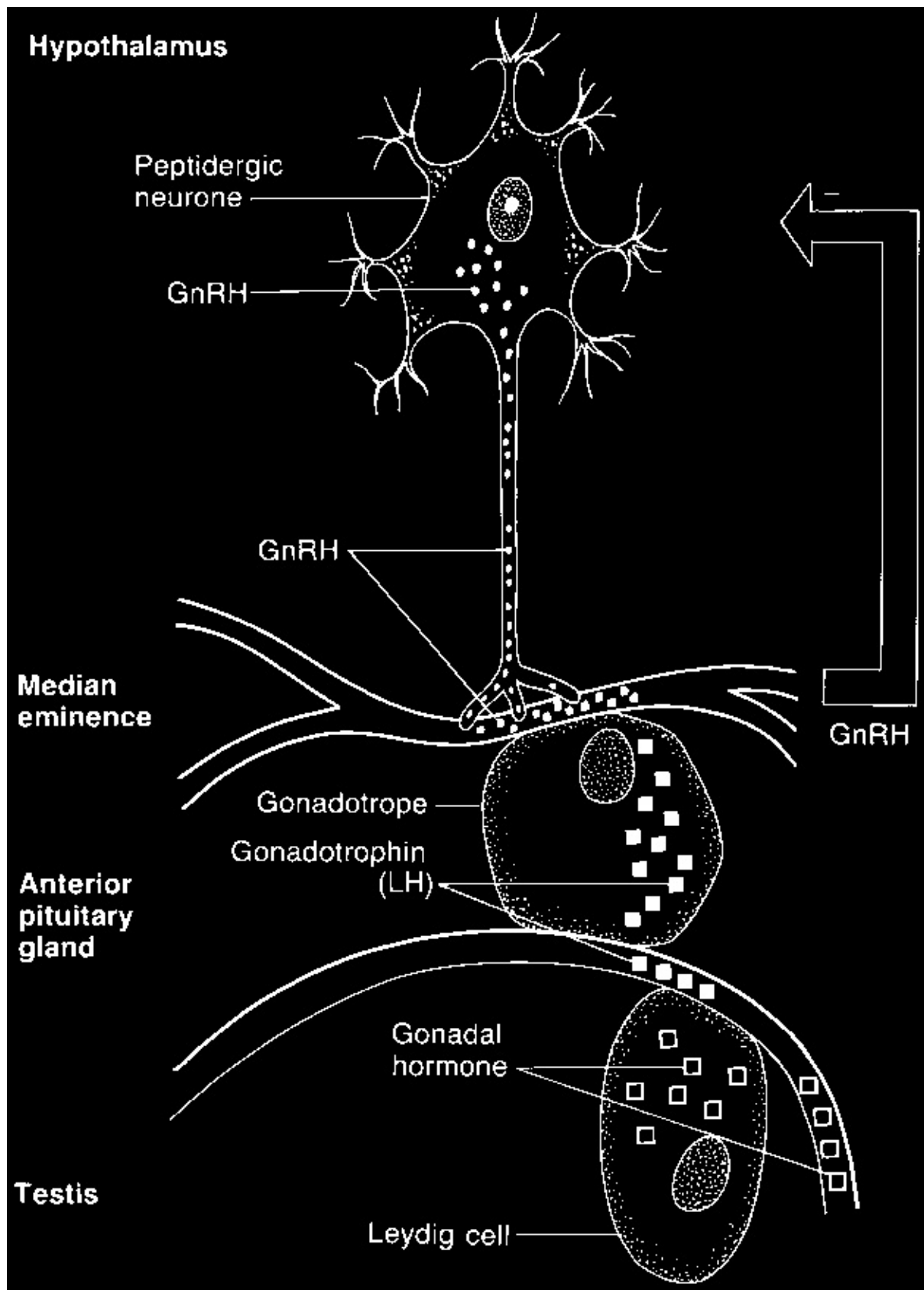


Fig. 33.8

Short loop negative feedback. GnRH induces the

Fig. 33.9

Ultra-short loop negative feedback. The hypothala-

gonadotropes to secrete LH; LH exerts negative feedback on the mic hormone (GnRH) regulates its own secretion.

release of GnRH by the hypothalamus.

depends on fluxes rather than constancy as the means

tors may be located in the cell membrane or within the

of communication.

cell, mainly in the nucleus (see Figs 33.1 and 33.11). The

polypeptide hormones, e.g. GnRH, and the glycopro-

tein hormones, e.g. LH, FSH, bind to receptors in the

Endocrine signals: generation

cell membrane (Fig. 33.11a), whereas the lipid-soluble

and reception

gonadal steroids can diffuse through the cell membrane

to react with intracellular receptors (Fig. 33.11b). The

Two basic types of endocrine signals are used: pulses

location of the specific receptors determines the mecha-

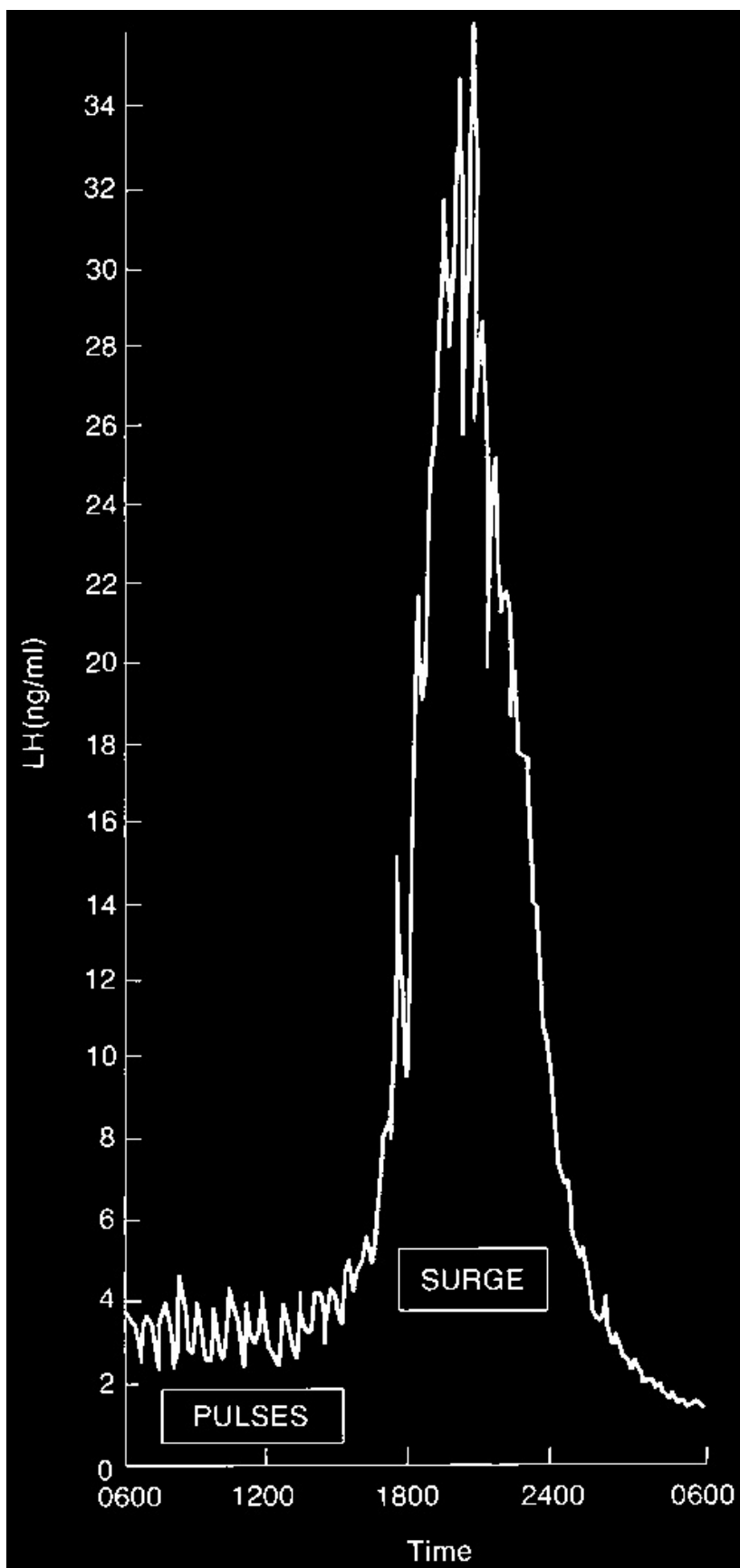
and surges (Fig. 33.10). A pulse of a particular hormone is a discrete burst of secretion of relatively short duration and modest amplitude. A surge occurs when a mechanism determines the speed at which the final sequence of frequent pulses, often of high amplitude, response is elicited.

produces a massive increase extending over a period of

When a gonadal hormone binds to its specific receptor in the nucleus of a target cell, it initiates a series of biochemical reactions that takes several hours to

12–24 hours. Endocrine signals are detected only by cells that have specific receptors to which a particular hormone will bind. When the hormone interacts with the cells that produce the new proteins that elicit the characteristic response to that hormone (Fig. 33.11b). By contrast, when a gonadotrophin binds to its receptor in the cell interacts with another cell type located near the pro-

membrane, it elicits the final response much more
ducer cell we call it paracrine stimulation; when it
rapidly because the resultant cascade of enzymatic
enters the blood stream and interacts with target cells
activity utilizes enzymes that exist already within the
elsewhere we call it endocrine stimulation. The recep-
cell. Binding of the hormone (the 'first messenger') to



(see p. 492). Alternatively, the hormone stimulus may decrease the sensitivity of the target tissue because it increases the degradation and/or decreases the synthesis of receptors; for instance, GnRH can 'down regulate' its own receptors in the adenohypophysis, especially when the gonadotropes are subjected to constant stimulation rather than pulsatile stimulation by the releasing hormone (see p. 497).

The inherent mode of secretion of hormones in the neuroendocrine system is pulsatile and it is controlled by an oscillator or pulse generator in the hypothalamus.

The activity of the pulse generator can be modulated both by neural inputs and by feedback signals from the median eminence, the pituitary gland and the gonads.

In turn, the pulse generator will seek to ensure that the characteristics of the signals emanating from the pituitary gland and the gonads are altered so as to elicit the appropriate responses from target tissues in the reproductive tract. In that way the hypothalamus exercises

fine tuning as well as gross control over reproductive events such as puberty, gametogenesis, oestrus, pregnancy, parturition and lactation.

Before we deal with the neuroendocrine regulation of those processes, it is appropriate to mention three systemic conditions that can constrain the reproductive system: energy balance, stress and neural–immune interactions.

Systemic constraints on reproduction

Energy balance

Although reproduction is critical for the survival of the

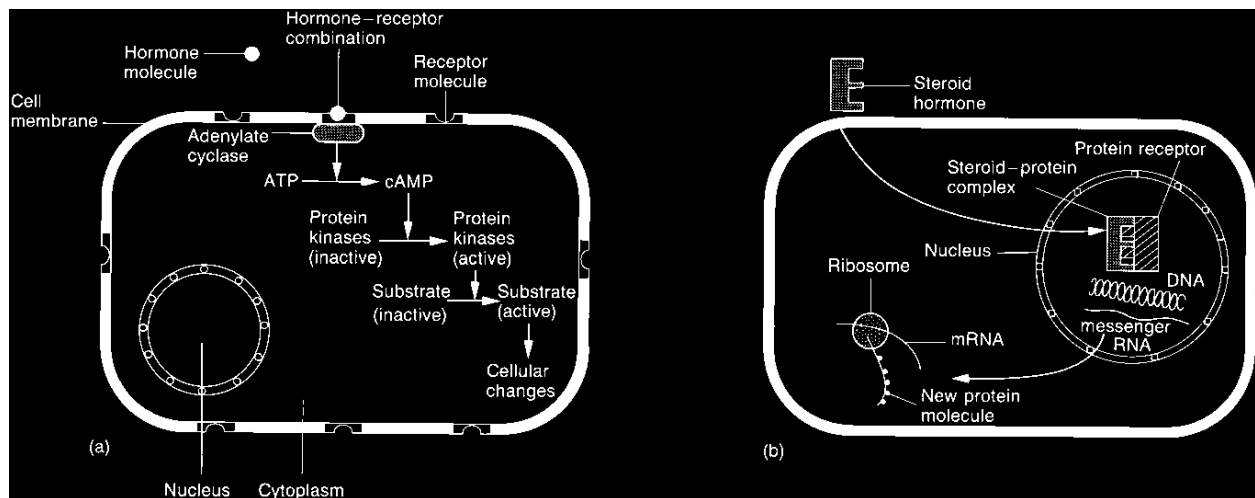
Fig. 33.10

Preovulatory surge of LH on day 18 of the oestrous species, processes that are of more immediate importance for the survival of the individual take top priority when an animal allocates available metabolic fuels among various energy-consuming activities (Fig. 33. 12). Hence, changes in the availability of metabolic fuels affect reproductive functions, through actions at multi-
the receptor activates an enzyme within the cell mem-

ple sites in the central nervous system, pituitary gland, brane that acts as a catalyst in a reaction that produces gonads and other peripheral organs (e.g. liver, skeletal a 'second messenger', e.g. cyclic AMP, that, in turn, muscles, adipose tissue). Negative energy balance leads alters the activities of the enzymes that produce the to metabolic trade-off in which reproductive activity is final and characteristic response by the cell (Fig. curtailed, sometimes suspended. The suspension leaves 33.11a).

the regulatory neuroendocrine apparatus intact; repro- The sensitivity of a target tissue to a particular ductive efforts can be resumed when the energy deficit hormone will vary depending upon the number of spe- has been repaired. cific receptors it has available for binding that hormone. In many simple-stomach species the neural mecha- At any time, the number of receptors depends on the nisms that control the pulsatile release of GnRH (and, balance between degradation and synthesis of recep- consequently, LH secretion and ovarian function)

tors. A hormonal stimulus may increase the sensitivity appear to respond to minute-by-minute changes in the of a target tissue by increasing the rate of synthesis of availability of metabolic fuels (Bronson, 1989; Wade & the specific receptors; a good example of this 'up regu- Schneider, 1992). The response in ruminants may not be lation' is the increase in LH receptors induced in the as immediate but it is significant, nonetheless. Accord- granulosa cells of the preovulatory follicle by FSH ing to Roche et al. (2000), cattle are very sensitive to



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Fig. 33.11

Interaction of hormones with target cells. In (a) a glycoprotein (the 'first messenger') binds with receptors in the cell membrane and activates adenylate cyclase in the membrane. Adenylate cyclase converts ATP to cyclic AMP (the 'second messenger') which, in turn, activates various protein kinases. The

resultant cascade of enzymatic activity produces the characteristic cell response to the endocrine signal. In (b) a steroid hormone enters the nucleus where it binds to its receptor. The receptor–hormone complex binds to a particular region of DNA and activates particular genes. The activated genes promote the synthesis of particular messenger RNA molecules that pass from the nucleus to the cytoplasm where they induce the synthesis of proteins (mainly enzymes) that are responsible for the characteristic actions of that hormone in that cell type.

Resting metabolism

Storage

Thermoregulation

1

Locomotion

Stress responses

Metabolic

Food

Partitioning

fuels

Lactation

2

Immune responses

Fig. 33.12

Partitioning of energy by a lactating cow. The sizes of the arrows indicate the order of priorities rather than the quan-

Growth

3

Reproduction

tities of metabolic fuels allocated to the different activities.

acute nutritional deprivation: after a lapse of three to five days, follicular growth is impeded and ovulatory responses that mobilize energy, promote its expenditure, and leave less available for use by the results in delayed puberty and, in adult females, it HPG axis; concurrently, cortisol released by the causes extended postpartum anoestrus and prolonged hypothalamic–pituitary–adrenal (HPA) axis acts as a calving intervals.

counter-regulatory signal at all critical points in the HPG axis and at target tissues for the gonadal steroids (see Fig 67.6). Short-term acute stressors active at Stress (see Chapter 67)

critical times in the reproductive cycle of a female

The effects of stress on reproductive functions are can disrupt the orderly timed sequence of hormonal complex: during the stress response increased activity changes on which reproductive success is predicated. In Reproductive Physiology in Cattle • 479 such circumstances, failure to establish a pregnancy may from activated immunocompetent cells can evoke a be a 'one-off' eventuality without residual effects on the variety of neural, neuroendocrine, metabolic and next attempt. On the other hand, stressors that gener-behavioural responses regulated by the CNS; the effect ate allostatic load (see Chapter 67) may lead to at-is akin to activation of the central neural and hormonal tenuation or suspension of reproductive activities for components of the stress response (see Chapter 67): the periods of weeks or months until the counter-regulatory main routes of communication are via the sympathetic signals are quenched. In such cases, inhibition of the nervous system and the HPA axis. The noradrenaline pulsatile release of GnRH may be the most significant and glucocorticoids released by the counter-regulatory

action in the context of self-preservation of the organ-loops invoked by the cytokines can have profoundism, but inhibition at other critical points in the HPG biological effects in terms of the expenditure of energy, axis may be responsible for more persistent carry-over immunosuppression and interference with the functions during the recovery phase. Recovery of reproductions of the HPG axis. Thus, for instance, the cross-talk ductive efficiency requires that the appropriate bio-between the brain and the immune system evoked by chemical features (receptors, G-proteins, transcription a severe bout of mastitis in early lactation can have a factors, other signalling proteins, enzymes, metabolic seriously negative impact on the restoration of cyclic cascades) be restored and functional at each of the ovarian activity and fertility.

critical points in the HPG axis. As happens at puberty and, again, in the early postpartum period, it may require more than one attempt to fully up-regulate the

Puberty (see also Chapter 5)

normal cascade of the HPG axis and reinstate the

ability to reproduce.

Puberty, the process by which animals become capable of producing offspring, is often given a very restricted definition. According to Short (1984), 'we generally
Neuro-immune interactions

refer to puberty as that moment at which the female

In recent years it has become increasingly evident that
first comes into oestrus and ovulates, or the male first

the interactions between behavioural, neural, endocrine produces spermatozoa in his ejaculate'. The trouble

and immune processes are as important to our under-

with this widely-held perception of puberty is that it

standing of homeostasis as are the interactions within
concentrates exclusively on the apparent end-point of
each of the individual disciplines. In the present

a protracted and complex physiological process and
context, we focus on the bidirectional network through

suggests that puberty is a discrete event that can be

which the CNS and the immune system exert profound
assigned to a particular day (or moment!). There is the
effects on each other. The nervous system impacts upon

additional problem that the apparent end-point (first the immune system through several routes, systemic release of the gametes) is not necessarily conclusive and local, including neuroendocrine pathways (e.g. the evidence that the animal has now reached the stage at HPA axis), autonomic nerves and systemic nerves. In which it can reproduce itself; for instance, it is clear that the reciprocal relationship, cytokines (see Box 33.1) many heifers may not be capable of doing so until well after first ovulation.

In fact, the onset of puberty is a gradual process that

Box 33.1.

Cytokines

is in train for several months during which there are significant morphological, physiological and behavioural

more cell types that regulate the activities of other cells. They responses to the progressive expression of both the

bind to specific receptors on the cell membranes of target

steroidogenic and gametogenic activities of the gonads.

cells and, like hormones, they may act locally in autocrine or The essential feature of the process is the maturation of

paracrine fashion, or they may be released into the blood—the brain–hypothalamic–pituitary–gonadal axis, which stream and interact with target cells elsewhere in the body.

culminates in the adult pattern of reproductive activ-

More than 100 cytokines have been identified; they are clas-

ity. The components of the hypothalamic–pituitary–

sified into several categories: interferons, insulin-like growth factors, interleukins, tumour necrosis factors, growth factors, gonadal axis are present before birth and, even at that

transforming growth factors, and so on. Amongst a vast range

stage, each component is capable of responding to

of activities, they participate in haematopoiesis, cell prolifer-appropriate hormonal signals. During gestation, steroid

eration, immune responses, inflammation, wound healing,

hormones released from the placenta have an

modulation of cell metabolism. The nervous system can

inhibitory effect on the fetal hypothalamus that

produce cytokines and it can respond to cytokines released

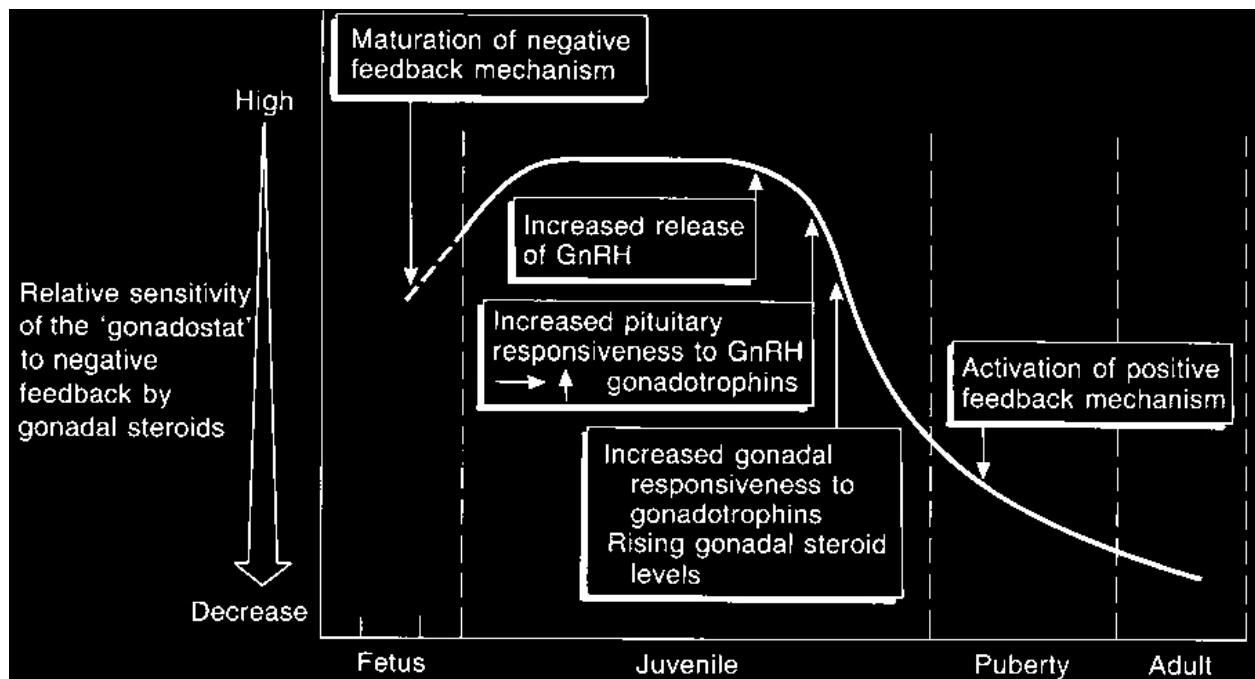
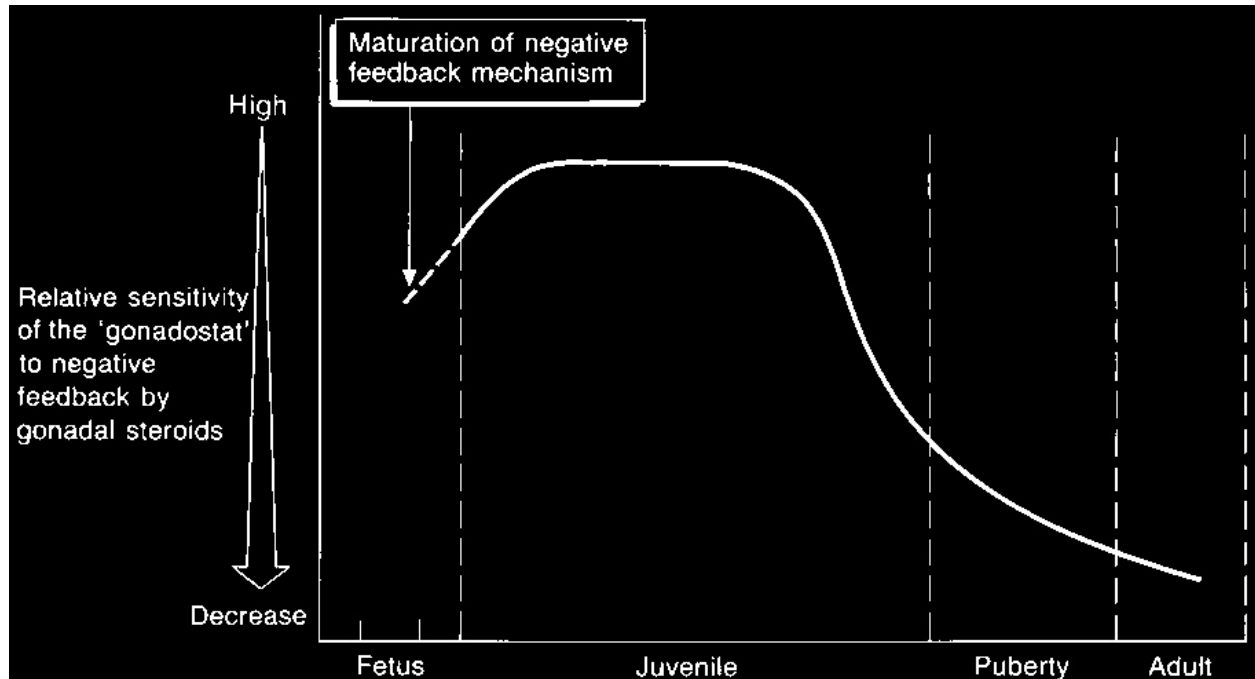
suppresses the activities of the axis. After birth the

peripherally; for instance, in fever, in sickness behaviour

axis becomes an independent regulatory system that

and in modulating neuroendocrine responses – which is the

secretes increased amounts of gonadotrophins and
 context in which we have introduced the term.
 gonadal steroids. However, the system is restricted to a



relatively low level of activity (juvenile level); for gonadotropes to GnRH and to an increase in the instance, in the female the level of activity is well below responsiveness of the gonads to the gonadotrophins that required for ovulation or cyclic ovarian activity.

(Fig. 33.14). Greater quantities of gonadotrophins are

The restraint is due partly to fine sensitivity of the released from the pituitary gland and, in turn, greater GnRH pulse generator to negative feedback by quantities of steroids are released by the gonads. For gonadal steroids and partly to inhibitory inputs from instance, in the female the sensitivity of the hypothalamic neural circuits that modulate the activities of the thalamus to the negative feedback effect of oestrogen hypothalamus.

declines progressively and, as it does so, the increasing

The transition to sexual maturity is brought about by quantities of oestrogen from the ovaries can no longer progressive changes in the neuroendocrine activities of hold the axis within the juvenile pattern of activity.

the axis in response to a variety of internal and envi-

There is a gradual movement of the regulatory system towards the adult pattern (Figs 33.13 and 33.14). Eventually, the ovaries produce a surge of oestrogen that exerts a positive feedback effect on the pulse generator of the hypothalamus to the negative feedback effects of gonadal steroids (Fig. 33.13) so that the frequency of surge of LH that leads to ovulation.

GnRH pulses is increased progressively. This leads As our knowledge of these changes expands, there is to an increase in the sensitivity of the pituitary increasing evidence that sexual maturity is regulated to

Fig. 33.13 *Maturation of the negative feedback system and changes in set point of the hypothalamic gonadostat extending from fetal life through puberty to adulthood (modified from Grumbach et al., 1974).*

Fig. 33.14

*The change in set point
of the hypothalamic gonadostat
(denoted by the dashed and solid
lines) and the maturation of the neg-
ative and positive feedback mecha-
nisms from fetal life to adulthood
(from Grumbach et al., 1974).*

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*a considerable extent by changing patterns of neural
generator is closely coupled with energy balance and
control over the hypothalamus. These alterations in
that it is allowed to progress from the juvenile to the
intrinsic neural activity do not appear to be dependent
adult level of activity in the female only when the ener-
gonadal hormones; however, they play a crucial role
getic status appears to be adequate to sustain pregnancy
in that they gradually release the GnRH pulse genera-
and lactation. It was anticipated that the hormone
tor from the restraints exercised by the central nervous
leptin, produced by adipose tissues, might prove to be*

system during the juvenile period, thus enabling it to the signal that triggers the onset of puberty: current respond to the gonadal steroids in the adult manner. It evidence is that leptin is just one of many permissive is evident that the classical 'gonadostat' hypothesis does metabolic factors that allow pubertal development to not furnish an entirely satisfactory explanation of the proceed; it may act as a permissive signal to increase onset of puberty in all its details. Nevertheless, it does GnRH secretion after the pulse generator has been sen- provide a conceptual framework that is adequate for sitized to energy balance by other developmental or the purposes of this text.

metabolic cues; it is not the trigger for the beginning of The first ovulation is likely to be silent; it appears puberty (Urbanski, 2001; Smith et al., 2002).

that the hormonal requirements for the expression Selective culling is an integral part of efficient of oestrus are small quantities of progesterone from a management of the dairy herd. It can be practised suc- regressing corpus luteum followed by a surge of oestro-

cessfully only when the stockperson has available an
gen from the preovulatory follicle – a sequence that
adequate number of high-quality heifers due to calve
is absent at the time of first ovulation. Thus, the first
down at the appropriate time. This requires careful
oestrus observed by the stockperson indicates that the
management of the prepubertal animal to ensure that
process of puberty is well advanced but it does not
the reactivation of the hypothalamic pulse generator is
provide a precise date for the ‘moment’ of puberty.
not delayed unduly by environmental influences, prin-
The earliest possible maturation of the individual
cipally nutritional factors and subclinical diseases.
components of the reproductive axis (and, therefore,
Similarly, young bulls intended for breeding purposes
of the axis itself) is determined by genes, i.e. there are
require careful management during the juvenile period
inherent breed variations in the onset of puberty.
because the onset of puberty in the male is also influ-
However, in all breeds this process is subject to delays
enced by a variety of factors (including breed, season

caused by a variety of endogenous and exogenous influences of birth, energy intake and liveweight gain) that affect the reactivation of the hypothalamic GnRH pulse person to ensure that the heifer is ready for breeding at generator.

the desired time. The principal environmental factors that influence the onset of puberty are: season of birth, level of nutrition, growth rate, photoperiod, high am-

Male physiology

bient temperatures, intercurrent diseases and presence of the male.

In the male, puberty is associated with changes in the Specific reference to these factors will be made in the pattern of LH secretion, a gradual increase in the clinical segment of the text. For the moment, suffice concentration of testosterone in the blood, rapid growth of it to say that, as a general guideline, the stockperson the testes and the initiation of spermatogenesis. In should devise a management strategy in the knowledge essence, the adult pattern of GnRH release is attained

that the onset of puberty is a labile process that is conditioned by competition between the reproductive system and the other body systems of the growing animal for energy and specific nutrients. Reproduction, particularly in the female where it involves pregnancy performed best at temperatures somewhat lower than body core temperature.

to which Nature assigns a relatively low priority for the prepubertal animal. Evidence is accumulating from work on a number of species that inadequate intake of energy or nutrients can depress or abolish the activities by which the scrotal temperature is reduced are of the GnRH pulse generator in the juvenile animal.

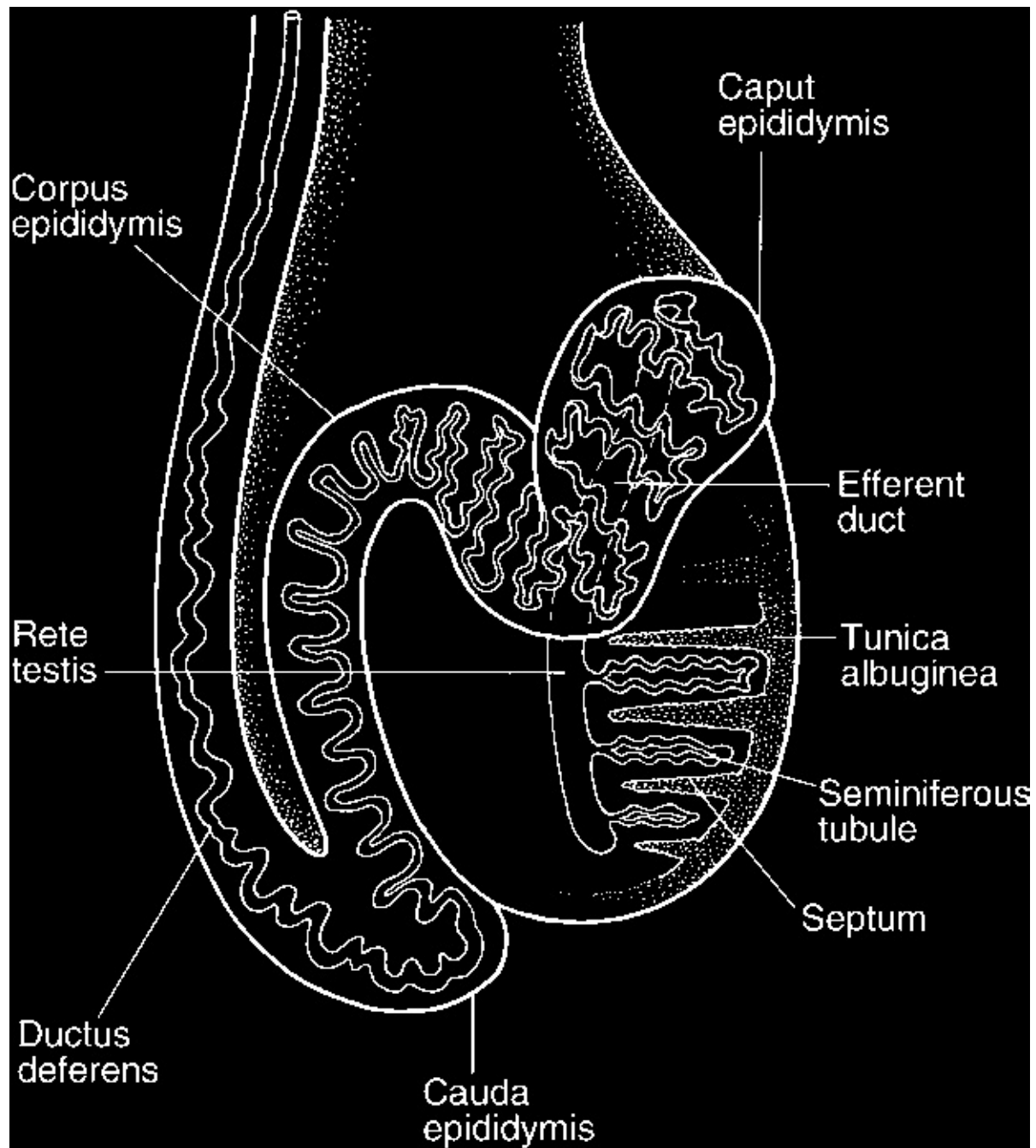
as follows:

This has been attributed, variously, to deficiencies in body weight, in rate of growth, in fat content or in (1)

Precooling of the arterial blood supply as it passes fat : lean ratio. There is no agreement on the particular through the vascular cone in the spermatic cord.

cue(s) that inhibit(s) the pulse generator but there is

The vascular cone consists of a coiled segment of general acceptance of the thesis that the function of the the spermatic artery that is surrounded by the



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pampiniform plexus of the spermatic vein.

the germ cells during their migration from the base to

Because of this anatomical arrangement the arte-

the luminal surface. The plasma membranes of adjacent arterial blood and the venous blood are flowing in parallel but in opposite directions; this allows for an efficient countercurrent exchange of heat between each cell (Fig. 33.17). These junctions constitute the blood–testis barrier that precludes the passage of many substances from the blood delivered to the testes is several degrees below body core temperature.

blood or interstitial fluid into the lumen of the seminiferous tubule. They also divide the intercellular spaces into two compartments: the basal compartment that

(2)

Sweating from the many sweat glands in the scrotal skin.

Physical contact with cold ground or other cold

(3)

contains the undifferentiated germ cells and the

objects.

adluminal compartment that provides the appropriate microenvironment for the more differentiated germ

In very cold weather, the temperature at the base of cells (see below).

the scrotum may be up to 7°C below core values but the

The basal surface of the Sertoli cell has specific recep-

bull can attempt to curtail the drop in temperature by

tors for FSH. The cells respond to the gonadotrophin

drawing the scrotum closer to the (relatively) warm

by secreting (i) a nutrient fluid that sustains the germ

body wall by contracting the cremaster and dartos

cells in the intercellular spaces, (ii) androgen-binding

muscles.

protein that binds and transports testosterone to the

The gametogenic function of the testes is much more

epididymis and (iii) inhibin that modulates the secre-

heat sensitive than the steroidogenic function; for

tion of FSH by negative feedback at the pituitary gland

instance, a retained testicle in a cryptorchid animal

(Fig. 33.18). Sertoli cells also have receptors for andro-

(‘rig’) may secrete androgens but the seminiferous tubules will remain infantile in structure and they will not produce spermatozoa. The retained testicle has a withdrawn.

propensity to develop neoplasms (see p. 182).

Sertoli cells are resistant to relatively high levels of heat, ionizing radiation and many toxins (e.g. cadmium, nitrofurans, cytotoxins) that destroy differentiating

Morphology of the testis

germ cells.

The testis is surrounded by a thick fibrous capsule, the tunica albuginea, from which septa project inwards to divide the substance of the testis into lobules (Fig. 33.15). Each lobule contains one to four highly convoluted seminiferous tubules and interstitial tissue that fills the spaces between the convolutions.

The interstitial tissue contains blood vessels, lymphatic vessels, nerves and the steroid-secreting Leydig cells.

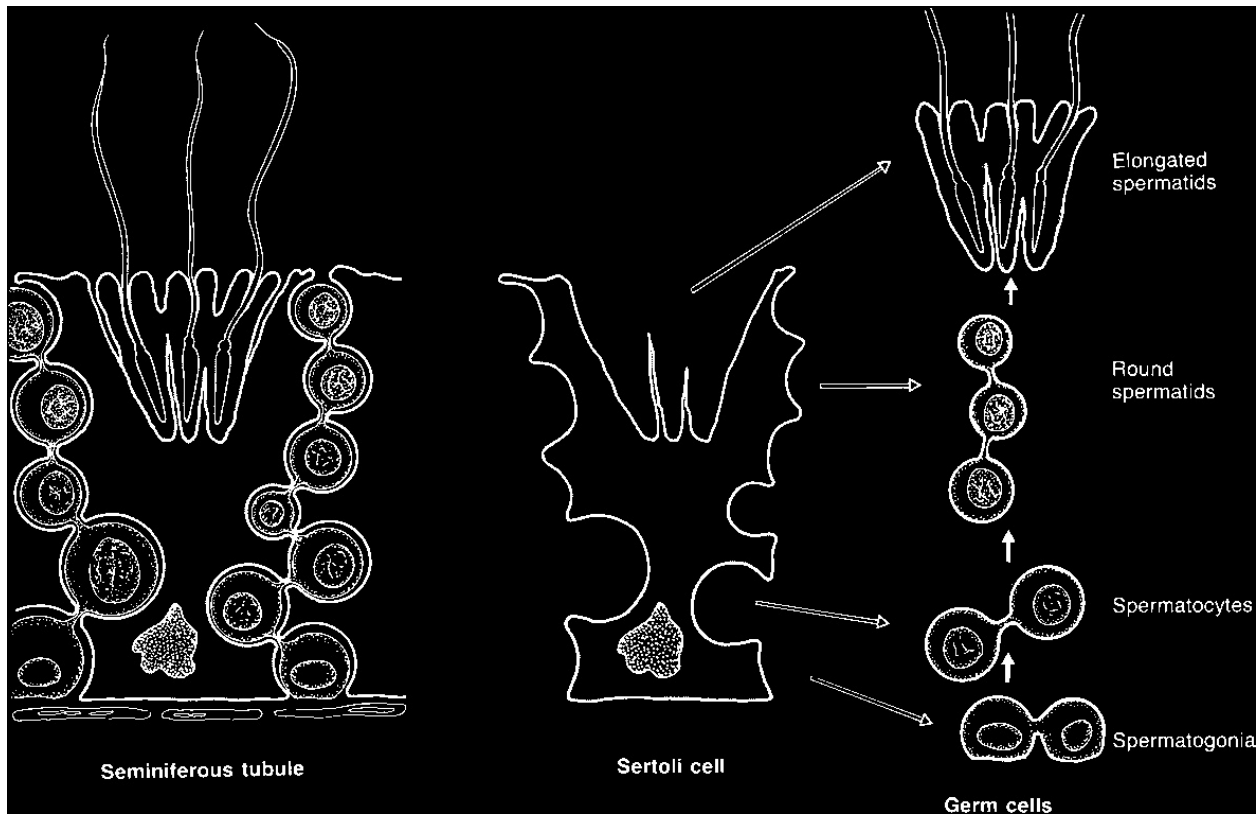
The seminiferous tubules are about 200 mm in diameter and they may be up to 70 cm long; they open at both ends into the rete testis (Fig. 33.15). The tubules contain two populations of cells: (i) a fixed population of non-proliferating somatic cells, the Sertoli cells, and (ii) a migratory population of proliferating, differentiating germ cells (Fig. 33.16).

Sertoli cells

The Sertoli cells have been described as the ‘backbone’ of the tubule. They are columnar cells that rest on the basement membrane and extend the full depth of the epithelial layer; they envelop the developing germ cells in deep recesses in their lateral walls and, ultimately, in their luminal surfaces. The Sertoli cells continually alter shape to accommodate the morphological changes in

Fig. 33.15

Outline morphology of the testis.



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Fig. 33.16

The histology of the seminiferous tubule. The diagram depicts the Sertoli cell and the germ cells in intimate contact as they are in vivo and, also, separately to show the outlines of the individual cells. (Adapted from Fawcett, 1994.) Germ cells

still attached to the Sertoli cell; the final stages take place at the luminal surface. The conversion of the spermatid to the spermatozoon does not involve cell division (see Table 33.1) is beyond the scope of this section, merely morphological reorganization (referred to

text. Suffice it to say that the spermatogonia are the as spermiogenesis). The principal changes are condensation of the nucleus and acquisition of the acrosome in the head region, reorganization of the mitochondria in the middle piece, development of the tail and loss of excess cytoplasm that moves caudally and is shed as the residual body, which is phagocytosed by the Sertoli cell. Cohorts of spermatogonia from this pool are moved to the adluminal compartment while one spermatogonium from the group remains behind in the basal compartment. It enters a resting phase during which it is highly resistant to ionizing radiation and toxic agents.

(Fig. 33.19).

The intercellular bridges are lost with the residual body and each spermatozoon now is a separate cell, that soon escapes from the luminal surface of the Sertoli cell (spermiation). When the fully developed spermatozoon is released from the Sertoli cell, it is highly resistant to ionizing radiation and toxic agents.

spermatozoon is released into the lumen of the semi-
This will be the stem cell from which the next cycle of
niferous tubule it is non-motile. It is transported pas-
spermatogenesis will begin (two weeks later). In the
sively from the tubule into the rete testis and thence to
meantime, the newly arrived cells at the base of the
the epididymis. Motility is acquired during passage
adluminal compartment proceed to undergo meiosis as
through the epididymis.

primary spermatocytes (first meiotic division) and sec-
In summary, during spermatogenesis the germ cells
ondary spermatocytes (second meiotic division). The
advance through three major processes:

haploid secondary spermatocytes are still attached to
each other by intercellular bridges and after a short

(1)

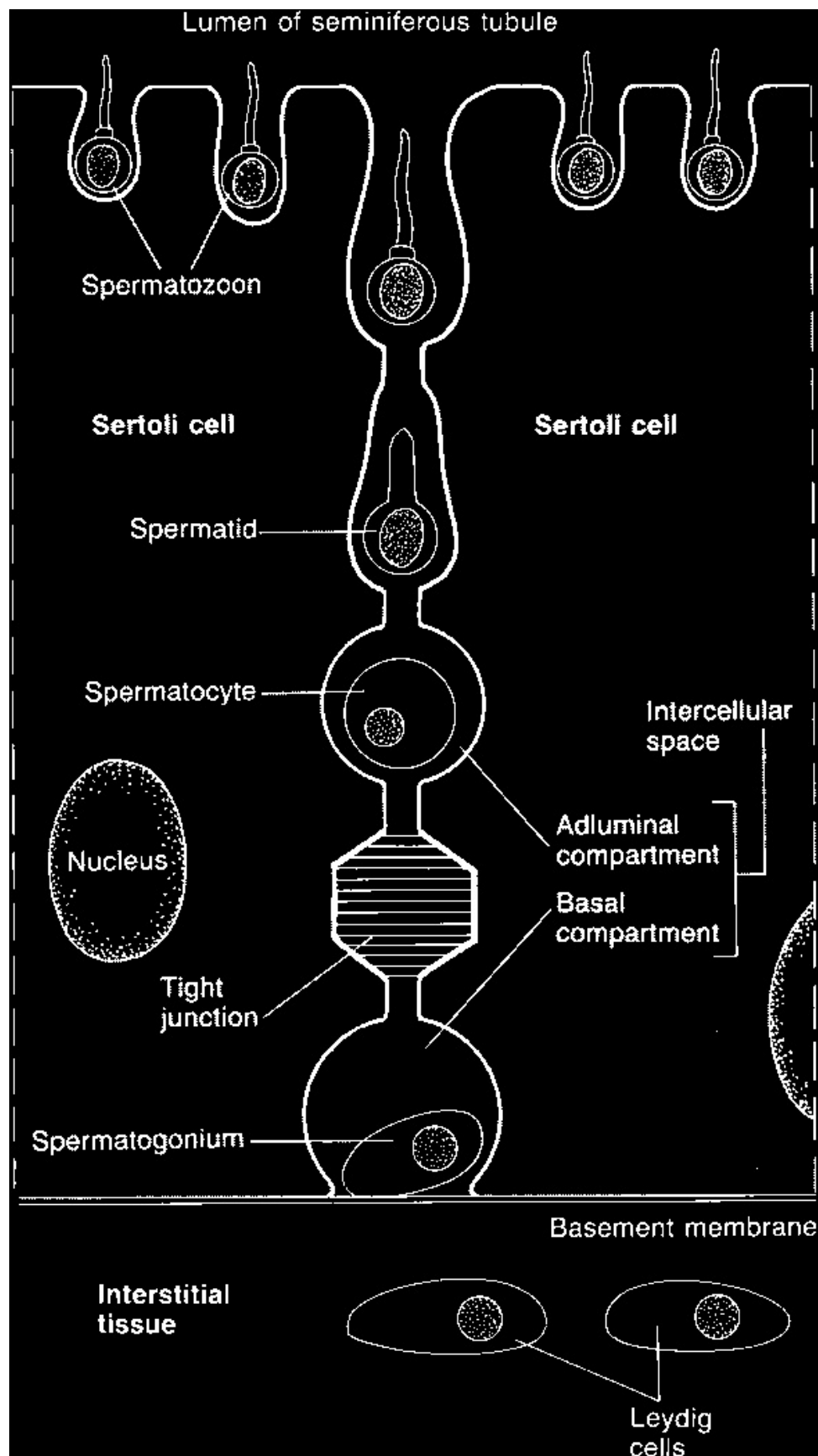
Proliferation of stem cells by mitosis in the basal
period each of them divides to yield two spermatids. As
compartment.

the process of spermatogenesis proceeds the germ cells

(2)

Reduction of the chromosome number by meiosis

*move progressively towards the lumen of the tubule,
in the adluminal compartment.*



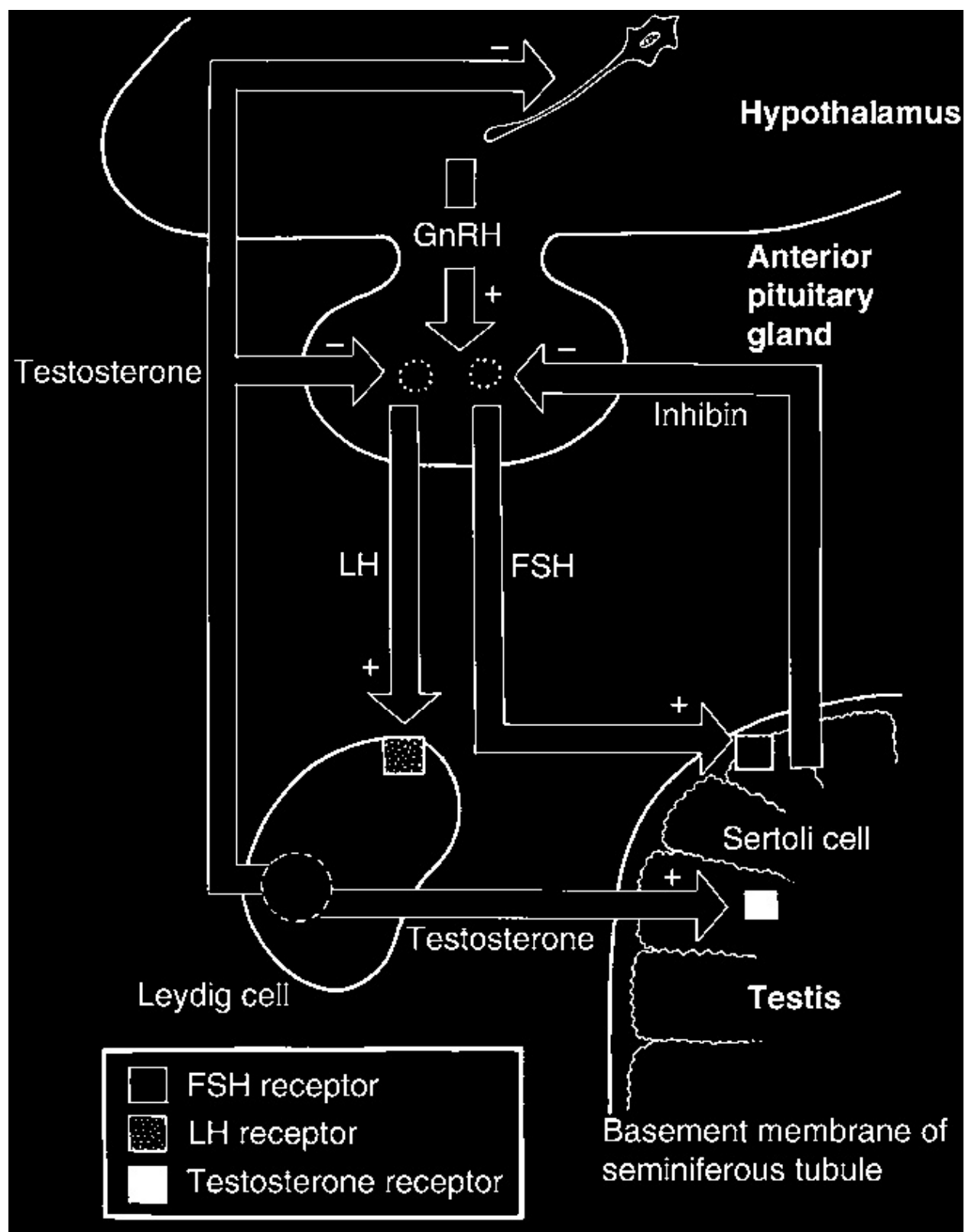


Fig. 33.18

Hormonal interactions in the hypothalamic–pituitary–testicular axis.

Table 33.1

Principal stages in the development of spermatozoa (spermatogenesis).

Fig. 33.17

The relationship between the germ cells and Sertoli cells during spermatogenesis.

Cell type

Number of chromosomes

Spermatogonium

$2n$

Primary spermatocyte

$2n$

(3)

Morphological transformation of a conventional

Secondary spermatocyte

n

cell (the spermatid) into the very specialized struc-

Spermatid

n

ture of the spermatozoon (spermiogenesis) in the

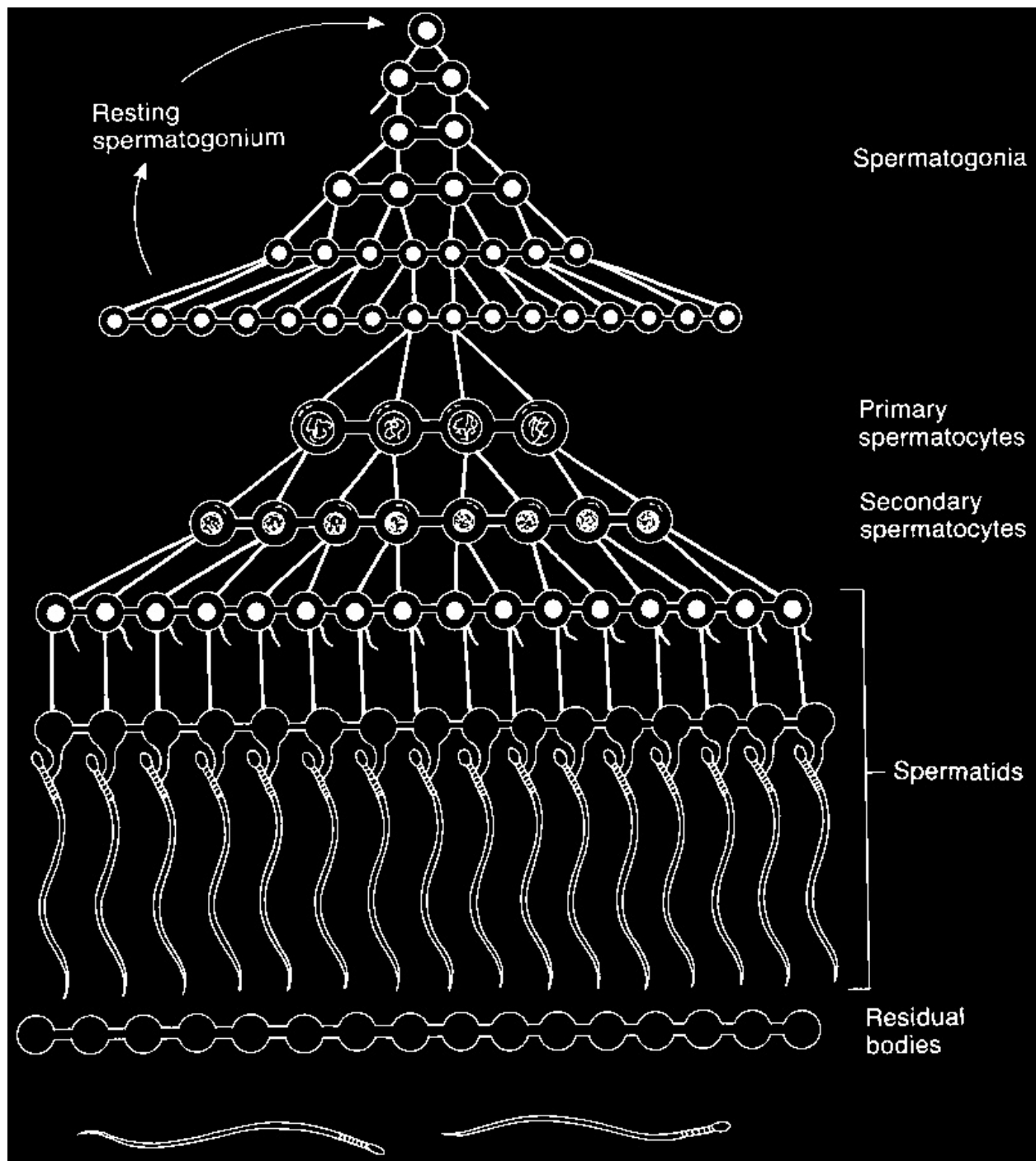
Spermatozoon

n

adluminal compartment.

This sequence of mitotic, meiotic and packaging events requires 56 days for completion in the bull. It is a very orderly sequence in which each of the component cells will be in train in the seminiferous epithelium at any has a fixed lifespan so that cellular differentiation proceeds at a fixed rate. The spermatogonium that reverts to the resting phase remains quiescent in the basal compartment for 14 days (equivalent to 25 per cent of the entire cycle) before it enters mitosis and begins the next seminiferous tubule will reveal a characteristic set of spermatogenic cycle. The cells derived from the new cell associations at any one time. In most mammals the

cycle advance in a similar orderly fashion behind the same set of cell associations is seen at all points in the cells from the previous cycle as they move progressively circumference of the tubule (this represents the 'cycle towards the luminal surface. In fact, this arrangement of spermatogenesis'); however, adjacent segments means that four successive cycles of spermatogenesis of the tubule tend to have a different set of cell



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Fig. 33.19

The process of spermatogenesis.

(Reproduced from Fawcett, 1994.)

associations, each either in advance of or behind the head of the sperm and allowing the lytic enzymes its neighbours (this constitutes the 'wave of to escape through the resultant pores (see p. 499). spermatogenesis').

Some of the progeny of the original spermatogonia do

Neuroendocrine control

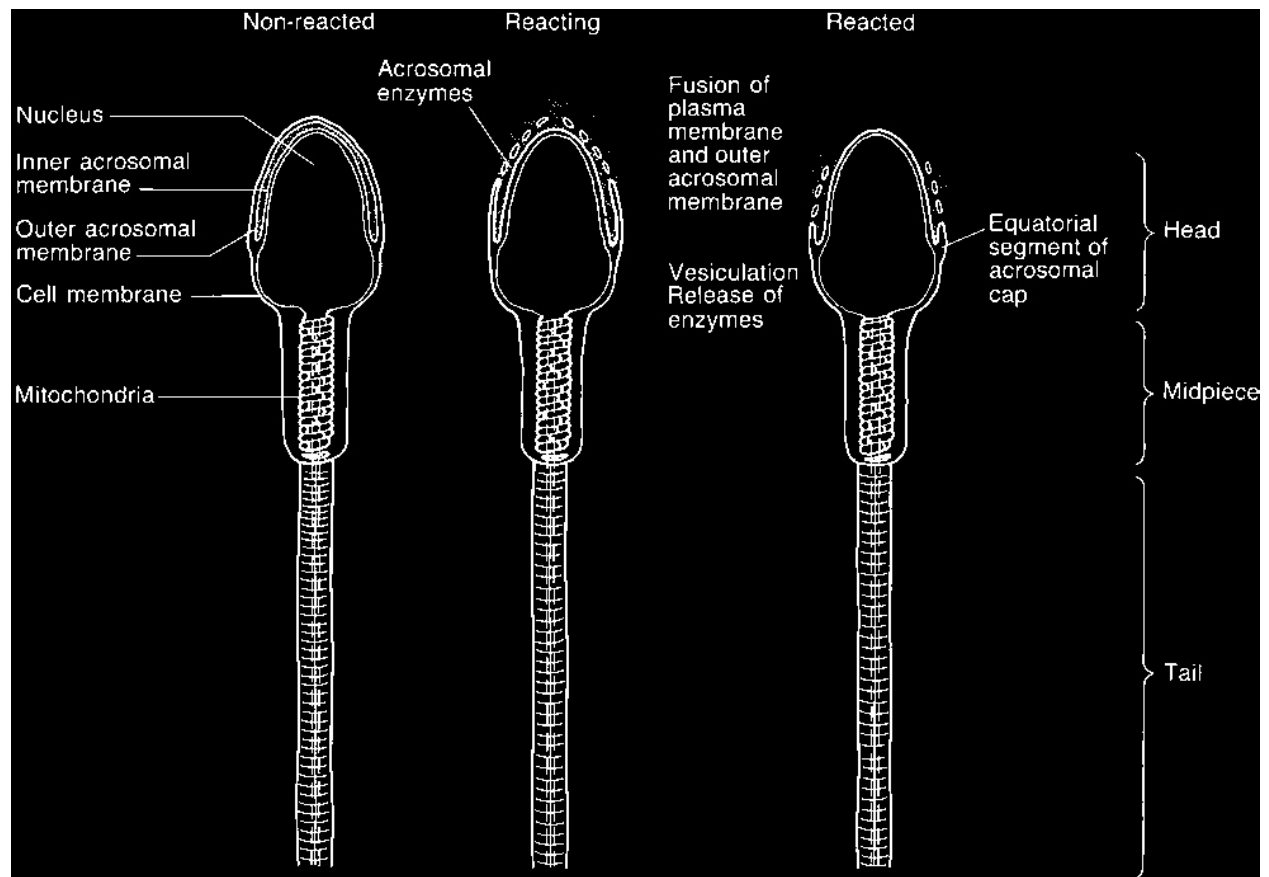
not complete the full course; large numbers (perhaps 20 per cent) of differentiating germ cells die without reaching maturity and these are removed by the Sertoli cells. The steroidogenic functions and the gametogenic functions are segregated anatomically: androgen synthesis by the Leydig cells in the interstitial spaces and spermatogenesis within the seminiferous tubules (Fig.

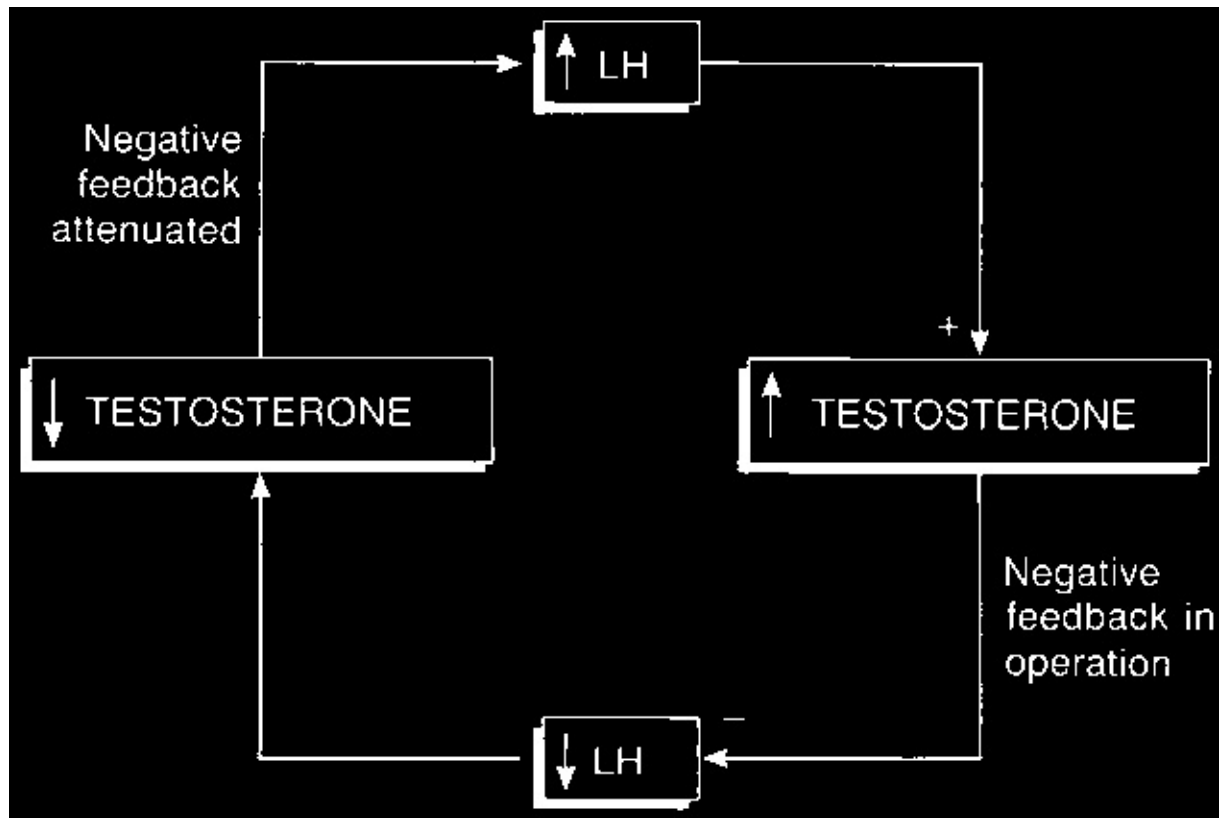
The spermatozoon

33.17). Both functions are controlled by the pituitary gland through the secretion of the gonadotrophins. The essential features of the mammalian spermatozoon are depicted in Fig. 33.20. Only one structure warrants Luteinizing hormone binds to specific receptors on the

further comment at this juncture: the acrosome, an organelle that undergoes significant morphological changes as an essential prelude to fertilization. The acrosome contains a number of lytic enzymes (particularly hyaluronidase, neuraminidase and acrosin); it has been described as a modified lysosome and, like all lysosomes, it has a bilaminar structure. The inner acrosomal membrane is adherent to the nuclear membrane, while the outer acrosomal membrane underlies the cell membrane. The negative feedback effect of testosterone on LH secretion is exercised at the level of the hypothalamus (influencing the secretion of GnRH) and at the acrosome reaction in the female genital tract, the

the pituitary gland (modulating the responsiveness of outer acrosomal membrane forms point fusions with the gonadotropes to GnRH). In this manner the the cell membrane creating a sequence of vesicles over hypothalamic–pituitary–Leydig cell axis maintains the





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Fig. 33.20

The spermatozoon undergoing the acrosome reaction.

There is no doubt that an intact hypothalamic–pituitary–testicular axis is essential for normal testicular function; however, apart from the fact that FSH is essential for the initiation of the process in immature animals, it is unclear what specific roles in the process of spermatogenesis are played by the individual

hormones that constitute the axis. On the basis of information available in the literature, the following deductions have gained wide acceptance:

- *Differentiating germ cells lack hormone receptors and, therefore, they are not affected directly by the hormones.*

Fig. 33.21

The reciprocal relationship between the concentra-

- Sertoli cells have specific receptors for FSH and for testosterone and LH in the general circulation. androgens but not for LH.
- Testosterone is required for the first mitotic division of the stem cell in the basal compartment.
- Testosterone is necessary for the reduction division of the primary spermatocyte. concentration of testosterone in the general circulation of the primary spermatocyte. within normal limits.
- FSH is essential for the final steps in the maturation of the spermatids. Sertoli cells respond to FSH by secreting fluids and proteins that sustain the differentiating germ cells. Fol-

- *There is controversy as to whether or not testostolicle stimulating hormone is absolutely essential for the terone is required for the earlier steps in the matuinitiation of spermatogenesis in immature animals but ration of the spermatids.*

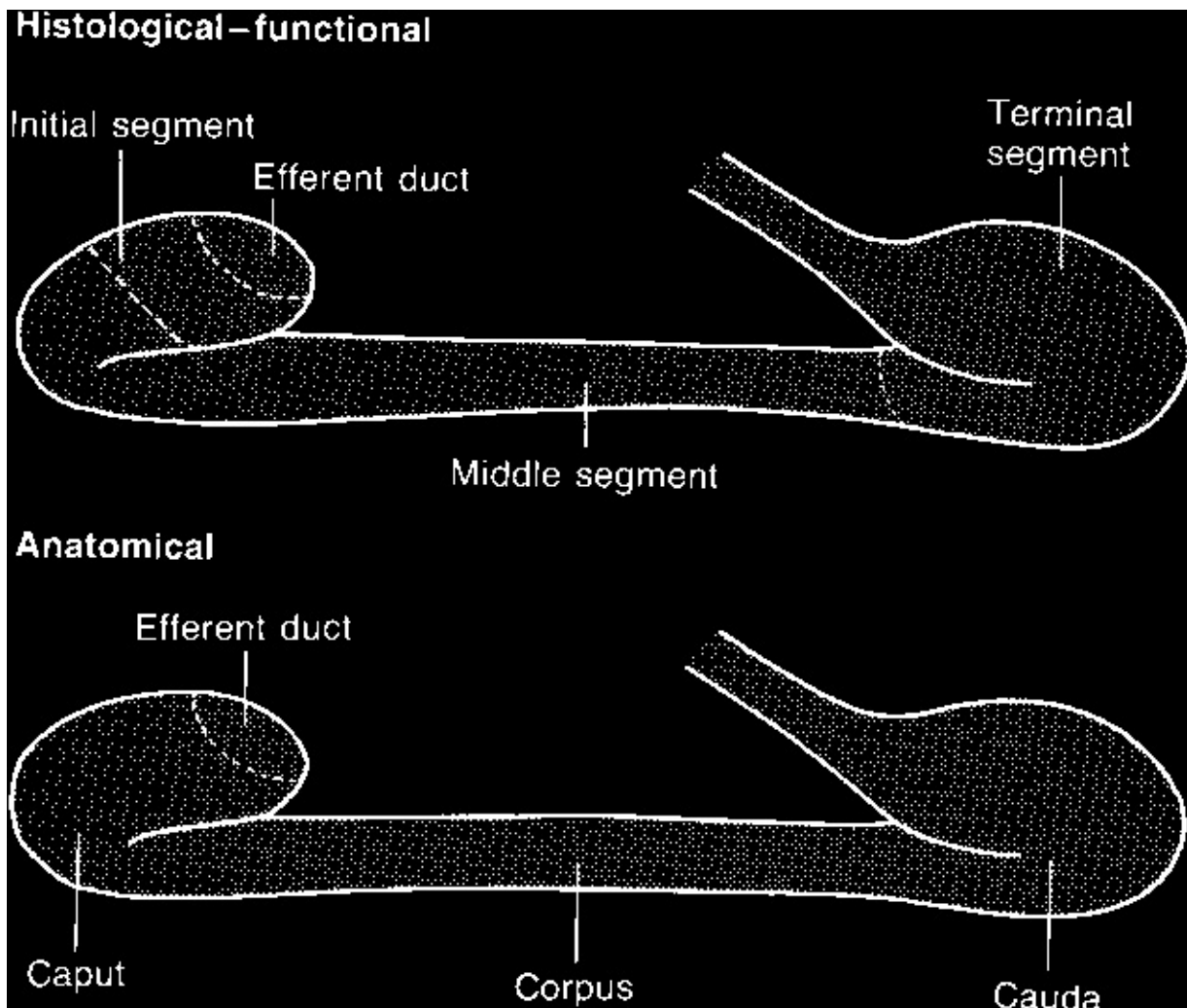
once the normal germinal epithelium has been estab-

- *It is likely that all the other steps in spermatogene-lished FSH does not appear to be essential for the startsis can proceed without the specific intervention ofof each successive spermatogenic cycle. Another actiona particular hormone.*

of FSH is to stimulate the conversion of testosterone to oestradiol by the Sertoli cells. The Sertoli cells also

Thus, FSH and testosterone exercise direct actions on secrete inhibin, a polypeptide that inhibits the release the seminiferous tubules, while LH does so indirectly of FSH by the pituitary gland (Fig. 33.18).

via the production of testosterone.



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bull is an exception in that the bulk of the water has been absorbed by the efferent ducts before the contents reach the epididymis. The secretions from the epithelial cells of the initial and middle segments induce significant alterations in the functions and composition of most of the component parts of the spermatozoon. The principal functional changes are the acquisition of pro-

gressive motility and the ability to fertilize an egg. Fine structural changes have been observed in the plasma membrane, the acrosomal membranes, the nucleus and the tail. Changes in the composition of the plasma membrane appear to be central to the process of maturation of the spermatozoon as it passes through the epididymis; in particular, its protein composition is altered either by adsorption of components from the epididymal fluid or by modification or loss of existing components in response to enzymatic activity of the

Fig. 33.22

The regions of the bovine epididymis.

epididymal fluid. It is important to note that these biochemical changes are associated with the acquisition of the ability to fertilize but they do not complete the process of maturation – this will occur only after the
It should be noted that even where specific hormonal ejaculated spermatozoa have been capacitated within stimulation is essential, the hormone serves to ensure the female genital tract (see p. 499).

that the event occurs but it does not alter the rate of

Ejaculated spermatozoa have species-specific receptors that bind to the zona pellucida of the egg. The progress of spermatogenesis, which is a feature that is determined by genes rather than by hormones. Spermatozoa acquire these receptors in the epididymis, under the influence of testicular androgens.

The ability of the epididymis to facilitate maturation

The epididymis

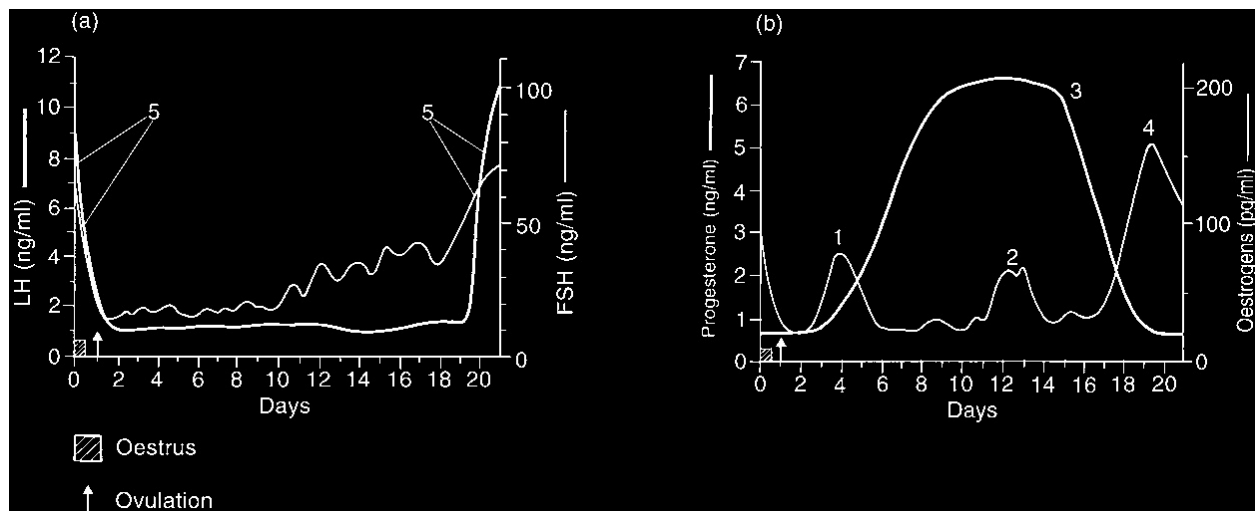
of the spermatozoa and to maintain the viability of the spermatozoa. When spermatozoa are transported from the rete testes in the cauda is critically dependent on testicular androgens. Obviously, some of the spermatozoa in the epididymis they are neither motile nor capable of fertilization. They acquire these attributes during passage through the efferent ducts to the initial segment of the epididymis. Testosterone reaches the epididymis in the arterial blood but it is probable that most of it comes via the efferent duct system bound to androgen-binding protein. The epididymis is a highly convoluted tubule lined by

protein. The epithelial cells of the epididymis have

The epididymis is a highly convoluted tubule lined by protein. The epithelial cells of the epididymis have

specialized epithelium and surrounded by connective
androgen receptors and they rapidly convert the testos-
tissue, which incorporates concentric layers of smooth
terone to dihydrotestosterone, which then regulates
muscles that increase in thickness from the initial
epididymal functions. It has been clearly established
segment to the terminal segment. Anatomists describe
that the maturation of spermatozoa is regulated by
the epididymis as consisting of three parts (Figs. 33.15,
dihydrotestosterone rather than by testosterone.
33.22): the caput (head), the corpus (body) and the
Movement of spermatozoa through the middle
cauda (tail). A more recent histological–functional clas-
segment is by continuous peristaltic contractions of
sification has designated at least eight regions in the epi-
the smooth muscles that surround the epididymal
didymis of the bull, but for the purposes of this text it
tubule. The rate of transport in this segment is not
is sufficient to refer to the three principal segments used
influenced by ejaculation and in the bull it is estimated
in that scheme: the initial segment, the middle segment

to take two to three days. It follows that the fertility of and the terminal segment (Fig. 33.22). The efferent sperm should not be depressed even when the bull is ducts empty into the initial segment, which is special-ejaculating frequently. By contrast, the muscles surrounding the terminal segment are relatively inactive. The middle segment, which includes most of the caput except when they are stimulated to contract during and corpus, is responsible for the sperm maturation. ejaculation; it follows that frequency of ejaculation can. The terminal segment, largely the cauda, stores the have a significant effect on the duration of storage of mature spermatozoa pending ejaculation. potentially fertile spermatozoa and on the numbers of. In many species the initial segment is the principal fertile sperm available for ejaculation at a particular site for absorption of water from the luminal fluids. The time.



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Seminal plasma

principal secretions (oestrogens and progesterone). For

problems associated with the oestrous cycle see

The fluid portion of the ejaculate is called the seminal

Chapter 35.

plasma. It consists of the testicular and epididymal

secretions in which the spermatozoa are suspended

when they pass from the cauda epididymis into the vas

Oestrus

deferens plus the secretions from the various accessory

It has been shown that behavioural oestrus results from

glands, which are added as the spermatozoa are pro-

exposure of the anterior hypothalamus to both proges-

pelled along the vas deferens and the urethra during terone and oestrogen in physiological concentrations ejaculation. The seminal vesicles contribute fructose, and in the proper temporal sequence. Progesterone on the primary source of energy for the spermatozoa. its own inhibits oestrus. Furthermore, in the absence of Prostaglandins in the seminal fluid may assist transport priming by progesterone, physiological concentrations of spermatozoa within the female tract by stimulating of oestrogen fail to elicit the signs of oestrus; thus, in the contraction of smooth muscle. The bull ejaculates 1–15

pubertal heifer the first preovulatory follicle produces ml of semen containing $0.8\text{--}2.0 \times 10^9$ spermatozoa/ml. a surge of oestrogen that feeds back positively on the (Infertility in bulls is dealt with in Chapter 38 pulse generator and induces an effective ovulatory and some aspects of artificial insemination in Chapter surge of LH, but it does not induce behavioural oestrus. 39.)

In the adult cow the normal sequence is a high concentration of progesterone (P4) for 10–14 days followed

by a rapid decline in P4 and an immediate increase in

Female physiology

oestrogen from the growing preovulatory follicle (Fig. 33.23). This sequence results in behavioural oestrus that appears to be coupled with the preovulatory surge of

Oestrous cycle

LH so that ovulation occurs approximately 24 hours

By definition, the oestrous cycle is the interval between after the onset of oestrus.

the onset of two successive periods of sexual receptiv-

The duration of oestrus may vary from 8 to 30 hours

ity (oestrus). Both the expression of oestrus and the

and the behavioural signs may recur frequently

duration of the cycle are the result of cyclic changes in

throughout the period or they may be evident during

the ovaries that involve two temporary endocrine struc-

two shorter periods at the beginning and end with a

tures (the ovarian follicles and corpora lutea) and their

quiescent period between them ('split oestrus').

Fig. 33.23

Changes in concentrations of gonadotrophins (a) and ovarian steroids (b) in

peripheral plasma during a 21-day oestrous cycle in the cow. During most of the cycle progesterone (P4) secreted by the corpus luteum (CL) is the dominant hormone, exercising strong negative feedback on the hypothalamic–pituitary axis. However, the tonic concentrations of gonadotrophins released during the luteal phase induce two waves of follicles that are associated with small peaks of oestrogen (1, 2). When the CL regresses, the concentration of P4 declines (3) and oestrogen concentration climbs to a peak, the oestrogen surge (4), which triggers a surge of gonadotrophins (5) that induces ovulation.

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Length of the cycle

Box 33.2.

Some regulators of follicular growth.

The cow is a polyoestrous animal and, if she is not preg-

From the pituitary gland

nant, she will tend to return to oestrus repeatedly

Follicle stimulating hormone (FSH)

throughout the year at intervals of 18–24 days (mean =

Luteinizing hormone (LH)

21 days). By convention, the day of oestrus is desig-

Prolactin (PRL)

nated day 0 and the length of the cycle is calculated

Ovarian steroids

from that baseline.

Androgens

There can be considerable variation (within the

Oestrogens

normal range) in the length of the oestrous cycle: even

Progesterone

successive cycles in the same cow may vary by several

Other regulatory factors

days. Most of this variation is due to differences in the

Inhibin

duration of the luteal phase of the ovarian cycle, i.e. in

Activin

the functional lifespan of the individual corpus luteum.

Insulin-like growth factor

Luteal regression may begin as early as day 15 or as late

Insulin

as day 19 of normal (20–24-day) cycles.

Follistatin

Vascular endothelial growth factor

Prostaglandins

Regulation of the cycle

a Chosen from among the scores of factors isolated from intrafollicu-From a neuroendocrine perspective, the oestrous cycle

lar/extrafollicular sites. They have important extra-ovarian actions but is controlled by the secretory activities of three principal components: peptidergic neurones in the hypothalamus, gonadotropes in the adenohypophysis and steroid-secreting cells in the ovaries. It is clear that ultimate control is exercised by the hypothalamus through characteristic cyclic changes in the pattern of GnRH release, which are reflected in the circulating levels of regulatory factors (Box 33.2), including steroid hormones (principally, oestradiol and progesterone) and the polypeptide inhibin; it also facilitates the maturation of the oocyte – but that is not a topic within the remit of this essay.

reinforced as a result of recent developments in ultrasound technology that have given the clinician visual

In its simplest form, the primordial stage, the follicle

consists of an oocyte surrounded by squamous pre-access to the morphological changes in the female granulosa cells. The resting primordial follicles are genital tract throughout the reproductive life of the thought to be under constant inhibitory influences to cow; it is likely that it will be strengthened by this pre-remain dormant. In due course, either due to a decrease entation, which emphasizes the central importance of in inhibitory influences or to positive stimuli by some the inherent rhythmicity with which cohorts of follicles paracrine or endocrine factors, growth is initiated in a emerge from the pool of resting follicles. In essence, the cohort of primordial follicles. This process of recruit- following text describes how the responses of the HPG ment begins long before puberty and is recurrent axis determine the fate of the oocytes that are brought throughout reproductive life. The fate of a recruited fol- forward every seven to ten days in anticipation of being licle depends on whether or not the mutual interactions chosen to perpetuate the species.

between the component parts of the HPG axis can

shepherd it to ovulation. Those follicles that fall by the wayside on the long path to ovulation undergo atresia; Follicular development and its control during the obviously, that is the fate of all follicles recruited before oestrous cycle

puberty; it is also the fate of most of the follicles

Basic pattern: Each ovary has a large pool of oocytes, recruited after puberty.

each contained within a follicle. The follicle is the basic

In the adult ovary, most of the follicles progress

functional unit of the female gonad; although it spends

slowly through several developmental stages to reach

much of its lifespan in a dormant state, it does have a

the antral stage after four months or so, at which point

brief period of progressive growth and development

the follicle has acquired the histological features

that ends in either ovulation or degeneration (atresia).

depicted in Figure 33.24. There is evidence that follicles

During this active phase it is an essential component

can develop to the preantral stage in the complete

of the hypothalamic–pituitary–gonadal (HPG) axis. It

absence of gonadotrophins; thereafter, they are

Theca externa

Theca interna

Basement membrane

Antrum containing

follicular fluid

Granulosa cells

Fig. 33.24

*The ovarian follicle has four distinct
layers: the theca externa, the highly vascular*

Cumulus oophorus

*theca interna, the basement membrane and
the avascular granulosa cell layer. The base-*

Oocyte

ment membrane prevents direct access of

Zona pellucida

capillaries to the granulosa cell layer.

First wave

Second wave

Ovulatory wave

Loss of

Loss of

Ovulation

dominance

dominance

Dominance

Dominance

Dominance

15

(Static)

(Regressing)

10

Fig. 33.25

A model explaining the terms

(Growing)

associated with each wave of follicular

Emergence

Deviation

development during the oestrous cycle of

Subordinate

heifers. Based on ultrasound analysis,

Diameter (mm)

5

most heifers have one (first wave) or two waves (first wave, second wave) of follicular development during the luteal phase and a single wave of development (ovula-

1

Cohort

Cohort

Cohort

tory wave) during the follicular phase.

Selection

Selection

Selection

(From Ireland et al., 2000, reproduced with

1

12

21

permission of the American Dairy Science

Association.)

gonadotrophin-dependent. FSH can stimulate growth

recorded as the first day on which a 4 mm or 5 mm follicle up to about 9 mm in diameter, while LH follicle is detected by ultrasonography as the largest in a new cohort of growing follicles. Over the next two or three days all of the emergent follicles grow at a similar rate but then there is an abrupt divergence of individual growth rates (‘in the trade’) (Webb et al., 1999; Ireland et al., 2000). The inherent pattern of follicular growth in cattle is to a halt and, soon, is followed by regression (Ginther et al., 2001). About 8 hours before the beginning of ovulation or atresia (Webb et al., 1999; Ireland et al., 2000).

become dominant; in the subordinate follicles it comes to a halt and, soon, is followed by regression (Ginther et al., 2001). About 8 hours before the beginning of

it persists during oestrous cycles, throughout most of deviation, the granulosa cells in the future dominant pregnancy and during the early anoestrus of the post-follicle acquire LH receptors. LH stimulates increased partum period. In practice, the emergence of a wave is production of oestradiol and of insulin-like growth

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16

(a)

FSH (–)

(n = 33 waves)

FSH

14

Follicular

Dominant

factor (s)

12

follicle

10

Subordinate

8

follicle

rowth

Diameter (mm)

g

Continued

6

8 h

Divergence

4

Atresia

0

1

2

3

4

5

6

7

8

Number of days from emergence

Fig. 33.27

Simplified model of deviation. The future dominant follicle emerges approximately 8 hours before the future largest 14

(b)

13

subordinate follicle. Under the influence of FSH the two follicles (n = 19 waves)

FSH concentration (ng/ml)

grow at the same rate so that the larger follicle retains its initial 12

12

advantage up to the point of deviation (marked by asterisk). As FSH

the follicles grow they secrete increasing quantities of oestradiol and inhibin, both of which diminish the secretion of FSH until 10

11

there is insufficient to sustain the growth of subordinate follicles; they regress. However, changes in the dominant follicle, such as 8

10

increased responsiveness to LH and the stimulatory activity of Diameter (mm)

IGF-1, enable it to survive and enlarge in the presence of low 6

9

concentrations of FSH. (After Wiltbank et al., 2000.)

4

8

reduced to what is the 'species-specific ovulatory

-2

-1

0

1

2

3

4

quota': in cattle, usually one follicle, occasionally two.

The beginning of the selection phase cannot be identi-

Number of days from deviation

fied by ultrasonography but the end of the phase is

Fig. 33.26

Deviation. The patterns of growth in the dominant

coincident with the onset of dominance, which can be

follicle and the largest subordinate follicle from the day of emer-identified.

gence. (a) The growth rates gradually diverge. (b) Before devi-Normally, each wave has a span of seven to ten days

ation, the growth rates did not differ significantly. The

from emergence to ovulation or to the onset of atresia

concentration of FSH reached a nadir on the day of deviation.

in the dominant follicle. The dominant follicle has

(Reproduced from Ginther et al., 1996 with permission of the the functional capacity to trigger the neuroendocrine

Society for the Study of Reproduction.)

cascade that culminates in ovulation but its fate is determined by the frequency of pulsatile LH secretion at the factor 1 (IGF-1). Systemically, the high levels of oestradiol and inhibin released by the dominant follicle suppress release of FSH from the pituitary gland; there is enables it to ovulate; the low frequency during the inadequate concentration of FSH to sustain the smaller luteal phase or during postpartum anoestrus results in follicles and they become atretic (Figs 33.26 and 33.27). atresia; an intermediate frequency (e.g. when there is Within the dominant follicle, oestradiol and IGF-1 suboptimal concentration of progesterone) is associated with extended dominance, which may have a deleterious effect on the competence of the oocyte. The next

the low concentration that contributed to the onset of wave cannot emerge until after the dominant follicle atresia in the subordinate follicles. If the largest follicle has begun to regress.

is ablated before deviation, the next largest follicle will proceed to dominance. Thus, the follicular wave has two

Cyclic pattern: Usually, the bovine oestrous cycle has phases (Fig. 33.28): the growth phase (initiated by FSH)

either two waves (modal days of emergence: days 0, 10) and the dominance phase (initiated when FSH is close or three waves (modal days of emergence: days 0, 10, to its nadir; LH dependent).

16) of follicular growth, each emerging about a day

Dominance is a consequence of selection, the process after a transient peak in serum concentrations of

that results in the number of growing follicles being

FSH. During the luteal phase both progesterone from

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Growth phase

Dominance

which the new dominant follicle goes on to ovulate

phase

approximately three days later (Fig. 33.28), by which

(a)

3.2 Oestradiol concentration (pg/ml)

time it has grown to 16–20 mm in diameter.

2.8

Dominant

10

follicle

Largest

2.4

subordinate

The phases of the oestrous cycle

follicle

2.0

8

Follicular phase: Each of the growing follicles has two 1.6

populations of steroid-secreting cells: theca interna cells

1.2

6

and granulosa cells (Fig. 33.29a). The cells of the theca

Diameter (mm)

Oestradiol

0.8

interna have specific receptors for LH and they respond

0.4

to this gonadotrophin by synthesizing androgens

4

0.0

*(androstenedione and testosterone) that diffuse across
the basement membrane into the granulosa cell layer.*

0.7

1.1

During the early stages of folliculogenesis the granulosa

(b)

1.0 LH concentration (ng/ml)

cells have receptors for FSH and they respond to this

0.6

gonadotrophin by converting the thecal androgens to

LH

0.9

oestrogen (Fig. 33.29b). As the follicles grow under the

0.5

0.8

trophic influences of both FSH and oestrogen, the gran-

0.7

ulosa cells acquire increased numbers of FSH receptors

0.4

FSH

0.6

and oestrogen receptors and they secrete increasing quantities of oestrogen-rich follicular fluid (Fig. 33.29c).

0.5

0.3

Follicular oestrogen passes into the circulation and

0.4

FSH concentration (ng/ml)

Deviation

exerts negative feedback on the release of FSH from the

0.2

0.3

pituitary gland: while the concentration of FSH is in

–48 –40 –32 –24 –16 –8

0

8

16 24 32

decline, the dominant follicle is selected and the subor-

Number of hours from follicle deviation

dinate follicles regress (Figs 33.26, 33.27). The dominant

follicle maintains FSH at low concentrations and

Fig. 33.28

The follicular wave has a growth phase and a

becomes dependent on LH for continued growth and

dominance phase. (a) Diameters of the dominant follicle and of secretion of oestradiol (Fig. 33.30). LH stimulates the

the largest subordinate follicle before and after deviation; also, secretion of sufficient oestradiol to induce oestrus and to

the contemporary concentrations of circulating oestradiol. (b) evoke the oestrogen surge that exerts a positive feedback

The contemporary concentrations of circulating FSH and LH.

on the hypothalamic–pituitary axis (Figs 33.30, 33.31).

(Reproduced from Kulick et al., 1999 with permission from Elsevier Science.)

The frequency of the GnRH pulse generator increases,

the sensitivity of the adenohypophysis to GnRH is

increased by a self-priming action of the GnRH and the

the corpus luteum and oestrogen from the cohort of
pituitary gland releases the preovulatory surge of LH.
growing follicles (>5 mm) restrain the hypothalamic–
The response of the dominant follicle to LH is both mor-
pituitary axis by negative feedback. The frequency
phological (growth, ovulation, formation of the corpus
of the pulse generator is reduced to one pulse of
luteum) and secretory (oestrogen, progesterone).
GnRH every 4–8 hours and the concentrations of
Ovulation is critically dependent on the timing, fre-
gonadotrophins in the peripheral circulation are insuf-
ficiency, and amplitude of the hormonal changes just
ficient to induce ovulation. As a result, neither of the
described. For instance, an inappropriate pattern of LH
dominant follicles in the first two waves can ovulate;
secretion could lead to atresia or to undue persistence
all of the follicles in the two waves undergo atresia.
of the dominant follicle (with detrimental effects on the
Luteolysis, which is required to remove this inhibi-
quality of the oocyte). The crucial hormonal event that
tion, occurs spontaneously when the uterus, under the

initiates the ovulatory process is the switch from negative influence of progesterone followed by oestrogen to positive feedback by progesterone and oestrogen to positive feedback by oestrogen (Figs. 33.30, 33.31); again, (PGF_{2a}) to kill the corpus luteum (see p. 495). The timing and rate of change of the oestrogen surge are resultant fall in progesterone concentrations releases critical: any significant deviation in either feature can the GnRH pulse generator, which increases its frequency to one pulse every hour (approximately). The result in delayed ovulation or anovulation.

concentrations of FSH and LH rise in the peripheral Luteal phase: After ovulation, the basement membrane circulation, stimulating the cohort of follicles that initiates the follicular phase of the cycle and from within between the theca cells and the granulosa cells breaks down and the avascular granulosa cell layer is invaded

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(a)

Basement

(b)

membrane

Theca

Oestrogen

LH

interna

Granulosa

cells

Androgen

LH receptors

FSH

FSH receptors

(c)

LH

FSH

LHR

FSHR

Theca

Granulosa

cells

cells

+

+

+

5

+

+

FSHR

ER

Mitosis

IGF-

Aromatase

1

+

+

+

+

+

+

6

6

Androgens

2

4

4

3

5

Oestrogens

Fig. 33.29

Formation of oestrogen by ovarian follicles: (a) depicts the structure of the follicular wall; (b) illustrates the essential features of the production of oestrogen: LH stimulates the synthesis of androgens by the theca cells, FSH stimulates aromatases within the granulosa cells to convert the androgens to oestrogens; (c) depicts the biochemical events in the growing follicle between emergence and divergence: (1) LH stimulates theca cells to produce androgens; (2) FSH activates aromatase in granulosa cells to convert the androgens to oestrogen; In the granulosa cells, oestradiol (3) stimulates mitosis and (4) up-regulates receptors for FSH (FSHR) and for oestradiol (ER); (5) synthesis and secretion of IGF-1 by granulosa cells are stimulated by oestradiol and FSH; (6) IGF-1 further stimulates mitosis and aromatase activity in granulosa cells. Outcome: growth of the follicle and expansion of the antrum due to the combination of increased numbers of secretory granulosa cells with increased numbers of FSH and oestrogen receptors per cell.

by connective tissue septae that carry theca cells and specifically, serum progesterone at luteal phase concentrations. The granulosa cells and the theca cells differentiate for a few days before it subsides, followed by serum FSH at normal preovulatory concentrations.

they can also synthesize and secrete oestrogen, oxytocin
Failure of this sequence or inappropriate serum concentrations of either hormone seem to affect the life-span of the corpus luteum (short luteal phase, prolonged support the very high metabolic rate of the mature luteal phase) and/or its capacity to secrete progesterone corpus luteum; so rich does the vasculature become that (inadequate luteal phase), phenomena that are endothelial cells comprise more than half of the common after the first ovulation at puberty or after constituent cells – in due course, these cells play a pivotal calving. It is not known how the (prefollicular phase) role in the process of luteolysis (see below).

progesterone exerts its influence on the activities of the Luteinizing hormone is the principal luteotrophin in next corpus luteum; it is possible that the FSH could do the cow; under normal circumstances, tonic secretion of so through its role in the induction of LH receptors on LH maintains the functional corpus luteum until PGF2a

the granulosa cells of the preovulatory follicle.

from the uterus causes luteolysis at the end of the cycle.

The corpus luteum grows progressively in size until

It is important to realize that ‘normal circumstances’

days 16–18 of the oestrous cycle. Histologically, the

include the correct antecedent hormonal pattern:

mature corpus luteum is seen to contain two mor-

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LH surge

Ovulation

+

FSH surge

9

+

8

Low FSH

Positive

feedback

Oestrogen surge

FSH

3

6

LHR

—

—

R

LH

4

5 Aromatase

LH R

7

IGF-

+

5

+

1

+

3

Negative

+

Mitosis

5

feedback

RE

E2

E

R

2

FSH

FSH

Oestradiol (E2)

R

2

FSH

Oestradiol (E

R

2)

Inhibin

FSH

R

Inhibin

Fig. 33.30

Dominant follicle becomes the ovulatory follicle. When luteolysis occurs, the LH

pulses attain the frequency that is required to progress the dominant follicle to ovulation. FSH induces mitosis and stimulates aromatase activity in granulosa cells and they secrete oestradiol and inhibin: within the follicle, oestradiol binds to receptors (ER) in granulosa cells in which it (1) up-regulates ER and receptors of FSH(FSHR), stimulates aromatase activity, mitosis and secretion of IGF-1: systemically, both oestradiol and inhibin depress FSH

secretion by the pituitary gland (negative feedback). (2) Granulosa cells have high number FSHR [see (4) in Figure 30.29]. (3) The low levels of FSH continue to induce mitosis and to stimulate aromatase activity in granulosa cells (thus producing oestradiol and enlarging the antrum). FSH (4) and oestradiol (1) stimulate granulosa cells to secrete more IGF-1 – which, in turn, (5) enhances the steroidogenic and mitotic actions of FSH and oestradiol on granulosa cells, and enhances the steroidogenic action of LH on theca cells. FSH

(6) increases the number of LH receptors (LHR) on granulosa cell. The cell division and the secretory activity accelerate the expansion of the dominant follicle: it may be 16 to 18 mm in diameter at the preovulatory stage. The rapid increase in secretion of oestrogen induced by the activities of LH, FSH and IGF-1 leads to a surge of oestrogen (7) which evokes, by positive feedback (8), coincident surges of the gonadotrophins (large surge of LH, smaller surge of FSH) and, consequently, ovulation (9).

phologically distinct populations of parenchymal cells:

to the recognized luteotrophin (LH) and luteolysin

small luteal cells (10–20 mm) and large luteal cells

(PGF_{2a}). Receptors for LH are numerous on the small

(20–35 mm). All of the small cells are derived from

cells but they are scanty or absent on the large cells.

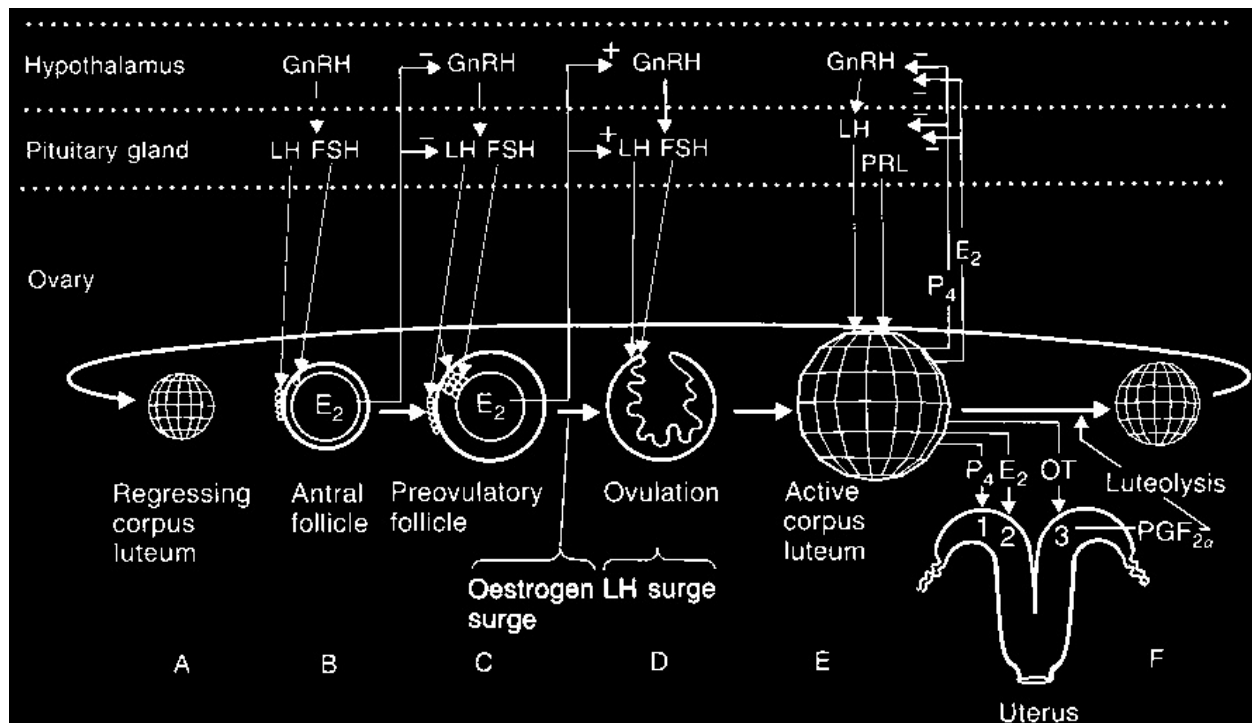
thecal cells and, until the sixth day of the oestrous cycle,

Therefore, LH stimulates the small cells to secrete prog-

nearly all of the large cells are derived from the granu-

esterone but it has no such effect on the large cells.
losa cells. After day six, some small luteal cells differ-
Despite this and the numerical predominance of small
entiate into large luteal cells, so that the large cells are
luteal cells throughout the luteal phase, it is generally
of mixed origin and the proportion of large cells
agreed that most of the progesterone is secreted by the
increases as the corpus luteum matures (from less than
large luteal cells. Progesterone is secreted as pulses at
2 per cent at days 3 to 5 to almost 5 per cent at days 10
a frequency that is different from that for LH. This does
to 18).

not diminish the importance of LH as the principal
It is significant for the clinical management of the
luteotrophin in the cow: LH does not have direct
oestrous cycle to realize that the small luteal cells and
control over the quantities of progesterone secreted by
the large luteal cells differ in their abilities to respond
the large cells but it does exert an important indirect



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Fig. 33.31

Interrelationships between the hypothalamus, pituitary gland, ovary and uterus during the oestrous cycle. When the corpus luteum (CL) regresses at the end of a cycle (A), the hypothalamus and pituitary gland are released from the strong negative feedback exerted by progesterone (P_4) throughout the luteal phase of the cycle. FSH and LH stimulate the growth and secretory activity of a dominant ovarian follicle (B) that secretes increasing quantities of oestradiol (E_2). The initial low concentrations of E_2 have a negative feedback effect until the preovulatory follicle produces a surge of oestrogen (C) that exerts a positive feedback and results in a surge of LH that causes ovulation (D). A new CL develops under the trophic influence of both LH and, perhaps, prolactin (PRL). It secretes P_4 and E_2 that reassert a strong negative feedback effect on the hypothalamic–pituitary axis. In addition, the P_4 causes an accumulation of fatty acid precursors in the endometrium (1). After day 10, E_2 induces the synthesis of prostaglandin from the stored precursors (2). Finally, oxytocin (OT) causes the release of the $PGF_{2\alpha}$ (3), which is transferred from the uterus to the ipsilateral ovary by a countercurrent mechanism (see Fig. 33.32) and induces luteolysis (F). (See Fig. 33.23 for changes in concentrations of the hormones in the peripheral plasma.)

control by regulating the differentiation of small cells transported in the venous blood to the lungs where into large luteal cells, a process during which the approximately 65 per cent of it is degraded into its inactive metabolite, PGFM, in one passage. However, some on fully and permanently. On the other hand, receptors PGF2a is brought directly from the uterus to the adjacent ovary by means of a counter current mechanism absent from the small cells. Since the secretion of (Fig. 33.32) that transfers the luteolytic agent from the progesterone by the young corpus luteum (days 3 to 5) uterine vein to the ovarian artery. Thus, in the cow, is invested almost entirely in the small cells, it is not surprising that it is refractory to the luteolytic action of the local counter-current transfer (McCracken et al., 1999).

luteal cells assume responsibility for most of the secre-

*Prostaglandin F2a is released from the uterus in
tory activity.*

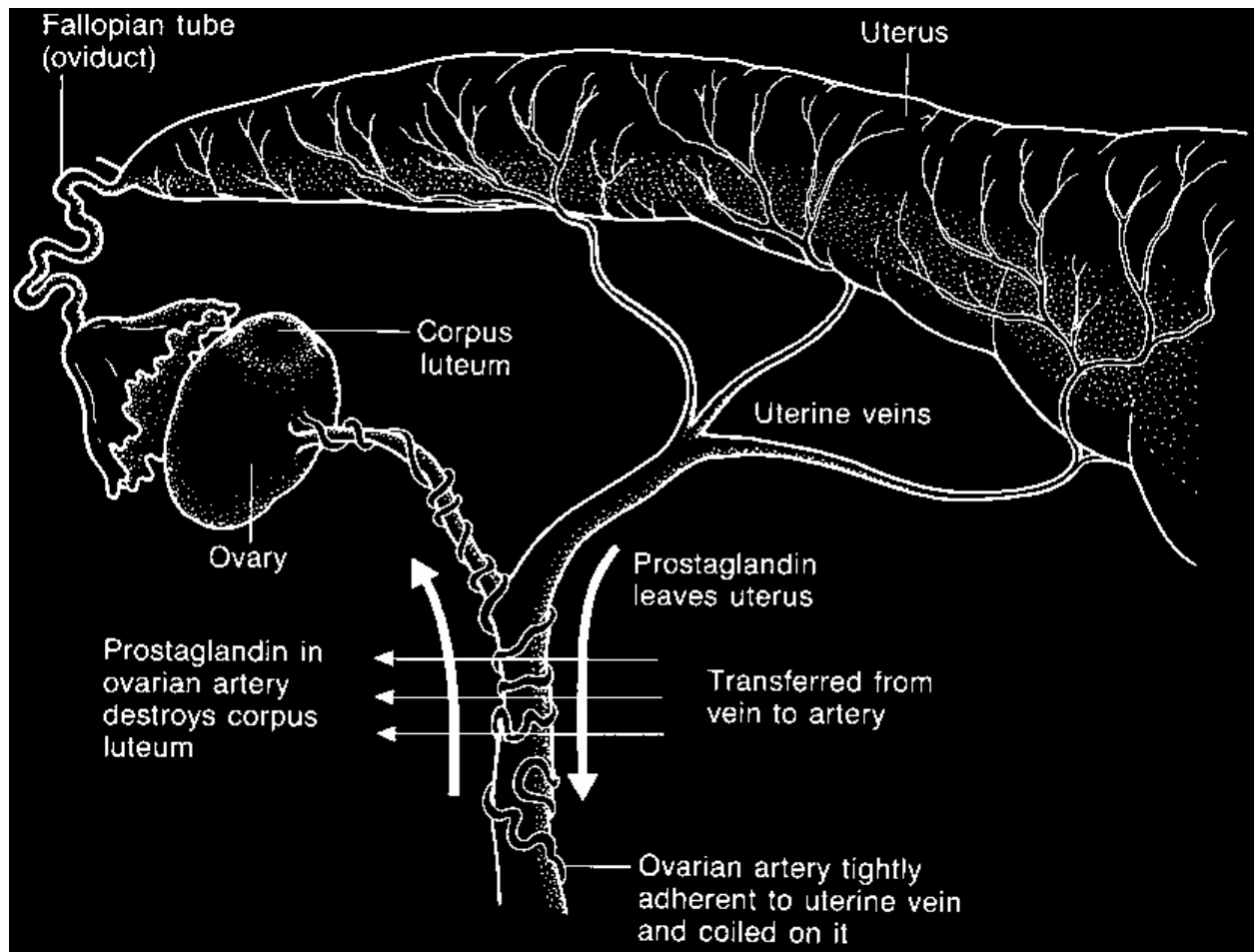
pulses and there is evidence that the minimum

*It is well known that during the first four or six days
requirement for normal regression of the corpus luteum
of the oestrous cycle exogenous oxytocin can inhibit the
at the end of the oestrous cycle (i.e. effective luteolysis
growth of the corpus luteum and block its secretory
by endogenous PGF2a) is a pulse lasting one hour
activity. This is possible because the small luteal cells
repeated about every six hours over a period of 24–30
have specific receptors for oxytocin and interaction
hours. (By contrast, after day 5 of the oestrous cycle the
between the receptors and the peptide leads to inhibi-
corpus luteum can be removed by a single injection of
tion of the LH-stimulated secretion of progesterone by
exogenous luteolysin, in an appropriate dose.)
the small cells. In the mature corpus luteum the large
The timing and magnitude of PGF2a release from the
luteal cells synthesize oxytocin and it has been sug-
uterus is determined by the sequential interactions of*

gested that this peptide may play a significant role in progesterone, oestradiol and oxytocin. The central and local 'large-cell-to-small-cell communication' during peripheral actions of oestradiol are critical for luteolysis-prostaglandin-induced luteolysis.

sis to occur (Binelli et al., 2001). During the early part Prostaglandin F2a is formed in the endometrium. It

of the oestrous cycle the endometrial cells have many leaves the uterus in the uterine vein and most of it is receptors for progesterone and coupling of the steroid



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Fig. 33.32

The transfer of PGF2a from the uterus to the ovary by a counter current mechanism.

*with its receptors effectively blocks the production of
tion of progesterone by the small luteal cells. Oxytocin,
PGF2a; however, it causes an accumulation of fatty acid
which is formed in the large luteal cells and has specific*

precursors in the endometrium. After about 10 days the receptors on the small luteal cells, is an obvious candidate messenger. Recent studies (Meiden et al., 1999) the endometrium begins to decline (possibly due to assign the pivotal role to endothelin 1 (ET-1), a peptide progesterone-induced loss of its own receptor: 'down synthesized and released by the resident endothelial regulation'), permitting oestrogen to effect changes cells. The proposal is that PGF2a elicits the release of both centrally and peripherally. Centrally, oestradiol ET-1 from the endothelial cells and of oxytocin from from developing ovarian follicles appears to alter the the large luteal cells; ET-1 acts on the steroidogenic frequency of the hypothalamic oxytocin pulse generator cells to reduce their progesterone output; both PGF2a tor, which produces a series of intermittent episodes of and ET-1 induce vasoconstriction and consequent oxytocin secretion; peripherally, it promotes the formation of hypoxic conditions; ET-1 secretion is enhanced both by action of endometrial receptors for oestradiol and oxy-

hypoxia and by oxytocin (Fig. 33.33). For structural
tocin. In the endometrium, coupling of oestradiol with
regression it is envisaged that ET-1 initiates a cascade
its receptors increases the synthesis of PGF2a from the
of events leading to recruitment and activation of
fatty acid precursors, while coupling of oxytocin (from
phagocytic cells that release cytokines responsible for
the hypothalamus and from the corpus luteum) with its
programmed cell death.

receptors leads to immediate pulsatile secretion of

It is obvious that the functional corpus luteum plays

PGF2a (McCracken et al., 1999). It is probable that the a primary role in the
control of the oestrous cycle, a fact

duration of the pulses is determined by the rapid down

that is central to all regimes that aim to control the

regulation of oxytocin receptors (within one hour) and

cycle. In the past, the practice was to remove it manu-

that the frequency of the pulses reflects the regenera-

ally per rectum, a procedure to be condemned. The

tion time of the oxytocin receptors in response to

mature corpus luteum (CL) is a very vascular organ;

oestradiol (six hours).

manual expression causes haemorrhage which will give PGF2a causes a significant reduction in luteal blood rise to local adhesions, if it is not fatal. The CL can be flow and it couples with specific receptors on the large removed by administration of exogenous luteolytic luteal cells. However, the other steroidogenic cells, the agents. It is insensitive to exogenous PGF2a during the small luteal cells, do not have receptors for PGF2a and first four days of the cycle, but between day 5 and day it has to be assumed that some type of intercellular 16 the administration of PGF2a or an analogue will communication between the large cells and the small cause rapid luteolysis followed, within a few days, by cells is involved in terminating the LH-induced secre-oestrus and ovulation.

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Messenger

Mechanisms by which luteal secretions are altered

Large luteal cells

Progesterone decreased

Oxytocin increased

PGF2

Arterioles: vasoconstriction

Progesterone decreased

(hypoxia)

+

Luteal cells

Progesterone decreased

+

+

Arterioles: vasoconstriction

Progesterone decreased

ET-1

(hypoxia)

Oxytocin increased

+

+

Luteal cells

Progesterone decreased

Fig. 33.33

Schematic representation of the

putative role of endothelin-1 (ET-1) in func-

Oxytocin

Arterioles: vasoconstriction

Progesterone decreased

+

(hypoxia)

ET-1 secretion increased

tional luteolysis in cattle.

Pharmacological regulation of the oestrous cycle

due recognition of these observations. A single bolus of

(see also Chapter 42)

exogenous GnRH is likely to be effective only when a

preovulatory follicle is present already.

The pharmaceutical industry has provided an array

During the luteal phase of the oestrous cycle the

of natural and synthetic hormones – GnRH, gonado-

corpus luteum acts as an effective ‘brake’ on the pulse

trophins, steroids, prostaglandins – that can be used to

generator: pulse frequency is reduced so that circulat-

regulate the oestrous cycle. The use of these agents for

ing gonadotrophins are maintained at tonic concen-

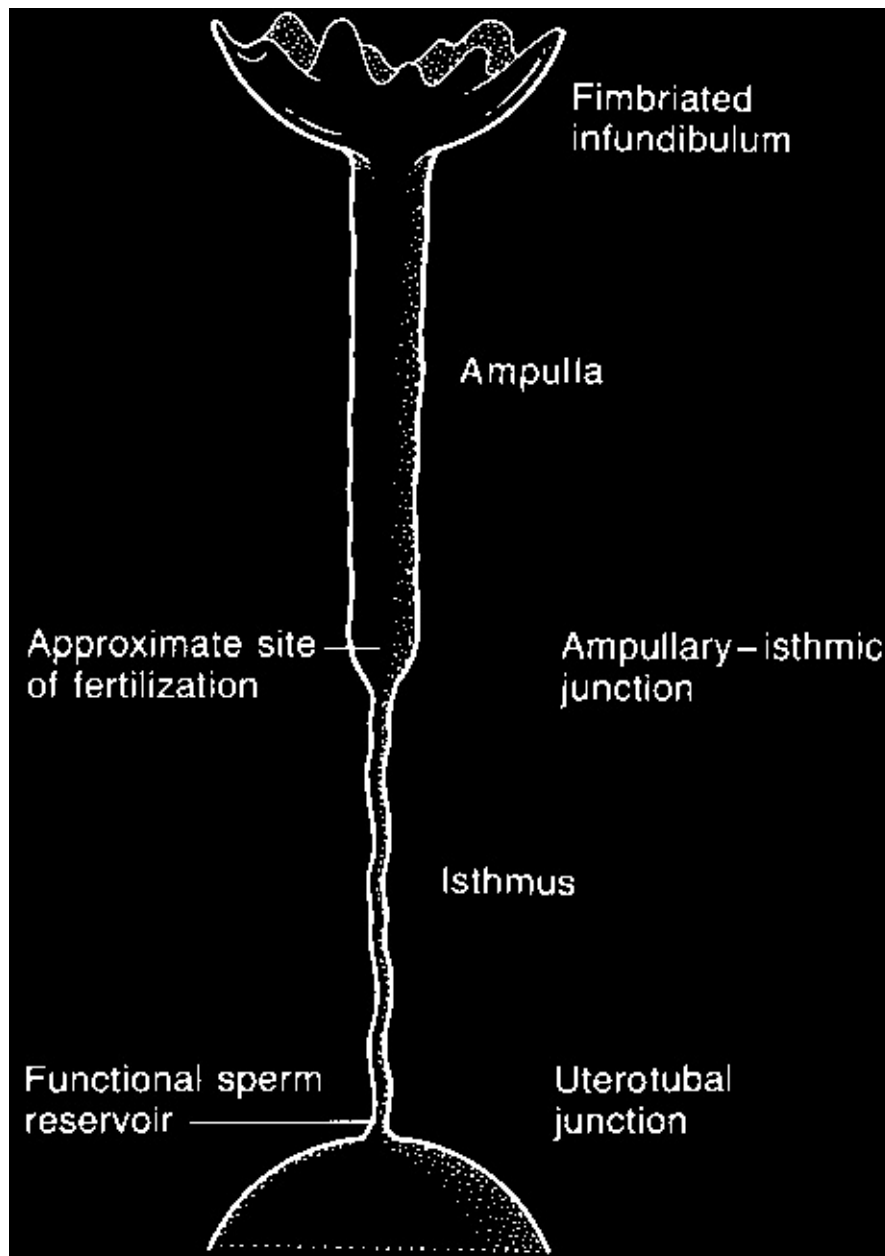
specific purposes will be described when we deal with
trations and the dynamic surges required to cause
the clinical conditions in which they have been found
ovulation cannot occur. Luteolysis removes the brake,
to be effective. Nevertheless, it is appropriate at this
the pulse frequency increases and ovulation occurs
juncture to emphasize a few basic principles that should
within four or five days. Hence, there are two methods
be borne in mind when the clinician is contemplating
by which control over the length of the cycle and
ing the use of reproductive hormones for therapeutic
ovulation can be achieved: the use of a luteolytic agent
purposes.

to kill the corpus luteum of the current cycle or the use
The GnRH generator imposes a pulsatile mode of
of a progestogen to create an 'artificial' luteal phase,
secretion on the hypothalamic–pituitary–ovarian axis.
which will be followed by ovulation shortly after with-
The component parts of the axis respond to hormonal
drawal of the progestogen. These methods can be used
signals that are encoded in the frequency and, to a lesser

separately or in combination. Since prostaglandin does extent, the amplitude of the pulses. However, these not kill the very young corpus luteum (less than five target tissues may not be able to decode a hormonal days after ovulation), the usual recommendation is to stimulus that is either non-pulsatile or pulsatile at non-give two injections 11 days apart (see p. 682).

physiological frequencies. As an example, repeated To date, neither of these methods has fully satisfied small pulses of exogenous GnRH will induce pulsatile the requirements of the industry. Ideally, synchroniza-releases of gonadotrophins and these responses will be tion of oestrus should make it possible to achieve high enhanced by the self-priming action of GnRH. On the rates of pregnancy to a single insemination at a pre-other hand, continuous infusion of GnRH or intermit-dictable interval after treatment, without the need for tent infusions of GnRH at markedly non-physiological detection of oestrus. Recent studies of the dynamics of frequencies will soon lead to complete refractoriness of follicular waves have revealed that the prime require-

*the pituitary gland to the continued stimulation (see
ment is 'to have a recently selected dominant follicle of
p. 477). Therapeutic regimes should be planned with
short duration dominance at the end of a progestagen*



or PGF2a treatment regimen' (Roche et al., 1999). Thus, the objective of current research efforts is to validate a

regimen that will regulate both the luteal and follicular components of the periovulatory phase.

As mentioned, the response of the corpus luteum to an exogenous luteolysin depends on the stage of its development, and even when it is fully responsive the intervals from treatment to oestrus and ovulation can vary by some days depending on the stage of development of the dominant follicle at the time of treatment. For instance, if the current largest follicle is no longer functionally dominant, clinical response will be delayed until the dominant follicle of the next wave does the business up to a week later. Scientists have shown that it is possible to manipulate follicular waves to deliver ovulation within a time span that would satisfy the level of predictability required to make fixed-time insemination a viable proposition. However, the problem remains that some of the procedures that have been employed under 'laboratory conditions' do not lend themselves to ready application under field conditions.

Fig. 33.34

Schematic diagram of the uterine tube (oviduct,

One such approach involved transvaginal ultrasound-guided ablation of all follicles greater than 5 mm (day 0) followed by PGF2a (day 4) and LH or GnRH (day 5):

all of 23 treated heifers ovulated within the same 24-hour period, 19 within the same 12-hour period

(Bo et al., 2002). Presumably, the next task is to replace (oestrogens and progesterone) released in the correct

surgical ablation by a pharmacological regimen that will

concentrations and in the proper sequence. Therapeutically consign the current cohort of emerging

tic procedures that significantly alter the relative concentrations of follicles to immediate atresia, permitting the emergence of a new cohort, one of which then can be driven

the transport mechanisms, which in some instances may

by exogenous hormones to ovulation within a predictable time span. It is known that the physiological

cause the loss of gametes or of the conceptus.

dictable time span. It is known that the physiological

cause the loss of gametes or of the conceptus.

dictable time span. It is known that the physiological

cause the loss of gametes or of the conceptus.

dictable time span. It is known that the physiological

cause the loss of gametes or of the conceptus.

dictable time span. It is known that the physiological

The transport of spermatozoa is not an entirely passive process: the motility of the male gametes makes molecules produced locally within the follicle (growth factors, activins, inhibins, cytokines etc.: Box 33.2). The of the hazardous voyage from vagina to oviduct during paracrine or autocrine actions of those molecules which the vast majority of the spermatozoa are lost, may hold the keys to precise manipulation of ovarian partly as a result of phagocytosis but largely by expulsion to the exterior in cervical mucus. It appears from the literature that two distinct populations of spermatozoa arrive in the oviduct:

function.

Pregnancy

- *Those that are transported rapidly but are incapable of fertilizing the ovum; and*

Transport of gametes in the female genital tract

- *Those that are transported more slowly to the function*

Fertilization takes place in the oviduct, close to the

tional sperm reservoir, are viable and are capable of undergoing capacitation and the acrosome reaction. Transport of the ovum from the ovary is achieved by muscular and ciliary activity and flow of fluids in the oviduct. After natural mating, spermatozoa have to traverse the cervix, the uterine horn and uterus to reach the ampulla of the oviduct. Spermatozoa have been recovered from the ampulla within 3–5 minutes after mating. This activity and flow of fluids are largely responsible for the rapid transport of the male gametes. The appropriate muscular activities, secretion and flow of fluids in the female genital tract are regulated by the ovarian hormones. During oestrus, the contractions begin at the cervix and move towards the oviduct; at the end of oestrus the direction of the contractions is reversed.

reversed. Most of the spermatozoa that reach the
The acrosome reaction (Fig. 33.20) involves multiple
oviduct during the initial rapid transport phase are
point fusions between the plasma membrane and the
damaged or dead and they do not include the sperm
outer acrosomal membrane over the front half of the
that fertilizes the ovum. It has been suggested that these
sperm head. The fusions give rise to a series of small
non-viable spermatozoa may play a significant physio-
vesicles formed by fragments of both membranes. The
logical role, by releasing products that elicit local mus-
vesicles are separated by small pores through which the
cular and secretory responses that facilitate transport
acrosomal enzymes escape. There is controversy as to
and sustenance of the gametes.

whether or not the escaping enzymes aid the passage of
According to Hunter and Wilmut (1984), viable
the spermatozoa through the cumulus oophorus in the
spermatozoa are transported more slowly: heifers
cow. As the reaction proceeds, the pores enlarge at the
mated early in oestrus took 8–12 hours to establish an

expense of the vesicles so that the vesicles have been adequate population of viable spermatozoa in the lost by the time the sperm penetrates the zona pellucida. Then the gametes were sequestered, in a relatively quiescent state, in the caudal 2 cm of the isthmus reaches the perivitelline space, it fuses with the plasma for a further 18–20 hours before they began a progressive migration towards the site of fertilization. Thus, the incorporated into the conceptus at fertilization.

caudal isthmus served as the functional sperm reservoir, The fusion of the egg and the sperm triggers a series the immediate source of viable spermatozoa at the time of reactions that prevent polyspermy by making both of ovulation. It is known that both the temperature and the zona pellucida and the plasma membrane of the the oxygen tension are lower in that segment of the ovum impenetrable to other spermatozoa.

isthmus than they are in the ampulla and it is thought It is often stated that capacitation requires a 6-hour

that these factors may be responsible for the depressed sojourn in the female tract. However, it is unlikely that motility of the spermatozoa in the reservoir up to the the processes would be synchronized to ensure that all time of ovulation. At ovulation, the temperature and the surviving spermatozoa would have been capacitated oxygen tension are elevated in the lumen of the caudal at a fixed minimum time after deposition in the female isthmus, the sequestered spermatozoa begin to exhibit tract. Asynchrony in the time to capacitation would activated motility (hyperactivation) and they migrate to appear to be more advantageous in that it would allow the site of fertilization. Hyperactivation is in response for small populations of potential fertilizing spermatozoa to the 'pick-up' of follicular fluid and the ovum by the zoa to be available sequentially over a period spanning oviduct.

several hours before and after ovulation. Indeed, there is evidence that only 10–20 per cent of bovine spermatozoa have undergone capacitation and the acrosome

Capacitation and the acrosome reaction

reaction within three hours of insemination, while other
The spermatozoa deposited in the female reproductive
spermatozoa do so several hours later. Again, the
tract are not immediately capable of fertilizing an
inappropriate use of exogenous hormones may hamper
ovum. During transport through the tract they undergo
capacitation and the acrosome reaction.

physiological changes that make them competent to
penetrate the zona pellucida and fuse with the ovum,

Maternal recognition of pregnancy

i.e. they undergo capacitation. It is thought that during
capacitation the spermatozoon loses some proteins it

Normally, the conceptus enters the uterus about 72
has acquired, by adsorption or incorporation into the
hours after ovulation, at the 8–16 cells stage. The cells
plasma membrane, during exposure to epididymal fluid
(blastomeres) form a solid mass (morula) still con-
or seminal plasma. This does not cause any visible
tained within the zona pellucida. By day 7 or 8, fluid
change in the morphology of the spermatozoon but it
secreted by the blastomeres accumulates in a central

does alter the physical and chemical properties of the cavity (blastocoele) and the zygote is now called a plasma membrane, permitting an influx of calcium ions blastocyst. The cells have been arranged into two distinct populations (Fig. 33.35):

of motility and for the acrosome reaction. The hyper-

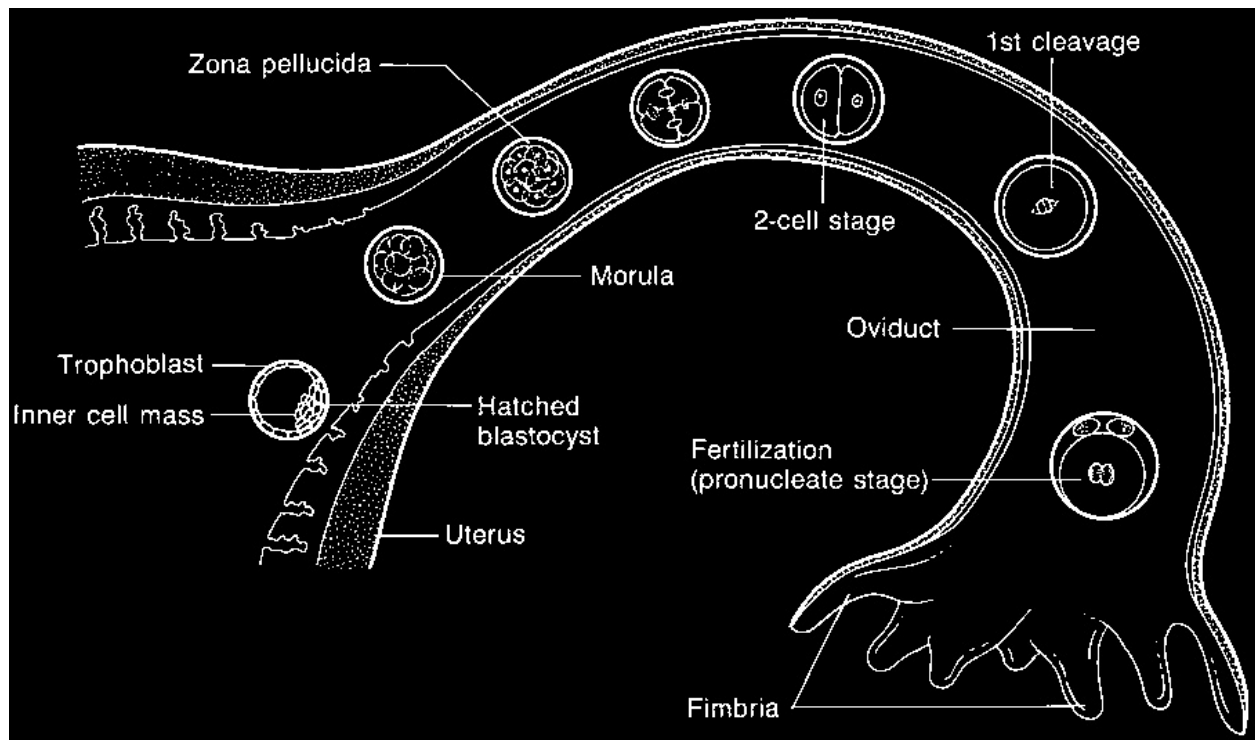
- The flattened trophoblast cells that form the wall of activated motility endows the capacitated spermatozoa the blastocyst and ultimately will form the chorion.

with strong thrusting power that facilitates migration

- A group of cells that forms the inner cell mass at from the isthmus to the ampulla and subsequent one pole beneath the trophoblast cells and is designed to pass through the zona pellucida. The acrosome reaction is an essential prerequisite for fertilization – a spermatozoon with an intact acrosome cannot penetrate the zona pellucida. The blastocyst hatches from the zona pellucida on day 9 to 11 and on day 13 it begins to elongate so that

tion is an essential prerequisite for fertilization – a spermatozoon with an intact acrosome cannot penetrate the zona pellucida. The blastocyst hatches from the zona pellucida on day 9 to 11 and on day 13 it begins to elongate so that

ovum.



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Fig. 33.35

Schematic diagram to illustrate the changes in the conceptus during transport down the uterine tube and into the uterine lumen (after McLaren, 1984).

during the third week it occupies most of the pregnant endometrium. The size of the blastodermic vesicle may (ipsilateral) horn and a portion of the non-pregnant be critical: a small conceptus may not secrete sufficient (contralateral) horn. The process of attachment begins

IFN-t to prevent luteolysis. Putative luteotrophic at day 19 or 20 when there are definite areas of adhe- factors include a lipid-like substance released by the sion between the trophoblastic epithelium of the con- ceptus between day 13 and day 18 of pregnancy and, ceptus and the endometrial epithelium; implantation is possibly, PGE2 from the endometrium.

completed between days 35 and 42. The conceptus plays Later in this text we shall pay serious attention to an important role in prolonging the functional lifespan the effects of nutritional status on the resumption of of the corpus luteum, an essential requirement for the normal ovarian and uterine functions after calving; maintenance of pregnancy. The critical period for the therefore, it seems appropriate to mention at this junc- extension of the lifespan of the corpus luteum in ture that the survival of the early conceptus may be put the cow is between day 15 and day 17; if there is an at jeopardy when the dam is fed excess protein in the elongated conceptus present at that time, the mother diet. The resultant production of large quantities of

will 'recognize' that she is pregnant and the luteolytic ammonia in the rumen leads to elevated concentrations pulsatile pattern of PGF2a release from the of urea in the blood which, in turn, so alters the ionic endometrium will be attenuated or abolished. composition of uterine and tubal secretions that the pH Since maternal recognition of pregnancy precedes is incompatible with the survival of the conceptus. Prob- the physical attachment of the conceptus to the lems of embryonic loss are dealt with in Chapter 36. endometrium, it is evident that the utero-ovarian regulatory mechanisms that 'rescue' the corpus luteum are Maintenance of pregnancy responding to a biochemical dialogue between the conceptus and the maternal tissue that is initiated by a Once the normal conceptus–endometrial–ovarian variety of signal factors released into the uterine lumen regulatory axis of pregnancy has been established, the by the preimplantation conceptus. uterus comes under long-term control by progesterone.

The bovine conceptus can synthesize a number of

In the cow, a fully functional corpus luteum is essential

products (steroids, prostaglandins, peptides, proteins)

to maintain pregnancy up to day 235; thereafter, the

that may interact with the maternal utero-ovarian axis.

adrenal glands seem to be able to secrete sufficient

There is evidence to suggest that the conceptus releases

progesterone to maintain pregnancy in ovariectomized

different signal factors at different times and that the

cows. The placenta contributes little to the circulating

relative importance of each of these factors may change

concentrations of progesterone at any time during

during these early days of pregnancy. A hypothesis

pregnancy. The principal luteotrophin is LH; there is

consistent with current information is that the bovine

little evidence that prolactin plays any significant role

conceptus initiates both antiluteolytic and luteotrophic

(McCracken et al., 1999). The corpus luteum maintains

activities. The main signal is the secretion of interferon-

the plasma progesterone concentration above 10 ng/ml

tau (IFN-t) which reduces the secretion of PGF2a by the

from the second week of gestation until term. Proges-

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terone exercises a 'block' on the uterine muscle in that

release increased amounts of cortisol. The cortisol

it depresses the amplitude of contractions, suppresses

induces the placenta to release increased quantities of

the reactivity to oxytocin and PGF2a and prevents the

oestrogens into the maternal circulation, and both the

development of synchronous coordinated contractions

cortisol and the oestrogens act on the endometrium to

that might expel the fetus prematurely. It does not

increase the output of PGF2a, which causes luteolysis.

abolish the spontaneous contractility of the myometrial

Thus the oestrogen:progesterone ratio has been

cells. Problems of fetal loss are dealt with in Chapter

switched strongly in favour of oestrogen and the

37.

inhibitory effect of progesterone on the uterine muscle

has been abolished. The oestrogens promote uterine

contractility by stimulating the synthesis of contractile

Parturition

protein and of receptors for both oxytocin and PGF2a, Since parturition depends on coordinated rhythmic and by facilitating the formation of gap junctions contractions of the myometrium as well as involuntary between adjacent myometrial cells. Gap junctions are contractions of the abdominal muscles and 'softening' intercellular structures that link cells and allow them to of the birth canal, the final preparation for delivery of exchange ions and electrical impulses. In the parturient the fetus must involve removal of the progesterone uterus they provide the routes through which electrical block. This is achieved by significant changes in the activity is propagated; in other words, they provide the hormonal profile coupled with the acquisition of gap structural basis for a functional syncytium that permits junctions by the myometrial cells.

synchronization of myometrial contractions. Thus, the The hormonal changes are initiated by the fetus, triggered by the recognition of 'stress' by the fetal hypot-uterine muscle to develop spontaneous rhythmical

thalamic–pituitary axis (Fig. 33.36). This results in the contractions. The transition of the myometrium from a release of ACTH that stimulates the fetal adrenal to quiescent state to an active state has been termed

Fetus

Hypothalamus

Corticotrophin-releasing factor (CRF)

Pituitary gland

Adrenocorticotrophin (ACTH)

Adrenal gland

Cortisol

Fig. 33.36

Hormonal control of parturition.

Placenta

Progesterone

The hormonal changes are initiated when the fetal hypothalamic–pituitary–adrenal axis

Oestrogen

releases increased amounts of cortisol. The

Prostaglandin

cortisol induces the placenta to release

increased quantities of oestrogens. The
oestrogen : progesterone ratio is switched
strongly in favour of oestrogen and the
inhibitory effect of progesterone on the

Mother

uterine muscle ('progesterone block') is

Uterus

Softening of cervix

abolished. The oestrogens promote uterine

Gap junctions in myometrial cells

contractility by stimulating the synthesis of

x

contractile protein and of receptors for both

Myometrial contractions

e l f

oxytocin and PGF2a and by facilitating the for-

e

Pressure on cervix and vagina

r

mation of gap junctions between adjacent

s'

myometrial cells. Gap junctions enable the

no

uterine muscle to develop spontaneous

Pituitary gland

s

s

rhythmical contractions. The more powerful

Oxytocin

u

F e r g

contractions during parturition are generated

in response to PGF_{2a}, augmented during the

Abdominal muscles

second stage of labour by oxytocin. (From

Contractions (pelvic reflex)

Hartigan, 1995.)

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‘activation’; the activated myometrium is capable of

concentrations of progesterone and oestrogen in the

generating high-frequency, high-amplitude contractions

circulation exert a prolonged negative feedback on

in response to mechanical or biochemical stimuli. The the maternal hypothalamic–pituitary–ovarian axis. The more powerful contractions during parturition are principal long-term effect of this inhibition is a pro-generated in response to $\text{PGF2}\alpha$, augmented during the gressive decline in the amount of LH in the pituitary second stage of labour by oxytocin. Both of these hormones influence smooth muscle activity by regulating the pituitary gland to GnRH, a low concentration of LH the concentration of calcium ions in the myometrial in the circulation and relatively quiescent ovaries. The cells. The concentration of free Ca^{2+} in myometrial cells inherent wave-like pattern of follicular growth is not increases about three-fold during contraction, through lost: it is in train within a few days after parturition, influx from extracellular sources and release from intracellular stores. A deficiency of calcium in a pregnant female at full term is associated with atony of the

able to trigger the neuroendocrine process that leads to uterine muscle and a tendency to prolapse of the uterus. oestrus and ovulation. Hence, immediately after Softening of the birth canal is achieved by the calving, the cow has a period of anoestrus, the length cascade of hormones: a rise in oestrogen concentrations of which can be influenced by age, season, nutritional followed by the activities of relaxin and PGF2a. Parturition can be induced after about day 255 of gestation or subclinical disease.

by a single injection of a synthetic glucocorticoid that simulates the effects of fetal cortisol, or after 270 days Re-activation of the by PGF2a. Retained placenta can be a problem with hypothalamic–pituitary–ovarian axis either method.

The primary objective of the neuroendocrine adjustment Tocolysis: Myometrial activity is inhibited by agents in the early postpartum period is to reinstate (such as β -adrenergic agonists and relaxin) that

the ovaries as fully functional components of the increase the intracellular levels of cAMP or cGMP, thus hypothalamic–pituitary axis and to restore the pituitary interfering with the interaction of free calcium ions with stores of LH to their normal levels so that the pituitary contractile protein.

gland can respond to pulses of GnRH by releasing Drugs that stimulate β_2 -adrenoceptors of the myome-
pulses of LH of sufficient frequency and amplitude to trial cells have been used to relax the uterine muscle in stimulate follicular maturation; in general, this is non-pregnant cows undergoing embryo transfer and in achieved by day 10 postpartum. In addition, the cow has pregnant cows at full term to delay parturition (tocoly-
to cope with the myriad of endogenous and exogenous sis) or to facilitate obstetrical operations such as stressors that might interfere with her ability to fetotomy, caesarean section or replacement of uterine progress the dominant follicle to its physiological prolapse. The duration of the tocolysis depends on the destiny as a preovulatory follicle. The fate of the first-

pharmacological effects of the agent and on the post-wave dominant follicle has a significant impact on the timing of the fetus at the time of treatment. For instance, duration of the interval to first ovulation: for instance, clenbuterol causes an outflow of calcium ions from the Beam and Butler (1999) reported the interval as 20 days myometrial cells and that renders the cells unresponsive for those that ovulated (46 per cent of their cows), 51 sive to oxytocin for some hours. When a single therapeutic dose of clenbuterol is given early in stage one of 48 days for those that regressed (31 per cent of cows) and labour it can delay parturition for 5–8 hours, but if it is cent of cows).

given in stage two after the cervix has been fully dilated Metabolic factors play key roles in the resumption of the delay may be as short as an hour or two. It has been reproductive activity (Fig. 33.37). Following parturition, reported that when uterine contractility returns, the the high producing dairy cow is unable to increase dry treated animal tends to have an 'easier' calving; it is

matter intake as fast as is necessary to meet the suggested that this is due to greater widening and increased nutrient demands for lactation; body reserves softening of the birth canal.

of fat and protein are mobilized to meet the deficit; in the circulation, the concentrations of non-esterified fatty acids and triglycerides are elevated, while the

Physiology of the postpartum period

concentrations of glucose, insulin, and IGF-1 are dimin-

During pregnancy, the mechanisms for the synthesis ished (Fig. 33.37) and the interval to first ovulation is and release of GnRH by the hypothalamus and those extended. According to Beam and Butler (1999), the for the release of the gonadotrophins by the pituitary interval from calving to the nadir of negative energy gland appear to remain intact and functional. The high balance has just as much significance for fertility as does

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E2

*

IGF-1

Response

E2

Insulin

to LH

LH surge

Fig. 33.37

*A schematic model describing
dominant follicle development (circles) and
FSH*

*function in relation to changing metabolic
and reproductive hormones, and energy*

Atresia

*balance (EB), during the first follicular wave
postpartum in dairy cows. The first-wave
follicle either ovulates (*) or undergoes*

IGF-1

E2

*atresia. LH pulse frequency is modulated
by the day of the EB nadir and, to a lesser
extent, the level of EB. The large upward
arrows indicate increased insulin-like*

growth factor-1 (IGF-1) and insulin leading to improved responsiveness to LH and greater oestradiol (E2) production by the Calving

LH

EB NADIR

dominant follicle. (From Beam & Butler, 1999, reproduced with permission of the Society for Reproduction and Fertility.)

EB LEVEL

the depth of the nadir. Available evidence seems to normal duration but with reduced concentrations of confirm that the modal interval to first ovulation has progesterone. Another group that may constitute become longer in recent years.

approximately 50 per cent of either the dairy herd or It is suspected that the negative energy balance may the beef herd has been reported to have a short luteal be a factor in the pathogenesis of abnormal cycles phase of five to ten days' duration during which the (short luteal phase, prolonged luteal phase, follicular

corpus luteum secretes reduced amounts of progesterone (Lamming et al. , 1981). These corpora lutea are anisms by which the negative energy balance can inhibit

not killed by the usual luteolytic process: their ability to ovarian activity are complex. For instance, there is some synthesize progesterone is lost prematurely. The tran-evidence that it may suppress LH pulse frequency; the sient low levels of progesterone after the first ovulation low levels of insulin and IGF-1 may retard the growth may have a significant (but, as yet, unidentified) effect of the dominant follicle; the steroidogenic activity of the on hypothalamic function and they almost certainly dominant follicle may be so reduced that it is incapable play a role in ensuring that oestrus is expressed before of exerting the positive feedback on the hypothalamus the second ovulation.

and the pituitary gland that is required for ovulation; if As mentioned above, a variety of factors can delay the follicles that ovulate are small, they may form small the onset of oestrous cycles. Although the events

corpora lutea that are incapable of secreting optimal leading to normal stores of LH in the pituitary gland concentrations of progesterone. It has been reported are relatively independent of the suckling stimulus that cows that experienced deep negative energy and environmental stressors, the events leading to an balance over the first 10 days postpartum subsequently increased frequency of LH pulses are susceptible to had subnormal progesterone concentrations in their inhibition by such factors.

third oestrous cycles.

In general, milked dairy cows ovulate earlier in the postpartum period than do suckling dairy cows or beef cows. For instance, Lamming's group at Nottingham has Oestrous cycles in postpartum cows reported that milk progesterone profiles on 505 milked The first ovulation may or may not be associated with dairy cows indicated a mean calving-to-first-ovulation oestrus; estimates of silent ovulation range from 40 to interval of 24 days compared with approximately 60 70 per cent. Frequently, oestrus is not observed until the

days for 365 suckled beef cows. Suckling depresses the end of the first full-length interovulatory period. The frequency of LH pulses, probably because it induces a first preovulatory surge of LH may be followed in some release of endogenous opioids that inhibit the normal cows by a complete cycle with a normal luteal phase. pulsatility of the hypothalamic–pituitary axis. There are However, other cows may have an initial cycle of high concentrations of prolactin in the circulation of

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lactating cows but, according to Lamming et al. (1981), been suggested that a very early ovulation from the ipsi-there is no evidence that hyperprolactinaemia plays a

lateral ovary in an infected cow may increase the risk significant role in the suckling-induced inhibition of of development of pyometra (Hartigan et al. , 1974). ovarian activity. The potency of the suckling stimulus It is perfectly ‘normal’ for bacteria to invade the declines with time after parturition. The duration of the bovine uterus during the first few days after parturition. period of inhibition can be influenced by other envi- Bacteriological studies have revealed that a very wide

ronmental factors, such as poor nutrition (e.g. when range of micro-organisms, both pathogens and commensals, gain entry but repetitive sampling showed that when early lactation) or season at calving (photoperiod?). parturition was uncomplicated the contamination of the For instance, cows that calved during January to June uterus was transient and that the isolates varied from took significantly longer to resume ovarian activity than one sample to the next. At the end of the fourth week did those that calved during July to December postpartum only 30 per cent of the cows had (Lamming et al. , 1981).

bacteria in the uterus, mostly non-pathogens (Griffin et al. , 1974). Under optimal conditions, most cows have eliminated the pathogenic contaminants before cyclic

Uterine involution

ovarian activity begins. On the other hand, cows that It should be emphasized that the uterus exercises some experience dystokia, traumatic damage to the

control over the activities of the ovaries by way of a
endometrium or retention of placental membranes are
local utero-ovarian axis and that uterine pathology can
less efficient in dealing with pathogens. There is a signif-
put a brake on the ovarian contribution to the larger
icant reduction in the efficiency of phagocytic cells in the
neuroendocrine axis. Synthesis and metabolism of
endometrium and uterine lumen and, as a result, poten-
PGF2a are elevated in the bovine uterus during the early
tial pathogens may persist and induce endometritis or
postpartum period and this is reflected in the presence
metritis. Again, in the majority of cows the endometritis
of a major metabolite (PGFM) in the blood at that time.
tends to be short-lived; the combination of phagocytosis,
There is accumulating evidence that ovarian function
local antibody production and myometrial contractions
does not resume until the concentration of this metabo-
that are normal features of the involutionary period
lite has fallen below a critical threshold. There appears
effect clearance of the micro-organisms and resolution
to be a significant negative correlation between the

of the endometritis. However, it is important to stress duration of elevated concentrations of PGFM after that infusion of irritants (antibiotics, iodine) that inhibit calving and the time required for completion of uterine phagocytosis, extensive intra-uterine manipulation that involution (Lindell & Kindahl, 1983). Persistent uterine damages the endometrium or an early ovulation that infections prolong the period of high concentrations of causes the onset of a precocious luteal phase may permit PGFM and the time to uterine involution. A problem a pathogenic micro-organism to become established may arise in the infected cow that ovulates very early within the uterus, particularly if the concentration of in the postpartum period because these continuously PGFM is still high. In such circumstances the elevated concentrations of prostaglandin are not sufficient to induce luteolysis and a mutual interdependence cause luteolysis. The result is that the defence mechanisms between the infection and the corpus luteum may be of the host are depressed by the progesterone

established, thus rendering the cow liable to develop from the corpus luteum and the pathogens cause continuous pyometra. However, the majority of cows do not cause damage to the endometrium, thus preventing the release of oestrogens until after the microbial population of the uterus has been reduced to transient contaminants, a stage, the pathogen and the corpus luteum have established a mutual interdependence that predisposes to the development of pyometra. The vicious circle can be broken by exogenous prostaglandin. The important point is that inappropriate medication early in the postpartum period can interfere with the physiological progress of involution and induce long-term pathological changes in the ovaries as early as the first week postpartum and at intervals of seven to ten days thereafter. During the second week after parturition the pool of antral follicles in the ovaries is increased both in dairy cows and in beef cows; the increase in numbers is

cal consequences.

largely due to the development of medium-size follicles

If a cow enters the second week postpartum without

(4–8 mm in diameter), particularly evident in the ovary

clinical evidence of an active inflammatory response, it

contralateral to the previous pregnancy. This predomi-

is unnecessary – and it may be unwise – to resort to

nance of the contralateral ovary seems to persist for

intrauterine antibacterial therapy: the drug may cause

about four weeks, with the result that the majority of

physical damage to the regenerating mucosa and –

first ovulations are from the contralateral ovary. It has

perhaps just as important – it may dampen the local

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immune response to micro-organisms that may chal-

reports of long-term (up to the third cycle) deleterious

lenge that system later, when the cow is inseminated.

effects on embryo survival in cows that experienced

If purulent endometritis/metritis develops it does not

deep negative energy balance during the first 10 days

abolish ovarian activity immediately and totally. Ini-

after calving. Were the carry-over effects due to a loss of developmental competence that resulted in embryo mortalities for some months before anoestrus supervenes. Obviously, the best prospects of full recovery to fertility will depend on diagnosis and treatment before the link between the hypothalamic–pituitary axis and the conceptus?

ovaries has been severed.

We have mentioned that ingestion of excess protein for problems seen in the postpartum period consult leads to elevated blood urea levels, lower pH of uterine Chapter 34.

fluids and lower fertility. The pregnancy wastage has been attributed to the deleterious effect of acidic uterine fluid on embryos (Butler, 2000) but

Uterine environment after resumption of

O'Callaghan and Boland (1999) took a different view:

cyclic ovarian activity

on the basis of experiments involving reciprocal

The quality of the luminal microenvironment of the embryo transfers, they suggested that the effects on female genital tract has a significant impact on fertility.

embryo quality are due to alterations in the tubal

The fluids secreted by the oviducts and by the uterus microenvironment or in the follicle rather than in the are the media in which the gametes are transported and uterus.

sustained; the spermatozoa are capacitated, hyperacti-

The iatrogenic introduction of micro-organisms can vated and acrosome-reacted, the ovum is fertilized and seriously diminish the quality of the uterine microenvi-

the conceptus is transported and sustained while it ronment during the breeding season. The uterus has a develops into the elongated blastodermic vesicle that higher degree of susceptibility to bacterial infection implants in the uterus. The appropriate secretions, mus- during the luteal phase than it has during oestrus. This

cular activity and flow of fluids are governed by oestrogen and progesterone released from the ovaries in the through the endometrium is inhibited by progesterone, correct concentrations, at the correct times and in a which also depresses the phagocytic activity of the cells specific sequence. Therefore, any factor that alters the once they have arrived in the endometrium. Therefore, delicate balance of ovarian hormones during the the clinician should exercise great care to avoid the oestrous cycle is a potential hazard to pregnancy, even introduction of contaminated or irritant material into when a normal fertile cow is bred to a highly fertile bull. the uterus during the luteal phase. For the same reason, The composition of the tubal and uterine fluids cows should not be inseminated at mid-cycle or after changes on regional and temporal bases – specifically conception. Persisting bacterial endometritis in a cyclic to meet the needs of the gametes and of the conceptus: cow could induce embryo loss because (i) it damages in particular, the changing needs of the conceptus as it

the endometrium so that it is not possible to establish progresses from a single cell zygote to an elongated an efficient interface for the transfer of nutrients; (ii) it vesicle. It follows that the preimplantation conceptus is induces embryo mortality or retards expansion of the vulnerable to damage by anything that disrupts the blastocyst which then fails to secrete enough IFN- γ to hormonal balance or otherwise alters the secretions. maintain the corpus luteum and/or (iii) it releases sufficient PGF_{2a} to induce premature luteolysis.

relationships between the various actions of the hormones. Problems of the postpartum period are dealt with mones, the secretory and muscular responses of the in Chapter 34, with endometritis being discussed from tubular tract, and the viability and developmental comp. 521.

petence of the oocyte. For instance, there are several highly sensitive junctures at which short-term acute stress might interfere with the maturation of the oocyte,

Take-home concepts for the clinician

fertilization, embryogenesis or implantation; it would be virtually impossible to ascertain whether the pregnancy wastage was due primarily to low developmental elements of reproductive physiology in cattle is a simplified account of some highly complex phenomena; the uterine microenvironment that was unable to sustain a aspiration has been to provide the broad concepts that fully competent conceptus. Frequently, it would be just ought to inform the diagnostic and therapeutic efforts as difficult to identify the cause of the acute stress. A of the clinician. So, what is the conceptual framework similar cause-and-effect conundrum attaches to the for the major themes presented here?

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Overall control of reproductive activities is vested in sale in the local store has been produced by cows that the HPG axis, within which the dominant role is played are 'hormone-laden'?

by the hypothalamus, literally the nerve centre that

coordinates and responds to the relevant neural, endocrine and immunological inputs. It exercises

References

control through the actions of a pulse generator that is responsible for the pulsatile release of GnRH which, in

Beam, S.W. & Butler, W.R. (1999) Effects of energy balance turn, regulates the release of the gonadotrophins that

on follicular development and first ovulation in postpartum

determine the nature, intensity and timing of events in

*dairy cows. Journal of Reproduction and Fertility, **Supple-the genital tract. For instance, it is the frequency and,***

ment 54, 411–24.

to a lesser extent, the amplitude of LH pulses that

Binelli, M., Thatcher, W.W., Mattos, R. & Baruselli, P.S. (2001) regulate the timing of ovulation. The pulsatility is

Antiluteolytic strategies to improve fertility in cattle.

modulated by feedback, by energy balance and by stress; Theriogenology, 56, 1451–63.

to take a practical example, it is likely that ovulation

Bo, G.A., Baruselli, P.S., Moreno, D., Cutaia, L., Caccia, M., will be suppressed when an animal is in negative energy

Tribulo, R., Tribulo, H. & Mapletoft, R.J. (2002) The

control of follicular wave development for self-appointed

*balance and/or is stressed by other environmental
embryo transfer programs in cattle. Theriogenology, 57, challenges.
53–72.*

*During the transition period the high-producing cow
Bronson, F.H. (1989) Mammalian Reproductive Biology.
is exposed to a plethora of stressors (both intrinsic and
Chicago University Press, Chicago.*

extrinsic), is relatively immunosuppressed and, once lac-

*Butler, W.R. (2000) Nutritional interactions with reproductive tation begins, is
in significant negative energy balance;*

*performance in dairy cattle. Animal Reproduction Science, in such
circumstances, reproduction has a relatively low*

60–1, 449–57.

*priority in the partitioning of energy and it is likely that Fawcett, D.W. (1994)
A Textbook of Histology. Chapman and the resumption of cyclic ovarian
activity will be delayed.*

Hall, London.

Of course, the deleterious effect of the energy deficit on

Ginther, O.J. (1996) Selection of the dominant follicle in cattle.

LH pulses is a major factor in the suppression of oestrus

Biology of Reproduction, 55, 1187–94.

*Ginther, O.J., Beg, M.A., Bergfelt, D.R., Donadeu, F.X. & Kot, and ovulation,
and in the low pregnancy rates to first*

K. (2001) Follicle selection in monovular species. Biology of service; however, the fact that approximately half of the

Reproduction, 65, 638–47.

mated cows calve to first service should convince us that

Griffin, J.F.T., Hartigan, P.J. & Nunn, W.R. (1974) Non-specific there are other contributing factors that tip the balance

uterine infection and bovine fertility I and II. Theriogenol-away from a satisfactory level of herd fertility. Thus,

ogy, 1, 91–106; 107–14.

management of the breeding programme demands

Grumbach, M.M., Roth, J.C., Kaplen, J.L. & Kelch, R.P. (1974) attention to hygiene and the physical facilities, to body

In Control of the onset of puberty (ed. by M.M. Grumbach, condition score, to feeding practices and to veterinary

G.D. Grove & F.E. Meyer), p. 158. Wiley, New York.

care throughout both the transition period and the

Hartigan, P.J. (1995) Cattle breeding and infertility. In Animal breeding season. The prime objective is to minimize the

Breeding and Infertility (ed. by M.J. Meredith), pp. 86–186.

allostatic load so that functions of HPG axis are not put Blackwell Science, Oxford.

Hartigan, P.J., Langley, O.H., Nunn, W.R. & Griffin, J.F.T.

at hazard by the coping strategies of an overburdened

(1974) Some data on ovarian activity in post-parturient

cow (see Chapter 67).

dairy cows in Ireland. Irish Veterinary Journal, 28, 236–41.

If there is a need to induce ovulation, the relevant

Hunter, R.H.F. & Wilmut, I. (1984) Sperm transport in the

conceptual framework derives from the inherent

cow: peri-ovulatory redistribution of viable cells. Repro-

duction Nutrition Development, 214, 597–608.

recruited follicles available every seven to ten days.

Ireland, J.J., Mihm, M., Austin, E., Diskin, M.G. & Roche, J.F.

Treatment should be administered at the appropriate

(2000) Historical perspective of turnover of dominant folli-

stage in a current wave or, as has been done by inves-

cles during the bovine estrous cycle: key concepts, studies,

tigators pursuing the aspiration to eliminate the need

advancements, and terms. Journal of Dairy Science, 83, for detection of oestrus after induced ovulation, in a

1648–58.

new wave that has emerged after surgical ablation of

Kulick, L.J., Kot, K., Wiltbank, M.C. & Ginther, O.J. (1999) Follicular and hormonal dynamics during the first follicular

the current wave (Bo et al. , 2002). The latter experiment wave in heifers. Theriogenology, 52, 913–21.

is an exciting application of basic physiological infor-

Lamming, G.E., Wathes, D.C. & Peters, A.R. (1981) Endocrine mation but it does prompt a rhetorical question: if an

patterns in the post-partum cow. Journal of Reproduction

effective pharmacological regimen capable of chemical

and Fertility, Supplement 30, 155–70.

ablation of cohorts of recruited follicles is devised and

Lindell, J.O. & Kindahl, H. (1983) Exogenous prostaglandin is then used widely on a whole-herd basis, how is the

F2a promotes uterine involution in the cow. Acta Veterinaria consumer likely to respond to the news that milk on

Scandinavica, 24, 269–74.

Reproductive Physiology in Cattle • 507

McCracken, J.A., Custer, E.E. & Lamsa, J.C. (1999). Luteoly-Short, R.V. (1984) In Reproduction in Mammals, 2nd edn (ed.

sis:

a neuroendocrine-mediated event .

Physiological

by C.R. Austin & R.V. Short), Book 3, p. 139. Cambridge

Reviews, 79, 263–323.

University Press, Cambridge.

McLaren, A. (1984) In Reproduction in Mammals, 2nd edn Smith, G.D., Jackson, L.M. & Foster, D.L. (2002) Leptin

(ed. by C.R. Austin & R.V. Short), Book 2, p. 21. Cambridge regulation of reproductive function and fertility. Theri-University Press, Cambridge.

ogenology, 57, 73–86.

Meiden, R., Milvae, R.A., Weiss, S., Levy, N. & Friedman, A.

Urbanski, H.F. (2001) Leptin and puberty. Trends in

(1999) Intra-ovarian regulation of luteolysis. Journal of

Endocrinology and Metabolism, 12, 428–9.

Reproduction and Fertility, Supplement 54, 217–28.

Wade, G.N. & Schneider, J.E. (1992) Metabolic fuels and

O’Callaghan, D. & Boland, M.P. (1999) Nutritional effects

reproduction in female mammals. Neuroscience and Biobe-

on ovulation, embryo development and the establishment

havioral Reviews, 16, 235–72.

of pregnancy in ruminants. Animal Science, 68, 299–314.

Webb, R., Gosden, R.G., Telfer, E.E. & Moor, R.M. (1999)

Rahe, C.H., Owens, R.E., Fleegen, J.L., Newton, A.J. & Harms, Factors affecting folliculogenesis in ruminants. Animal

D.G. (1980) Patterns of plasma luteinizing hormone in the

Science, 68, 257–84.

cyclic cow: dependence upon the period of the cycle.

Wiltblank, M.C., Fricke, P.M., Sangsritavong, S., Sartori, R. & Endocrinology, 107, 498–503.

Ginther, O.J. (2000) Mechanisms that prevent and produce

Roche, J.F., Austin, E., Ryan, M., O'Rourke, M., Mihm, M.

double ovulations in dairy cattle. Journal of Dairy Science,

& Diskin, M.G. (1999) Regulation of follicle waves to

83 , 2998–3007.

maximize fertility in cattle. Journal of Reproduction and

Wiltbank, M.C., Gumen, A. & Sartori, R. (2002) Physiological Fertility, Supplement 54, 61–71.

classifications of anovulatory conditions in cattle. Therio-Roche, J.F., Mackey, D. & Diskin, M.G. (2000) Reproductive genology, 57, 21–52.

management of postpartum cows. Animal Reproduction

Science, 60–1, 703–12.

Chapter 34

The Postpartum Period

I.M. Sheldon, D.C. Barrett and H. Boyd

Introduction

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tion or abnormal ovarian function causes subfertility

Uterine involution

508

which results in substantial economic loss for the cattle

Regeneration of the uterine epithelium

509

industry. Retained fetal membranes, endometritis and

Elimination of bacterial contamination

509

cystic ovarian disease, for example, delayed conception

Return of ovarian cyclicity

509

by 25, 31 and 64 days, respectively compared with cows

Monitoring the postpartum period

511

with a normal postpartum period (Borsberry &

Herd records

511

Dobson, 1989).

Individual clinical cases

511

History

511

The events that comprise the postpartum period are

Clinical examination

511

uterine involution, regeneration of the endometrium,

Principal factors affecting the postpartum period

512

elimination of bacterial contamination of the uterus

Enhancement of the postpartum period

513

and the return of ovarian cyclical activity. The initial

Postpartum lesions of the genital tract

513

stimulus for these changes to occur is the expulsion of

Lacerations and haemorrhage

513

the fetus along with the associated membranes and

Uterine prolapse

514

fluids at calving.

Retained fetal membranes

515

Pneumovagina and urovagina

519

Vaginitis

519

Acute puerperal metritis

519

Uterine involution

Endometritis

521

Pyometra

524

Postpartum anoestrus

525

Uterine involution is the return of the genital tract after

Cystic ovarian disease

526

calving to normal non-pregnant dimensions. This

Prevention of postpartum disease problems

526

involves physical shrinkage, necrosis and sloughing of

Breeding decisions and the management of the pregnant

caruncles, and the regeneration of the endometrium.

and periparturient cow

526

There is considerable loss of tissue, with the

Therapy immediately postpartum

527

mean weight of the uterus decreasing from about 13 kg

Conclusions

527

before parturition to about 1 kg during a 30-day period

(Kaidi et al. , 1991). The uterus and cervix contract

rapidly immediately after calving and it is often difficult

Introduction

to insert a hand through the cervix 24 hours

postpartum; the cervix only admits two fingers by 96

The postpartum period is the time during which the

hours postpartum. Uterine involution occurs in a

genital tract recovers after parturition, and returns to a

decreasing logarithmic scale with the greatest change

normal state ready for the next pregnancy. Despite a

during the first few days after parturition (Fig. 34.1).

trend for extended lactation for higher genetic merit

By 10 to 14 days postpartum the entire genital tract is

dairy cows producing high milk yields, the majority of

palpable per rectum in normal animals, although the farmers still select an earliest service date, or voluntary previously gravid horn can still be identified because it waiting period, between 40 and 60 days after calving. is longer and has a greater diameter than the con- Similarly, in seasonally calved herds, a short period for tralateral horn. Although the time required for com- recovery following pregnancy is often essential for plete involution is 40 to 50 days, the changes in uterine those animals that calve during the later part of the diameter are almost imperceptible after 20 days post- season. Monitoring the uterus and ovaries in the post- partum. Involution is delayed by postpartum disease partum period is important because delayed uterine including milk fever, retained fetal membranes and involution, the presence of postpartum uterine infec- uterine infection.

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Box 34.1.

Uterine defence mechanisms.

200

180

- *Intrauterine neutrophils*

160

- *Physical barriers of the vulva and cervix*
- *Persistent uterine contractions and involution*

140

- *Caruncular sloughing (to expel adherent bacteria)*

120

- *Secretory immunoglobulins*

100

- *Cell mediated immunity*

80

- *Intrauterine pH (rises at oestrus to 7.0, from 6.4 during dioestrus, which reduces bacterial growth)*

60

- *Resident vaginal bacterial flora*

40

Uterine horn diameter (mm)

20

0

5

10

15

20

25

30

Day postpartum

uterus. The postpartum environment of the uterine lumen supports the growth of a variety of aerobic and

Fig. 34.1

The mean external diameter of the previously gravid anaerobic bacteria. Many of these bacteria are simple (糝) and non-gravid (糝) uterine horns between 7 and 28 days contaminants in the uterine lumen and are removed by postpartum.

a range of uterine defence mechanisms (Box 34.1).

In one study, 93 per cent of the uteri obtained at an abattoir within 15 days of calving yielded bacteria on

Regeneration of the

aerobic and anaerobic culture of luminal swabs and uterine epithelium

endometrial tissue (Elliot et al. , 1968). The number of uteri from which bacteria were isolated had declined to

Following the loss of the allantochorion there is necro-

78 per cent by 16 to 30 days, 50 per cent at 31 to 45 days

sis of the uterine caruncle and this has usually sloughed

and 9 per cent by 46 to 60 days postpartum. In the live

by day 12 postpartum. Sloughing of the uterine carun-

animal, there is a constantly fluctuating bacterial flora

cles contributes significantly to the rapid reduction in

in the first 7 weeks postpartum due to spontaneous

weight of the involuting postpartum uterus because

contamination, clearance and recontamination. How-

the caruncles account for over half of the weight of the

ever, uterine infection is commonly associated with

uterus. The sloughed caruncles form the lochial dis-

Escherichia coli, Arcanobacterium pyogenes, Fusobac-charge, along with the remains of fetal fluids and blood

terium necrophorum and Prevotella (formerly Bac-from the ruptured umbilicus. The lochia is usually a

teroides) species. Indeed, A. pyogenes, F. necrophorum yellow to brown, viscous fluid without an unpleasant

and Prevotella species have been shown to act syner-

odour. The uterus of normal animals contains one to

gistically to enhance the likelihood of uterine disease two litres of lochia immediately postpartum and the and increase the risk of clinical endometritis and its greatest discharge is in the first two to three days; it has severity.

virtually disappeared by 14 to 18 days after calving.

There is initially regeneration of the endometrium in the intercaruncular areas and then by centripetal

Return of ovarian cyclicity

growth of the cells over the caruncle. Epithelial regeneration is complete by about day 25 postpartum, but the

During the bovine oestrous cycle, ovarian follicular deeper layers of tissues are not fully restored until 6 to development occurs in a wave pattern starting with the 8 weeks postpartum. Whilst these epithelial changes are emergence and recruitment of two to six follicles of 4 taking place the caruncles are shrinking. By 40 to 60 to 6 mm diameter (Savio et al. , 1988). One of these days postpartum the caruncles are only 4 to 8 mm diameter and 4 to 6 mm high, compared with 40 to 70 mm

dominant follicle, whilst the remaining follicles become diameter and 25 mm high at calving.

atretic and regress (Fig. 34.2). Each follicular wave is preceded by an increase in circulating FSH concentra-

tion (Adams et al. , 1992). Selection of the dominant fol-
Elimination of bacterial

lice results in oestradiol production, and negative contamination

feedback on FSH, with the follicle subsequently becom-

ing LH dependent (Ginther et al. , 1996). Early luteal At calving, the vulva is relaxed and the cervix dilated,

phase progesterone secretion, however, suppresses LH

allowing bacteria to gain entry to the vagina and the

pulse frequency and the first dominant follicle under-

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Uterine involution

LH

Progesterone

FSH

0

5

10

15

20

days

Fig. 34.2

Cartoon of resumption of ovarian cyclical activity in a dairy cow during the first 20 days postpartum. As uterine involution (--) progresses, there is a transient increase in plasma FSH concentration (—), followed by the emergence of several follicles > 4mm diameter (•), with subsequent selection of a dominant follicle (●) and atresia of the subordinate follicles (○). The fate of the first dominant follicle is dependent on LH pulse frequency, which in the present case is sufficient to cause ovulation (), and the subsequent formation of a corpus luteum secreting progesterone (...), heralding the return of ovarian cyclical activity.*

goes atresia. Conversely, at the other end of the cycle, quency; ovulation generally occurs once the LH pulse after luteolysis the LH pulse frequency increases, frequency is one per hour. Therefore, factors that sup-stimulating growth of another dominant follicle that press LH pulse frequency in the postpartum period can increases plasma oestradiol concentrations, which delay the return of ovarian cyclical activity. Those subsequently stimulate an LH surge and ovulation. factors causing anoestrus tend to be more common in Throughout pregnancy, regular periodic emergence

the beef suckler cow, and include suckling or the maternal bond, low body condition score and inadequate recurrent FSH surges, except for the last 21 days of pregnancy due to very high plasma progesterone and dairy cattle is negative energy balance.

oestrogen concentrations. After parturition steroid hormone concentrations decrease to basal values, there is an increase in plasma FSH concentration within days of calving and that stimulates the emergence of the first dominant follicles or corpora lutea in the ovary ipsilateral to the previously gravid uterine horn, compared with the contralateral ovary (Sheldon et al. , 2000).

dominant follicle is selected around day 10 to 12 post-

partum (Savio et al. , 1990; Beam & Butler, 1997). These ipsilateral ovary, although less frequent, is a positive

events occur in all postpartum cows irrespective of perimarker of subsequent fertility.

parturient disease, environment or dietary deficiencies.

Early return of cyclical ovarian activity is generally

However, the first dominant follicle has three possible

accepted to be beneficial for subsequent fertility

fates: ovulation and formation of the first postpartum

(Darwash et al. , 1997); however, others have reported corpus luteum (return of ovarian cyclical activity),

the converse (Smith & Wallace, 1998). Furthermore, it

atresia with the emergence of one or more follicular

is suggested that an early postpartum first ovulation in

waves without ovulation (anoestrus) or formation of an

the presence of uterine infection can lead to pyometra

ovarian follicular cyst (Beam & Butler, 1997). In

with persistence of a CL in the presence of pus within

Europe, 70–80 per cent of first dominant follicles are

the uterine lumen (Olson et al. , 1984).

ovulated in dairy cattle fed an appropriate diet whereas

Farmers rarely observe oestrus at the time of the first

in the beef suckler cow ovulation of the early postpar-

postpartum ovulation in cattle, and in a significant number of animals the first luteal phase is relatively short. Using continuous observation, oestrus is detected. Ovarian cyclicity in beef suckler cows is an expression of their failure to ovulate, rather than a failure to develop dominant follicles. The principal factor determining the fate of the dominant follicles is LH pulse frequency prior to ovulation is important for oestrous expression.

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Monitoring the postpartum period

animals an examination is made three to five weeks postpartum to identify abnormalities of the puerperium

Herd records

that might lead to subsequent infertility. This may include the following procedures.

Recording the basic fertility parameters including the

date of calving, dates of observed oestrus and service dates is invaluable for analysis to determine whether a

Physical examination

herd is achieving its fertility targets (see also Chapter Body condition scoring of cows at drying-off, at calving, 41 (b)). The importance of efficient and accurate heat at routine postpartum examinations and at the time detection cannot be stressed too often to farmers. In of insemination is an effective method to assess the addition, the recording of calving ease, the occurrence adequacy of nutrition in a herd and to monitor energy of retained fetal membranes, abnormal vulval discharge balance. Indeed, changes in body condition score reflect and other peripartum problems identify those herds, the effect of negative energy balance on fertility as well and animals within a herd, that warrant particular attention as other markers of energy balance such as plasma concentration during the postpartum period.

centrations of insulin-like growth factor 1 (IGF-1) and b-OH butyrate (BHB). In addition, the presence of

Individual clinical cases

ketosis can be established by analysis of urine, milk or serum, and is associated with postpartum uterine infec-

In many cows presented immediately after calving, tion (Markusfeld, 1984). The use of milk analysis as a diagnosis of postpartum problems is quite simple, for method of assessing the nutrition of dairy cattle and example retained fetal membranes or prolapse of their metabolic status is discussed further in Chapter 47 the uterus. However, a cow that is acutely ill could be (see p. 807).

suffering from a variety of conditions and, therefore, a Palpation per rectum of the genital tract is the pre-systematic approach to diagnosis is necessary both dominant method of assessing uterine involution and for differential diagnosis and to ascertain the severity the return of ovarian cyclicity. Uterine involution of the condition. Investigation of the individual cow may be considered to be complete when the genital should include the history, a thorough physical exami- tract has contracted such that the uterine horns are of nation and further investigations as indicated.

equal diameter and length and the uterus has returned to lie within the pelvic cavity. Return of ovarian cyclic-

History

ity is usually determined by the palpation of a corpus luteum in the ovary; palpation of follicles in the ovary

Questions should be asked to ascertain whether the does not indicate return of ovarian cyclicity. Anoestrus

cow was ill before calving and whether the illness

cows may have ovarian follicles up to 18 mm diameter,

was related to reproduction or was a condition that is

but there is no corpus luteum. Palpation of ovarian

obviously related, such as a pathological vaginal

structures more than 25 mm in diameter may indicate

discharge. Information about previous feeding and

the presence of a follicular or luteal cyst. However, a

management should be gathered, particularly relating

substantial proportion of cystic structures identified

to the transition period, with the object of determining

before day 30 postpartum will spontaneously regress

whether the diet could have predisposed to metabolic

(Chapter 35).

diseases. Details of other postpartum problems in the

Palpation per rectum alone is not sufficient to determine in this and previous breeding seasons should also

determine the adequacy of uterine involution and elimination be obtained.

tion of bacterial contamination. A vaginal examination

The place where the cow calved should be inspected

is essential for the assessment of the presence or

to assess the level of environmental hygiene during

absence of postpartum uterine bacterial infection. Gen-

calving. The farmer should be asked if the calving was

assisted and, if so, how much and what help was given,

by inserting a lubricated, clean, gloved hand between

by whom and what hygienic precautions were taken. It

clean vulval lips. During the examination, vaginal and

is also important to determine whether the client has

cervical or lacerations and abnormalities are noted and

given any treatment already.

a sample of vaginal mucus withdrawn and examined for

character, volume and smell. The combination of a

vaginal examination and a rectal palpation can be used

Clinical examination

to score the severity of endometritis, which is a prog-

A thorough clinical examination must be undertaken,

nostic indicator of the success rate of treatment for

although the optimum time after calving for examina-

endometritis (Sheldon & Noakes, 1998). Observation of

tion is debatable. Most often in outwardly healthy

the vagina, vaginal mucus and the cervix with a specu-

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lum may provide additional information; sterile, dis-

nately, the cost of collection and evaluation for the

posable specula are available.

individual animal precludes the widespread use of the

technique in cattle.

Ultrasonography

Bacterial culture

Real-time B-mode ultrasonography using a linear array

transrectal probe has revolutionized the veterinarian's

Guarded transcervical swabs can be used to collect

ability to monitor the return of ovarian cyclical activity

samples of intrauterine fluid for bacterial culture and and uterine involution. Visualization of the genital tract identification. It is essential that both anaerobic and before day 10 postpartum is difficult, particularly in aerobic cultures are used, which requires the use of older animals, and a 5 MHz probe is more appropriate a transport medium and specialized microbiological for examination of the enlarged uterus at this time. techniques. The predominant pathogens associated with However, a 7.5 MHz probe is superior for examination endometritis are A. pyogenes, E. coli, F. necrophorum of the ovaries. Ultrasonography is particularly useful and Prevotella melaninogenicus. Sensitivity tests for for identifying ovarian follicles; a dominant follicle is selection of an appropriate antibiotic might be of value usually defined as a follicle ≥ 9 mm diameter in the for herds with a high incidence of endometritis. absence of other large growing follicles. However, caution has to be exerted when considering structure and function; the detection of a large follicle or follicu-

Principal factors affecting the

lar cyst alone does not indicate that it is endocrinolog-

postpartum period

ically active (Beam & Butler, 1997).

Use of the ultrasound machine's internal callipers

There are a variety of factors than can influence the allows measurement of the diameter of the uterus and duration of the postpartum period. For convenience, we ovarian structures and comparison with the expected have already considered the various components of this normal values determined by sequential measurement period as separate entities; however, this does not of uterine horn diameter during the process of involu- reflect the normal situation. Uterine involution, bacte- tion. In addition, abnormal contents of the uterine rial infection and the return of ovarian cyclicity are all lumen are readily detected. The entire length of both closely linked.

uterine horns should be scanned, because often pus is

Abnormalities of the puerperium such as dystokia, localized within a limited section of the genital tract. retained fetal membranes, stillbirths, abortions and However, the use of transrectal ultrasonography does

twins delay uterine involution and the return of ovarian not negate the value of performing a manual or speculocyclicity. In addition, these problems are associated with lar examination of the vagina before examining the an increased risk of metritis and/or endometritis. Con- remainder of the genital tract.

versely, early postpartum ovulation prior to the elimination of bacterial contamination may predispose to pyometra if uterine infection persists in the presence of

Progesterone analysis

a corpus luteum.

Estimation of plasma or milk progesterone by enzyme-

Suckling has been widely reported to delay the return

linked immunosorbent assay (ELISA) or radioim-

of ovarian cyclicity. It is interesting to compare studies

munoassay (RIA), collected two to three times per

using milk progesterone estimation to determine the

week after calving is adequate to determine the first

mean time to postpartum resumption of ovarian cycles

postpartum luteal activity and abnormal patterns of

in dairy (Bulman & Lamming, 1978) and beef cows

ovarian activity. Milk samples are more practicable in (Peters & Riley, 1982), which were 24.0 ± 0.6 and the field because of the ease of collection. Samples of 59.9 ± 2.5 days, respectively. It would appear that this 10 ml of milk can be stored for at least a month in con-inhibitory influence, mediated by reduced LH secre-tainers with a potassium dichromate tablet as a preser-tion, is a function of the maternal bond attributable to vative (Lactab Mk III, Thompson & Capper, Runcorn) the dam's own calf, rather than simply an effect of suck-and refrigerated at 4°C.

ling or milk yield (Williams & Griffith, 1995). It has also been suggested that higher milk yields are associated

with longer acyclic periods, although it is difficult to sep-

Endometrial biopsy

arate the effects of milk yield and confounding factors

Histological examination of endometrial biopsies has

such as nutrition.

not been used as frequently in cattle as in mares.

Inadequate nutrition, and particularly a diet deficient

However, endometrial biopsies are valuable indicators

in energy, is the most important cause of postpartum of subsequent reproductive performance. Unfortun-
anoestrus. Body condition score (BCS), particularly in
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beef suckler cattle, is an important factor affecting the
The first action of the obstetrician after the delivery and
resumption of ovarian cyclicity and cows with a body
resuscitation of a calf should always be to check the
condition score less than 2.5 are likely to have a signif-
icant delay (see p. 10). In dairy cows, changes in BCS
ity should be assessed and appropriate action taken;
after calving are more important than BCS at calving.
severe haemorrhage involving a large artery can be
rapidly fatal.

Traumatic and post-traumatic lesions are treated

Enhancement of the

according to general principles of wound management.

postpartum period

If the calf has been delivered without the use of
epidural analgesia, but surgical intervention is required

Shortening the postpartum period would increase the to control haemorrhage, then the use of an epidural opportunity for rebreeding cattle after calving. Unfortunately, most practical attempts to advance uterine of analgesia obtained by this method it may be advanced involution have been disappointing; a variety of therapeutic agents to include xylazine in the epidural (Ko et al. , peutic agents have been used in the early postpartum

1989; Holden, 1998). When a vaginal vessel is bleeding period with no significant effect.

severely, an attempt must be made to clamp the trau-

A number of therapeutic agents have been used to matized vessel with artery forceps which can them be try to induce ovulation in the early postpartum period left in place for 24 hours. Ligation and the suturing of and, therefore, shorten the interval to return of ovarian lacerations would also be indicated in some cases, cyclicity; these agents include GnRH, progesterone and although it is often impractical due to the inability to eCG (see p. 678). However, results are inconsistent and

gain adequate access to the site. If bleeding vaginal often of little long-term benefit to the fertility of the vessels cannot be clamped or ligated the only other herd.

option is to apply pressure using materials such as clean In the absence of consistently effective pharmacotowels or bedsheets or a pressure pad containing ice in logical methods of shortening the postpartum period, it an attempt to bring about constriction of the bleeding is particularly important for the veterinary surgeon to vessels and promote blood clotting.

be familiar with the management factors affecting this Where haemorrhage is occurring from uterine period and to give appropriate advice to clients to avoid lacerations, oxytocin may also be used to promote contheir negative effects. For example, veterinarians should traction of the uterus. In cases of uterine rupture, use condition score targets for key points in the manlaparotomy and surgical repair may be the only alteration cycle such as at calving and drying off, in dairy native to emergency slaughter. Following severe haem-

cattle, or weaning, in beef suckler cows. In addition, it
orrhage a blood transfusion may be indicated as a life
is important to ensure the cows are fed an appropriate
saving procedure.

diet, paying particular attention to the energy density

In less severe cases the bleeding may stop sponta-
of the ration.

neously, although lacerations, severe bruising and tissue
trauma may result in the later development of necrotic
vaginitis. Where perivaginal fat prolapses through

Postpartum lesions of the

vaginal lacerations and becomes necrotic, this may have

genital tract

to be removed to allow closure of the vaginal tear.

Infection with *F. necrophorum* or *Clostridium* species Traumatic injuries to the
genital tract, bony pelvis and

may result in a locally severe reaction and serious

nerves of the pelvis sometimes occur during normal
systemic illness or death.

parturition. However, they are more common following

In the case of necrotic vaginitis emollient fluids and

dystokia. Postpartum problems include traumatic antiseptics are applied locally along with an appropriate systemic antibiotic such as a cephalosporin or uterus or vagina, tearing of the cervix or vulva, potentiated amoxycillin. Following any difficult parturition with associated trauma to the reproductive tract haematoma, as well as other conditions such as retention of the fetal membranes and prolapse of the uterus the cow is likely to benefit from the administration of systemic non-steroidal anti-inflammatory drugs (Noakes, 2001a).

(NSAIDs) (Chapter 62).

The response to treatment of many vaginal lesions is good and the clinical signs will disappear within about

Lacerations and haemorrhage

10 days. In these cases there is no interference with

There is usually, but not always, a history of a protracted future reproduction. In a few cases infection spreads or particularly difficult per vaginam delivery of the calf.

and affects the uterus, with resultant poor prognosis for



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*fertility. Scar tissue may cause narrowing of the birth
with reduced abdominal straining may actually aid
canal and interference with the next calving.*

replacement of the prolapsed organ if the procedure

For further information on nerve damage, the reader can be undertaken swiftly and treatment with calcium should consult Chapter 32.

borogluconate is not delayed too long.

The replacement of the uterus is aided by the correct positioning of the cow. If the cow is standing the uterus

Uterine prolapse

may be lifted on a sheet with the aid of two assistants

Uterine prolapse is a relatively common complication and replaced from that position. However, if the cow is

of third stage labour in the bovine, with the uterus

recumbent she should be placed in sternal recumbence

prolapsing within hours of the calf being delivered and

in the frog-leg position as described by Plenderleith

prior to the cervix contracting (Noakes, 2001b). The

(1986) (Fig. 34.3) and if possible positioned facing down

usual presentation is of a complete inversion of the

hill. This positioning of the cow is achieved by rolling

gravid uterine horn. While prolapse of the uterus may

her onto one side and pulling the opposite leg straight

follow a dystokia with a high degree of traction, it is also

back, before rolling her to the other side and repeating commonly seen as a sequel to a normal or unassisted the process. If necessary, ropes can be used to hold the second stage labour. Plenderleith (1986) reports that cow in this position until the uterus has been replaced. the condition is recorded in 0.5 per cent of assisted calv- Replacement of the uterus is greatly aided by the ings and 0.3 per cent of all calvings, while Correa et al. administration of an epidural anaesthetic, possibly (1992) report the condition in 0.6 per cent of calvings.

including xylazine (Ko et al. , 1989; Holden, 1998); this The main predisposing factor in the aetiology of this

also reduces postoperative straining and improves pain condition is considered to be uterine inertia immedi- relief for the cow. Before replacing the uterus it must ately postpartum. Hypocalcaemia (milk fever) (see p. be carefully checked for trauma and cleaned as much 781), which results in a reduction of smooth muscle tone as possible using a warm, non-irritant, antiseptic solu- in the uterus, and elsewhere, has been shown to increase tion or normal saline; lacerations of the uterus may

the risk of uterine prolapse threefold, while dystokia require suturing. There is no absolute need to remove increased the risk fivefold (Correa et al. , 1992). The the placenta, but often the most effective way of clean-increased risk of hypocalcaemia in older cows may ing the organ is to remove the heavily contaminated explain why multigravida are more commonly affected fetal membranes. However, if the placentomes are still than primigravida. Factors that are considered to pre-intact and the fetal membranes are not removed easily dispose to prolapse of the uterus are summarized in they should be left in place.

Box 34.2.

Box 34.2.

Possible predisposing factors for uterine prolapse.

- Fetal oversize and maternal/fetal disproportion
- Prolonged dystokia
- Fetal traction
- Hypocalcaemia
- Retained fetal membranes
- Chronic disease, particularly cattle in very poor body

condition

When notified of a case of uterine prolapse, the veterinarian should inform the client to wrap the prolapse in a clean sheet to prevent further trauma and contamination and reduce heat loss.

When faced with a case of uterine prolapse in a cow with overt clinical signs of hypocalcaemia, treatment of the hypocalcaemia should probably be carried out prior to replacing the uterus, in case regurgitation and aspiration of rumen contents occurs or the cow succumbs

Fig. 34.3

Recumbent cow in the 'frog-leg' position for replacement of a uterine prolapse. Reproduced from Plenderleith (1986) hypocalcaemia which maintains a cow in recumbency with permission.

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The weight of the uterus is best supported by an assistant on either side of the cow while the surgeon replaces those cows that were subsequently mated, 78 per cent

the uterus little by little starting at the vulval lips. Gentle were found to be pregnant, although 9 per cent subse-but continuous pressure is applied with a clenched fist or

quently aborted. This indicates that prompt attention

the open palm of the hand as portions of the uterus are

to uterine prolapse gives a reasonable survival rate and

pushed back into the vagina. Care must be taken to

acceptable subsequent conception rates. Possible com-

ensure that fingers are not pushed through the often

plications following uterine prolapse are listed in Box

friable uterine wall. Once the uterus begins to be

34.3.

replaced it is best to maintain momentum, as a single

abdominal contraction from the cow while the uterus is

Box 34.3.

Possible complications of uterine prolapse.

not held can result in the entire uterus once again

becoming prolapsed. As the uterus disappears within the

- *Hypocalcaemia*

vulval lips the veterinarian should continue to press it

- *Haemorrhage*

forward to the full length of his/her arm. It is important

- *Metritis*

- *Endometritis*

that the whole of the uterus passes through the cervix

- *Peritonitis*

and that the total length of the uterine horn is fully

- *Toxaemia/septicaemia*

replaced and any remaining inversion of the tip of the

- *Necrosis of devitalized tissue in longstanding cases*

uterine horn reduced. This can be achieved using some-

- *Uterine rupture*

thing to extend the veterinarian's reach such as a clean

smooth-ended bottle. Alternatively a volume (5–10

litres) of warm clean water, normal saline or dilute anti-

Retained fetal membranes

septic solution can be instilled into the uterus and then

Introduction

the majority can be siphoned back out. This has the

added advantage that it also has a cleansing action, and

Retained fetal membranes (RFM) is a common condi-

any fluid remaining in the tip of the uterine horn may act

tion that is defined as non-separation of the fetal mem-

*to hold the uterus in place until the recurrence of normal
branes by 12 hours after calving (some authors extend
uterine contractions results in its expulsion.*

this period to 24 hours before they consider the pla-

*There is debate within the profession as to whether
cental retention to be pathological). The incidence*

*there is a need to suture the vulval lips closed after
varies from study to study but tends to be about 5–10
replacing a uterine prolapse. If suturing is considered*

per cent (Kossaibati & Esslemont, 1995; Parkinson,

necessary it is best to use Buhner's technique with

2001); in a survey of >160 000 calvings in the Nether-

obstetrical tape, although many other techniques are

lands the average incidence was 6.6 per cent (Joosten et

*available (Hooper et al. , 1999). Following replacement al., 1988). In problem
herds the incidence may be much of the uterus every effort must be made to
restore*

higher. The year of calving, season of calving, parity of

uterine tone and promote involution and closure of the

dam, calf mortality, calving difficulty and fetal presen-

cervix. To this end hypocalcaemia must be treated and

tation have all been shown to affect the incidence of

oxytocin may be administered; parenteral antibiotics

RFM (Mee, 1991).

and analgesics are also indicated. Some uterine prolapse cases prove fatal due to trauma and haemorrhage includ-

Diagnosis and consequences of retained

ing rupture of the uterine and/or ovarian blood vessels, fetal membranes

shock or the often concurrent hypocalcaemia. However, the prognosis depends largely on the following:

The diagnosis of RFM is self-apparent, the major variable being the effect of the condition on the cow's

- *The time lapse between the development of the health. In many cases health is unaffected. However, prolapse occurring and professional treatments the majority of cows have mild disturbances of appetite being undertaken.*

and body temperature. In a small number of cows, acute

- *The degree of trauma that has taken place to the metritis develops causing severe illness, with typical uterus.*

signs of toxæmia and septicaemia. Without effective

• Associated periparturient conditions such as treatment this condition can be fatal. Some of the hypocalcaemia and haemorrhage. effects of RFM are summarized in Box 34.4. Economically, RFM represents one of the most significant postpartum abnormalities with each case reported to have undertaken in New Zealand (Oakley, 1992) showed a direct cost of approximately £85, mainly due to a that 19 per cent of cows died within 24 hours of treatment and a further 16 per cent died or were lost to the effects on subsequent poor fertility are also considered

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Box 34.4.

Some of the effects of retained fetal membranes. However, on some occasions retention is associated with uterine atony: for instance, after

- *Reduced dry matter intake*

overextension of the myometrium as in cases of

- *Withholding of milk from the food chain and possibly a lowered milk production*

hydrops allantois, or prolonged labour or in

hypocalcaemic cows. It is unlikely, however, that

- *Increased incidence of postpartum metritis, endometritis and pyometra*

uterine atony in the absence of some disturbance

- *Increased time to first service*

of the process of caruncle/cotyledon separation

- *Reduced conception rates/increased services per would result in RFM.*

conception

- *Increased days open*

In addition to the above mechanisms, nutritional

- *Longer calving interval*

problems such as vitamin E/selenium, iodine and

- *Predisposition of cows to other conditions such as vitamin A deficiency have been reported to be associated with left-displaced abomasum and laminitis*

ated with an increased incidence of RFM in some herds,

- *Mortality*

although individual cows are not normally considered to develop RFM due to nutritional deficiencies.

Laven and Peters (1996) have reviewed the aetiology and pathogenesis of fetal membrane retention in the cost is estimated to be around £300 per case detail.

(Kossaibati & Esslemont, 1997).

Treatment

Aetiology

Over time the natural sloughing of the maternal

The specific pathogenesis of RFM still remains uncertain and uterine involution contributes to the retention due in part to our poor understanding of normal loss of the membranes. If left untreated, freeing of placental separation in the bovine. However in simple terms there are considered to be two main ways in which fetal membrane retention occurs:

sis over several days. The toxic products of this putre-

(1)

Interference with the separation of the fetal villi

faction accumulate within the uterus, causing a fetid

from the maternal caruncles. This may be associ-

odour that taints the milk making it unfit for human

ated with the following:

consumption.

(a)

Premature calving (induced or spontaneous)

*Moller et al. (1967) describe a large clinical trial in without the normal
maturation of the pla-which the results of a wide variety of treatments were*

centome involving hormonal and structural

studied. They pointed out that cows that are ill and cows

changes in preparation for separation.

with metritis must be treated, but they concluded that

This maturation involves changes in the

the evidence did not indicate a need to treat cases of

molecular structure of collagen within the

uncomplicated placental retention. In fact it appeared

placentome and alteration in binucleate cell

that any type of drug therapy for this condition tended numbers in the trophoctoderm. Spontaneous to depress subsequent pregnancy rates, whereas cows premature calvings will include many twin that were not treated (that is, membranes not removed, births as well as abortions.

no drug therapy administered) had pregnancy rates that (b)

Prolonged gestation may result in excessive were not significantly different from those achieved by growth of the dam's caruncles.

cows that passed the membranes at the proper time. In (c)

Trauma with oedema of the villi, which cows that remain healthy, treatment may therefore not occurs after Caesarean section, torsion of the be indicated. However, in a small proportion severe uterus and other dystokias.

metritis and toxaemia may develop, which may be fatal (d)

Infectious conditions (with or without abor-

if left untreated.

tion) result in inflammatory changes that

bind together the maternal and fetal tissue in

*Manual removal of fetal membranes: Veterinarians have
the placentome.*

been arguing for decades over the need for manual

(e)

Hyperaemia of the placentomes.

removal of retained fetal membranes. It should be

(f)

Necrosis of the villi.

borne in mind that although the results of many field

(2)

Observations on uterine contractions after calving

trials tend to favour non-removal, the arguments

suggest that in many cases of retained fetal mem-

in favour of manual removal are said to include the

branes, their frequency and amplitude are greater

following:

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(1)

It removes a major source of infection and putre-

*Advocates of conservative treatment may intervene
fying protein.*

*only if the animal develops signs of systemic disease, in
(2)*

*It removes the unpleasant smell, which can taint
which cases the treatment is systemic administration of
the milk, and physical presence of the retained
a broad-spectrum antibiotic. Other veterinarians simply
membranes.*

*cut off the part of the membranes that is visible to
(3)*

*The cow may be less likely to develop systemic
reduce the smell and only apply other treatment in
illness.*

those (few) cases that become systemically ill.

(4)

The cow may be less likely to have disturbed

*It would seem that current practice in the field is
fertility later.*

often at odds with research findings regarding treat-

(5)

The cow may be less likely to suffer from reduced ment of RFM. Laven (1995) undertook a survey of the milk yield.

methods used by British veterinarians for treating RFM

(6)

A small number of conservatively treated cows and found that manual removal was used in some cases become very ill and require life-saving treatment.

by at least 92.5 per cent of respondents. Ecboic agents including prostaglandin F

These points are not necessarily sufficient justifica- 2a (or analogues of PGF2a)

and oxytocin were sometimes used by 84.2 per cent, tion for manual removal of RFM, as point 2 is a benefit with 15.7 per cent using oestradiol (no longer permitted to the farmer rather than the cow and points 3, 4 and 5 ted in many countries) in an attempt to potentiate the have been contradicted by many published trials. Points effects of oxytocin. Most veterinarians reserved the use 1 and 6 are true, although there is little evidence that

of antibiotics for cows that were systemically ill, but 18 toxaemia occurs because the placenta has not been per cent used them in animals with no overt illness. removed. Toxaemia can occur after removal of the Only 1.6 per cent of respondents routinely left cases of placenta (Peters & Laven, 1996).

RFM untreated.

Those in favour of manual removal claim that the At the present time it is difficult to say categorically danger of reproductive or general disease is greatly that cows should or should not be treated by manual reduced with gentle handling, a willingness to stop removal of the membranes, or which cows would manual removal where the attachment is firm, and benefit from manual removal rather than other medical the concurrent use of local or systemic antibiotics. If or conservative treatment. However, it is in conditions manual removal is to be used this should not be such as RFM, where we need evidence-based medicine attempted until a minimum of 96 hours after calving most, that treatment decisions of this type should be

and in many cases it is necessary to leave for a longer based on sound scientific and economic analysis to time period than this. Manipulation should be confined provide decision support rather than individual to gentle traction on the membranes and not involve arbitrary decisions.

the manual breaking down of each individual placen- tome as this results in significant trauma to the Ecboic drugs: Prostaglandin F endometrium.

2a or analogues have

been administered systemically to encourage the

The arguments used against manual removal of the removal of fetal membranes. Stevens et al. (1995)

retained fetal membranes include the following:

evaluated the use of an intrauterine infusion of oxy-

(1)

Just after calving the cow is ready to deal with the

tocin, subcutaneous injections of fenprostalene or a

large amount of infected material and decompos-

combination of both. They concluded that the interval

*ing necrotic caruncles that are sloughed off into
from parturition to expulsion of the fetal membranes
the uterus in the normal course of events; the need
was unaffected by treatment, as was the frequency of
to get rid of fetal membranes as well presents no
subsequent displaced abomasum, ketosis and mastitis.
insurmountable problem to most cows.*

*However, Stocker and Waelchli (1993) found that the
(2)*

*Intrauterine manual intervention should be
administration of PGF2a after Caesarean section
avoided because it interferes with the natural
decreased the risk of retention of fetal membranes.
defence mechanism by reducing phagocytosis for
Similarly, oxytocin used up to 36 hours after calving
several days.*

*may increase myometrial contractions and hasten
(3)*

*Manual removal of the membranes is never com-
expulsion of the membranes. Trial results have been
plete and numerous villi and remnants of placenta*

variable regarding the use of oxytocin (Stevens & are left attached.

Dinsmore, 1997), however its administration may be of (4)

Manual removal causes trauma and adds to the some benefit to counteract the effects of uterine relaxation likelihood of local infection persisting.

ants used during obstetrical procedures or Caesarean (5)

If the cow is ill, systemic treatment is sufficient to sections. Oestrogen alone has also been used (but is deal with the problem and manual removal may probably contraindicated and is no longer available for worsen the cow's condition.

use in food producing species in some countries). In

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short, ecboic drugs are unlikely to be effective after a RFM, with close monitoring of the cow so that sup-diagnosis of RFM has been made, but they may reduce portive therapy can be instigated rapidly in the small the incidence of RFM in certain groups of cows if

number of cases that develop systemic illness. Veterinary administered very soon after the delivery of the calf. nary efforts should be concentrated on preventing the high incidence of RFM at the herd level and minimizing the reproductive and economic consequences of the collagenase as an infusion into the umbilical artery; a condition.

dose of 200 000 iu of collagenase was dissolved in 1 litre of saline and infused between 24 and 72 hours after

Prevention of retained fetal membranes

parturition. The technique was reported to give an 85 per cent success rate in cases of spontaneous RFM.

General preventive measures for postpartum disease

However, no further reports of this technique

problems including RFM are discussed at the end of

have appeared in the literature and it has not been

this chapter (p. 526). With specific reference to RFM,

accepted and taken up widely. This author has no per-

attempts at prevention by routine treatment of all cows

sonal experience of this technique and feels that there

at calving are generally ineffective and impractical
is a need for further research before it could be
because of the relatively low incidence and morbidity
recommended.

of the condition. However, incidence of retention is
increased greatly when parturition is induced with cor-

Intrauterine medication: Various intrauterine medica-
tosteroids and there is some evidence that PGF_{2a} can
tions have been advocated over the years including

be an effective therapeutic agent in such cases. Gross et

intrauterine infusions of oxytetracycline (Stevens et al., al. (1986) used
dexamethasone to induce parturition in 1995). Brooks (2001) compared two
different intrau-66 cows; they injected PGF_{2a} into 40 cows within one

terine pessaries administered after manual removal of

hour of delivery and treated 26 cows with saline (con-

RFM, one containing penicillin, streptomycin and for-

trols). The incidence of retention in the PGF_{2a}-treated

mosulphathiazole and another made up of 8 per cent

cows was 8.8 per cent compared with 90.5 per cent in

iodoform in a vegetable gelatine base. He was unable

the controls. This approach to prevention is supported

to detect any difference in the efficacy of the two prod-

by findings that suggest that cows which subsequently
ucts as determined by subsequent fertility, but did not
develop RFM have lower levels of endogenous PGF2a
compare the treatment groups with negative controls.
at the time of calving than those without RFM

Intrauterine antimicrobial and antiseptic treatments
(Wischnal et al., 2001).

are probably best reserved for the treatment of the
sequels of RFM, including endometritis (see p. 521),

Effects of retained fetal membranes

rather than being seen as a treatment for RFM itself.

There is even a school of thought that advocates not

The effect of retained fetal membranes on subsequent
suppressing bacterial putrefaction of the membranes by
fertility depends on whether acute metritis, endometri-
administering local intrauterine antimicrobials as this
tis or pyometra develops or not. While there is argu-
slows the release of the membranes. Some intrauterine
ment about the best treatment at the time of retention,
medications may also interfere with the local immune
there is general agreement that it is important to

response within the uterus.

monitor cases closely until the membranes are lost.

They should also be examined again about 18–30 days

Other treatments: Other treatments such as intravenous after calving to identify and treat those cows that have

calcium borogluconate, corticosteroids, multivitamins

developed endometritis or pyometra (see pp. 521, 524).

(Laven, 1995) and oral calcium chloride gel (Hernandez

In an extensive field study with cows that retained

et al., 1999) are advocated by some. However, their use the placenta for at least 48 hours, Leslie et al. (1984) is controversial and like the more established treatment

reported that 14 per cent of the cows developed pyome-

options there is a lack of evidence to support their

tra and 15 per cent developed chronic endometritis. In

use. In some cases these treatments may actually be

four herds in which a conservative approach to treat-

detrimental and thus contraindicated.

ment was employed, metritis developed in 40 of 73

It should perhaps also be borne in mind that other

cases of retained fetal membranes (Sandals et al. , 1979).

treatments administered to the postpartum cow such as

From a study of days to service, days to conception and NSAIDs (e.g. flunixin meglumine) may even induce services per conception these authors concluded that RFM (Waelchli et al., 1999).

the condition of retained fetal membranes alone has

In summary, there is little scientific evidence to little effect on subsequent fertility. In a large field study support anything other than conservative treatment of (Halpern et al. , 1985) 45 per cent and 41 per cent of

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primiparous and pluriparous cows with retained fetal ventral shelf of tissue can be formed cranial to the membranes developed metritis. In the heifers there was urethral orifice.

no significant adverse effect of retained fetal membranes on fertility. In adult cows with no subsequent

Vaginitis

metritis, retention for five or more days caused lower pregnancy rates than controls. Retention for more than

Vaginitis is often associated with a more extensive seven days caused a long calving-to-service interval and

infection of the genital tract. However, inflammation low pregnancy rate. Borsberry and Dobson (1989), may also be associated with pneumovagina, urovagina working in the UK, reported that RFM alone extended or following trauma during parturition. In addition, the calving interval by 25 days, but when it was associated with a subsequent endometritis this increased to where natural service is practised. Infectious pustular 51 days. McDougall (2001) also reported that cows with vulvovaginitis caused by bovine herpes virus-1 RFM have lower pregnancy rates and take longer to (BHV-1) is probably the most common. However, conceive than unaffected herd-mates under New other potential pathogens such as *Ureaplasma diver-* Zealand management systems. Rowlands and Lucey sum, *Mycoplasma bovis*, *Haemophilus* (1986) reported that the lactation yield following somnus and BHV-4 have also been implicated as

retained fetal membranes was 7 per cent lower than causes of a mild granular vulvovaginitis (Cook, expected (Box 34.4). 1998).

The presenting sign of vaginitis is usually tenesmus; there may also be an abnormal vaginal or vulval

Pneumovagina and urovagina

discharge and changes in the mucosa, varying from Pneumovagina may be caused by damage to the vulva hyperaemia through necrosis to gangrene in rare cases. during parturition or inadequate closure of an episiotomy incision. Occasionally there is a more extensive retroperitonitis and septicaemia. A mild vaginitis has little effect on cause of pneumovagina or urovagina evident on subsequent fertility unless accompanied by endometritis. Both conditions are often associated with pneumovagina or urovagina. However, severe cases a history of dystokia or delivery of a large calf; the of vaginitis may cause extensive scarring leading to affected cow frequently has a BCS < 2. Pneumovagina

infertility or in some instances to dystokia at the sub- and urovagina may be unobserved until a genital tract sequent parturition.

examination or artificial insemination (AI) is per-

Treatment should be based on the severity of the

formed. Occasionally, the herdsperson will observe a

lesions and correction of predisposing factors. The

persistent dribble of urine from the vulva or hear air

vagina should be gently douched with normal saline or

being sucked through the vulval lips.

a mild antiseptic solution, following administration of

The treatment of pneumovagina is to perform a

an epidural anaesthetic. The duration of activity of the

Caslick's operation. Briefly, under epidural anaesthesia,

epidural anaesthesia can be extended by the addition

the lips of the vulva are scarified along a 1 cm wide strip

of xylazine to the anaesthetic solution (Caron & Le

from the dorsal commissure to the level of the ischial

Blanc, 1989). Douching of the vagina may be repeated

arch, leaving 2.5–3.0 cm at the ventral commisure to

on several occasions. In addition, infection of deeper

permit urination. The lips of the vagina are sutured with tissues should be treated using systemic antibiotics.

a non-absorbable material. Concurrent vaginal and/or Inflammation can be reduced and analgesia provided uterine infection is treated appropriately. Sutures are using NSAIDs.

removed in 10 to 14 days and the animal artificially inseminated at the next oestrus.

Acute puerperal metritis

There are a number of treatments for urovagina.

Some cases will respond spontaneously, particularly

Microbial contamination of the uterine lumen at par-

those cases identified before 4 to 6 weeks postpartum

turition is a normal event (Elliot et al., 1968) (see p.

and in animals that increase BCS. The return of ovarian

509), and in most cows this contamination is eliminated

cyclical activity, or possibly administration of PGF2a to

and does not result in severe infection. For this reason

induce oestrus, may help resolution in some cases.

acute puerperal metritis is relatively uncommon.

The use of AI can be successful even in the presence

However, in those cases where it does occur acute of urovagina, in other cases. In severe or long-inflammatory changes are seen in the endometrial, standing cases surgical intervention may be necessary. myometrial and peritoneal layers of the uterus within A caudal extension to the distal urethra can be contained days of calving. Pathogenic bacteria produce toxins structured using the ventral vaginal wall or a transverse and result in the development of septicaemia, toxæmia

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and possibly also pyæmia. Cows with acute puerperal (2)

The natural defence mechanisms are less effective metritis have a very serious life-threatening condition than normal. Factors that may cause this include that requires intensive treatment.

the following:

(a)

Dystokia;

(b)

Manipulation within the uterus after calving,

Diagnosis

such as inappropriate and rough attempts to

*A foetid, reddish to brown discharge flows from the
remove RFMs;*

vulva and abdominal straining is noted. Affected cows

(c)

Uterine inertia;

also commonly exhibit pyrexia, tachypnoea, tachycar-

(d)

Concomitant infection with immunosup-

pression, anorexia, rumen stasis and dehydration. Severe

pressant agents such as bovine virus diar-

rhoea virus.

rhoea virus.

develop signs of shock including a subnormal rectal

temperature. The infection may extend through the

The effect of diet before calving is unresolved; it is

uterine wall causing a localized or generalized peri-

possible that dietary energy intakes that result in fatty

tonitis. Therefore internal examination of suspected

infiltration of the liver, insufficient protein and

cases of acute metritis must be undertaken with micromineral deficiency may also impair uterine extreme care. The uterus is often friable and liable to defence mechanisms.

trauma, and therefore uterine exploration should be minimized in the acute stages of the condition. Vaginal or rectal exploration of cases of metritis often results in

Treatment and prognosis

acute discomfort and may be followed by persistent straining.

Good nursing is vitally important. The cow should be

It is probable that in the future the diagnosis and

placed in a well-bedded, comfortable warm box and

monitoring of response to treatment of acute infections

encouraged to eat and drink. Systemic broad-spectrum

such as metritis will be aided by the use of acute phase

antibiotic treatment applied in the correct dose, and

protein estimations going some way towards removing

maintained for a minimum of 5 days, is essential.

the need for internal examinations (Eckersall, 2000;

Suitable antimicrobials include cephalosporins, poten-

Sheldon et al. , 2001).

tiated amoxycillin or possibly tetracyclines. However, for good genital tract penetration postpartum oxytetracycline should be prescribed at a dose of 10 to 15 mg/kg

Aetiology

b.i.d. This 'off-label' use is above the normal recom-

The physiology of the response to normal postpartum mended dose and would require additional precautions infection suggests that there are two main factors that to be taken to prevent problems with milk or meat lead to a pathological result.

residues. See also the section on antimicrobial treatment of endometritis (p. 522) for a fuller explanation of (1)

The uterus is exposed to an unusually high the efficacy of antimicrobials in the treatment of polymicrobial challenge that includes some of the intrauterine infections.

more pathogenic organisms such as A. pyogenes, Severely affected cows should also be treated aggressively coliforms and anaerobes such as F. necrophorum

sively with intravenous fluid therapy using either high and *Prevotella melaninogenus*. In rare cases volumes (20–30 litres or more) of isotonic fluid or low *Clostridium* spp. may cause severe disease and volumes of hypertonic fluid (3 litres of 7.2 per cent rapid death. The risk of postpartum intrauterine saline) to combat the effects of the toxemia and to infection may be increased by:

reduce the development of shock. Non-steroidal anti-

(a)

Dystokia with resultant trauma inside the inflammatory drugs (NSAIDs) such as flunixin meglumine; uterus;

mine are also indicated both to provide analgesia and

(b)

The use of dirty equipment and dirty calving to reduce the effects of endotoxins (see p. 1050). It boxes;

might be expected that the administration of flunixin

(c)

The delivery of an emphysematous calf,

meglumine would inhibit normal uterine involution in resulting in the uterus being heavily infected cows with metritis, due to reduced biosynthesis of with anaerobic organisms;

PGF2a. However, at least one study suggests that the

(d)

Retained fetal membranes;

administration of flunixin meglumine to cows with

(e)

Synergistic action between infectious

metritis accelerates uterine involution and shortens the

agents, such as A. pyogenes and anaerobic

calving to first oestrus interval (Amiridis et al. , 2001).

organisms (F. necrophorum and Prevotella

If the cow is continually straining, caudal epidural

melaninogenicus).

anaesthesia can be used; it may be advantageous to

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include xylazine in the epidural in order to prolong its

Endometritis

duration of action (Ko et al. , 1989; Holden, 1998).

Introduction

Any manipulation of the uterus should be avoided while the cow is systemically ill. However, after the Endometritis is defined histologically as an inflammatory phase of the disease a technique of gentle irrigation of the endometrium. However, it is normally characterized clinically by the presence of a mucopurulent infected material in the uterus and may facilitate vaginal discharge, 21 days or more after calving, and myometrial contractions and uterine involution. This associated with delayed uterine involution. The incidence can be done using a wide diameter smooth ended tube, incidence of endometritis is about 10 per cent in dairy such as a stomach tube attached to a funnel. The operator (taking extreme care not to traumatize the uterus) However, there is wide variation in the incidence of passes the tube through the cervix and pours in several endometritis between herds, ranging from a few cases litres of a flushing solution made up of warm sterile

to more than 40 per cent of the herd affected.

normal saline. The funnel is then lowered below the level of the uterus and the fluid along with debris from the uterus is siphoned out. This procedure can be repeated several times to remove as much as possible of the uterine contents (Parkinson, 2001). While this procedure disrupts the delicate hormonal milieu of the hypothalamic–pituitary–ovarian axis (see p. 489; Chapter 67) and disrupts follicular growth and development.

removal of the uterine contents in this way seems to be associated with an increased incidence of cystic ovarian disease. Uterine infection has been reported to be associated with an increased incidence of cystic ovarian disease. authors, it should be noted that controlled studies to evaluate this form of therapy are lacking. Furthermore, the presence and persistence of pathogenic organisms causing endometritis is assumed to preclude the establishment of pregnancy. Thus,

*be punctured or damaged and irrigation fluid may flow
endometritis has a detrimental effect on fertility,
in a retrograde direction up the oviducts, resulting in
extending the calving to conception interval, and
salpingitis and adhesions of the ovarian bursae.*

*increasing the number of services per conception and
Many clinicians introduce antibiotics into the uterus
the proportion of culls for failure to conceive.*

*once the cow has responded to parenteral treatment,
The economic cost of endometritis depends on the
but the points discussed regarding the use of intrauter-
detrimental effect on fertility, increased culling rate
ine antibiotics for the treatment of endometritis (see p.
and, to a lesser extent, the cost of treatment. The esti-
522) should be borne in mind.*

mated direct cost of a case of vulval discharge was £71

*With acute metritis the prognosis refers to the possi-
for treatment and a 300 litre reduction in milk yield
bility of death, adverse effect on health, milk yield and
(Kossaibati & Esslemont, 1997). However, the total cost
future reproductive function. Most cases make a fairly*

was £160, including an increased calving interval of 18 days and an increase of 0.3 services per conception. In the worst affected cases, if the cow survives the acute metritis episode

Aetiology

treatment can be considered a reasonable success.

It is assumed that the uterus and its contents are sterile during a normal pregnancy and prior to parturition.

Consequences of metritis

Then at the time of parturition or just afterwards the

A small proportion of metritis cases develop embolic uterine lumen becomes contaminated by micro-organisms from the animal's environment, skin and faeces, a consequence of a bacteraemia or laminitis following through the relaxed perineum, vulva and dilated cervix.

the toxaemia. In those cases that do respond to treat-

There are a variety of predisposing factors for

ment, after recovery there may be adhesions affecting endometritis (Box 34.5). Cows with a uterine infection

the uterus, the oviducts and ovarian bursae, resulting in associated with A. pyogenes more than 21 days post-reduced fertility. The majority of cases of acute metritis will also progress to endometritis.

invariably subfertile to the first service. In addition,

In a meta-analysis of papers published since 1960,

there is synergism between A. pyogenes, F. necrophorum and Prevotella melaninogenica was shown to be associated with a seven-day

increase in time to first service and a 20 per cent

severe cases of endometritis. Interference with uterine reduction in conception rate to first service, resulting in defence mechanisms such as uterine involution or neutrophil function will delay the elimination of bacterial contamination. Dystokia, the delivery of twins or a

a delay to conception of 19 days (Fourichon et al. , 2000).

contamination. Dystokia, the delivery of twins or a

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Box 34.5.

Predisposing factors for endometritis.

to assess the degree of uterine and cervical involution,

and the nature of the vaginal discharge. The majority of

- *Retained fetal membranes*

systems use a combination of uterine horn and cervical

- *Dystokia and/or assistance at calving*

diameter and a score for the contents of the vagina (Fig.

- *Caesarian section*

34.4) (Sheldon & Noakes, 1998).

- *Twins*

- *Stillbirths*

- *Induced parturition*

Treatment

- *Dirty calving environment*

- *Postpartum ovarian inactivity*

The treatment of endometritis has caused considerable

- *Diet—possibly vitamin E/selenium deficiency*

debate and has been reviewed extensively in the liter-

ature (Bretzlaff, 1987; Sheldon, 1999). The value of

treatment of clinical cases has not been accepted by

all clinicians, despite evidence that cows administered a

dead calf and assistance at calving increase the oppor-

placebo, rather than an accepted therapy, had longer

tunity for faecal contamination of the genital tract.

intervals from calving to conception and higher culling

Retained fetal membranes are a predisposing factor for

rates (Steffan et al. , 1984). Treatment success rates are endometritis and are associated with an increase in

higher for mild cases of endometritis compared with

severity of endometritis (Sheldon & Noakes, 1998).

severe cases and the success rate is reduced if the

Uterine infection has a seasonal incidence, being

vaginal discharge has a foetid odour.

highest during the housed period, presumably because

The three treatments most often used are parenteral

of environmental contamination. A dirty calving envi-

PGF2a or analogues (see p. 524), oestrogen (see p. 523)

ronment may increase the risk of endometritis. Noakes

and intrauterine antibiotic. There are a limited number

et al. (1991) described two hygienically contrasting

of products licensed for endometritis; in addition, a nil

farms: on one with a relatively clean environment the

milk withholding period is usually demanded in dairy

incidence of endometritis was 2–3 per cent, compared

cattle.

with an incidence of 15 per cent for one with a dirty environment. However, there was no difference in the Antimicrobial treatment: A number of antimicrobial quantitative or qualitative uterine bacterial flora in selection criteria have been suggested for endometritis cows calved on either farm.

including efficacy against the causal organism in the A delay in the return of cyclical ovarian activity after uterine environment, but without inhibiting natural calving appears to predispose to endometritis. Con- uterine defence mechanisms. Fluid and tissue debris versely, if the interval from calving to first ovulation is reduce the effect of sulphonamides, aminoglycosides too short, it has been suggested that pyometra could and nitrofurazones, and aminoglycosides are ineffective occur because *A. pyogenes* and Gram-negative anaero- in an anaerobic environment, such as the uterine lumen. bic bacteria would remain within the uterus after ovu- The chosen antibiotic must reach appropriate concen- lation, allowing bacterial growth to continue following

trations at the site of infection and persist a sufficient corpus luteum formation (Olson et al. , 1984).

length of time to eliminate the infection. At less than 30 days postpartum, mixed bacterial infections may render intrauterine infusion of penicillin ineffective,

Diagnosis and assessment

due to penicillinase produced by bacteria. In contrast, Clinically, endometritis is characterized by the presence of a mucopurulent discharge in the vagina, associated with delayed uterine involution. The definitive diagnosis of endometritis would be made based on histological examination of endometrial biopsies. However, oxytetracycline and cephalosporins are broad spectrum, and are effective in the uterine lumen in the presence of pus and reduced oxygen tension. In addition, pharmaceutical preparations of oxytetracycline and cephalosporins produce effective antimicrobial concentrations in the endometrium, but with minimal concentrations in the genital tract per rectum are the most trations in milk. Any milk withdrawal period would

useful techniques for the diagnosis of endometritis. make a treatment for endometritis unattractive under Visual or manual examination of the vagina for an practice conditions; for example, parenteral tylosin gave abnormal uterine discharge is essential for the diagnosis of endometritis, although the contents of the vagina uterine lumen, but has a significant milk withdrawal do not always reflect the contents of the uterus. Flakes period (Cester et al. , 1993). Metronidazole and chlo- of pus in the vagina could come from the uterus, cervix ramphenicol are prohibited in food producing animals or vagina and slightly cloudy mucus is often regarded in the UK. The use of the intrauterine route of administration has prevailed for many years and the pharma-

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Clinical sign

Point score

Smell

Foul

3

No smell

0

Bloody

3

Vaginal discharge

>50 ml pus

3

Character

< 50ml pus

2

Flecks

1

Normal

0

External diameter

Pregnancy

Primiparous

Multiparous

of largest uterine

horn

Large

> 5.5 cm

> 6.0 cm

2

Medium

3.5 to 5.5 cm

4.0 to 6.0 cm

1

Normal

< 3.5 cm

< 4.0 cm

0

Pregnancy

Primiparous

Multiparous

External diameter

Large

> 7.0 cm

> 7.5 cm

2

of cervix

Medium

4.5 to 7.0 cm

5.0 to 7.5 cm

1

Normal

< 4.5 cm

< 5.0 cm

0

Clinical assessment from total score

Severe

8–10

Moderate

4–7

Mild

1–3

Normal

0

Fig. 34.4

Scoring system for the assessment of the severity of endometritis, suitable for use from 21 days postpartum.

ceutical industry continues to develop antibiotic prod-

A. pyogenes when either endogenous or exogenous ucts administered by this route, despite the widespread oestrogen was present compared to during dioestrus, use of systemic antibiotics for treatment of other bacterial infections in cattle.

therapeutically. Despite the potential of oestrogens for the treatment of endometritis, results have been equiv-

Hormones: The hormonal treatment of endometritis ocal and vary with the pharmaceutical preparation and is based upon observations that the genital tract is dose administered. The treatment-to-conception interval more susceptible to infection when under progesterone val was longer for cases of endometritis treated with a dominance and more resistant under the influence of single intramuscular injection of 3 mg oestradiol ben-oestrogens (Rowson et al. , 1953).

zoate per 500 kg body weight, compared with those

Oestrogens: Rowson et al. (1953) demonstrated that treated using prostaglandin or intrauterine oxytetracycline the bovine uterus was more resistant to infection with cline (Sheldon & Noakes, 1998). Furthermore, regula-

tory authorities have prohibited the use of oestrogens appear ill; however, they are anoestrus and may be pre-in several countries.

sented to the veterinarian as 'not-seen-bulling' or for

Prostaglandins: The parenteral application of pregnancy diagnosis.

PGF_{2a}, or its analogues, has been used widely for the

Pyometra may be a sequel to the conditions discussed treatment of endometritis. The majority of authors

earlier in this chapter and therefore those factors that

report that the presence of an active corpus luteum is

predispose a cow to RFM, metritis and endometritis can

required for optimal results when using prostaglandin

also be considered to predispose to pyometra. In addi-

to treat endometritis, which supports the hypothesis

tion pyometra may also be seen in cows after service

that the subsequent induced oestrus increases the

(either natural or by AI).

uterine resistance to bacteria (Jackson, 1977; Sheldon &

In most cases pyometra is a sequel to chronic

Noakes, 1998). Conversely, Pepper and Dobson (1987) reported that the efficacy of prostaglandin therapy for endometritis as the uterus ceases to release the endogenous luteolysin PGF₂a. Research has shown that the endometritis did not depend on the presence of an active CL, as determined by measuring milk progesterone concentrations. An alternative hypothesis is associated with inadequate secretion of luteal oxytocin for the mechanism of action of prostaglandin as a treatment (Vighio et al., 1991). The result is that the CL persists and the genital tract remains closed, physical expulsion of uterine detritus. under the influence of progesterone. The infection is not eliminated and the cervix remains closed, thus not eliminated and the cervix remains closed, possibility of using the direct myometrial effect of prostaglandin therapy, to reduce the incidence of

purulent exudate in the uterine lumen. Occasionally endometritis and improve subsequent fertility. A there will be slight leakage through the cervix, routine injection of prostaglandin shortened the resulting in a small amount of pus accumulating in the calving-to-conception interval in normal cows compared with control animals (Etherington et al. , 1988). discharge. In a small number of cases the persistent However, a number of subsequent trials gave conflicting results. Meta-analysis of 24 trials where PGF2a was rather than a persistent CL. In some instances the introduced to cattle within 40 days of calving reduction of infection during dioestrus, such as occurs reported no beneficial effect on first-service pregnancy when a cow is artificially inseminated at an incorrect rate, although there was a small reduction in days open time, results in pyometra. Pyometra may also occur for treated cows (Burton & Lean, 1995). secondary to the death of a fetus, either caused by, or

followed by, bacterial invasion by *A. pyogenes* and
Other treatments: A variety of antiseptics have been
persistence of the CL of pregnancy. Infection with
administered by intrauterine infusion for the treat-
Trichomonas fetus also causes embryonic death and
ment of endometritis including solutions of Lugol's
pyometra formation (Bon-Durant, 1997).

iodine, polyvinyl pyrrolidone iodine, povidone-iodine,
When examined per rectum, cases of pyometra have
chlorhexidine and metakresol sulphonic acid. Many of
enlarged uterine horns and must be accurately differ-
these solutions cause a necrotic endometritis within 24
entiated from a normal pregnancy before treatment can
hours of infusion that may stimulate release of PGF_{2a}
be administered. There are a number of distinguishing
in addition to possible direct effects on pathogenic
points:

organisms. However, despite clinical resolution of

- The uterus feels 'doughy' and the uterine wall is

the endometritis using antiseptic solutions, the effect on
thicker than during pregnancy.

fertility may not be beneficial. Nakao et al. (1988)

- *It is not possible to palpate the amniotic vesicle or administered a 2 per cent polyvinyl pyrrolidine iodine 'slip' the allantochorion membranes.*

intrauterine infusion and reported that treated cows,

- *Placentomes are not palpable or visible on ultra- particularly those with a purulent discharge, had an sound examination, although the caruncles of the increased calving-to-conception interval compared with previous pregnancy are occasionally detected.*

controls. In addition, some of these antiseptic products

- *Ultrasonography will demonstrate the absence of a are not licensed for use in food producing animals.*

fetus and the presence of pus rather than normal fetal fluids.

Pyometra

The best and most appropriate treatment of pyometra

Pyometra is associated with the accumulation of pus in is to bring about luteolysis with PGF2a or PGF2a the uterus and by the persistence of functional luteal analogues. However, this treatment should never be

tissue on one or both ovaries. Affected cows do not administered if there is any suspicion that the cow

The Postpartum Period • 525

may be pregnant, as it will cause abortion in early >1.0 BCS points during the first 30 days after calving pregnancy.

take longer to the first postpartum ovulation than those

The administration of PGF2a to cases of pyometra losing less than one unit (Beam & Butler, 1999).

usually results in dilation of the cervix and expulsion of

However, monitoring BCS over periods greater than 6

the purulent uterine contents, with oestrus occurring

weeks postpartum often has a less clear association with

three to five days later. There is evidence to suggest that

the length of the anoestrus period. The pituitary secre-

treatment of pyometra is improved by the administra-

tion of FSH appears to be insensitive to NEB. Thus, suc-

tion of two doses of PGF2a 8 hours apart (Archbald

cessive waves of follicular growth start within 10 days

et al., 1993). This is probably due to the fact that a single of calving. However, in the anoestrus cows, dominant

dose of exogenous PGF2a has only a very short duration of action, as the inadequate release of ovarian oxy-pulse frequency. Insulin-like growth factor-1 (IGF-1) is a likely hormonal mediator between nutrition and fertility. However, the precise mechanisms and how they may be modulated are not yet clear. Shortening the PGF2a.

postpartum interval to the NEB nadir is a more tangible management objective to reduce the incidence of pyometra go on to become pregnant, despite having a prolonged first luteal phase and a pro-anoestrus in a herd.

longed calving to pregnancy interval of 125 days com-

The suckling calf inhibits the release of GnRH from

pared to 74.8 days (Etherington et al., 1991). However, the dam's hypothalamus, so suppressing LH pulse frequency. However, this inhibitory influence occurs as a

degeneration, which may increase the rate of late consequence of the maternal bond between cow and embryonic and early fetal losses. Comparison between calf, and it is not dependent upon sensory innervation pregnancy rates at first pregnancy diagnosis 33 to 70 of the udder (Williams & Griffith, 1995). The frequency days postservice and a subsequent diagnosis 120 to 150 and duration of suckling by a single calf are unrelated days postservice have shown that attrition rates are 2.6 to the period of anoestrus, although suckling following times higher in cows with previous pyometra than in adoption of an additional calf will delay the return of cows without the condition (López-Gatius et al., 1996). ovarian cyclicity. If the single calf is removed from its dam there is a corresponding increase in the frequency of LH pulses within two to six days. The inhibition of

Postpartum anoestrus

GnRH release is dependent on the cow being suckled

Introduction

by her 'own' calf, which she identifies by sight and smell.

The mechanisms underlying this effect are unclear; in

Postpartum anoestrus is a greater problem for beef part, they may involve increased opioid tone within the cattle than dairy cows, and it is often evident in specific brain.

herds.

Diagnosis

Aetiology

At least two examinations, 10–14 days apart, are

The two main predisposing factors are inadequate required to make a clinical diagnosis of anoestrus. Both nutrition, specifically energy deficiency, and suckling or ovaries are small and there is no CL detectable at either the maternal bond. Other associated factors are that it of the examinations; however, follicles up to 18 mm is more common during late winter, in the absence of a diameter may be identified. An alternative or additional method of diagnosis is to use milk progesterone associated with pituitary or ovarian damage.

analysis; two low values, at 10–14-day intervals, is

Negative energy balance (NEB) is common to most

indicative of anoestrus.

postpartum cows and is the difference between the dietary intake of metabolizable energy and the expenditure for maintenance and lactation (see p. 103). Consequently, the balance between milk yield and

The most important consideration when presented with feeding (including diet composition, feed supply and an anoestrus animal is to correct the underlying causes, appetite) is an important determinant of the severity of in particular dietary deficiencies. The quantity and NEB. However, in first lactation an animal's metabolizable energy may also have to contribute to continued milk yield should be determined. In addition, dry-cow growth. The effect of NEB is most easily identified by management should be examined, paying particular monitoring body condition score (BCS). Cows that lose attention to the transitional period. Monitoring BCS

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during the dry period and early lactation will help to

Control of these conditions is not simply a matter of quantify the severity of dietary energy deficiency. Two good management at the time of calving.

groups of pharmaceutical products may be used to treat anoestrus: GnRH and analogues, and progestagens.

Breeding decisions and the

GnRH: The pituitary LH content and responsiveness management of the pregnant

to GnRH are complete by day 10 postpartum and so and periparturient cow

exogenous GnRH administered to the anoestrus cow will induce a transient LH and FSH release. However, The most important way of reducing puerperal problems is to avoid dystokia. Where dystokia does occur it ovarian follicular wave. If there is a dominant follicle

is essential that it is dealt with correctly at an early stage present, ovulation will occur. So, when daily ultrasound

and that any developing problems observed after monitoring was used to identify a suitable dominant calving are also treated as soon as possible. Attention

follicle, administration of GnRH consistently induced

to all aspects of hygiene is also a basic requirement.

ovulation (Roche et al., 1992). However, the majority of If it were purely a veterinary matter it would be rel-animals did not express oestrous and had short luteal

atively simple to reduce the incidence of dystokia in the

phases after ovulation.

Gonadotrophin releasing

majority of herds. However, the measures required may

hormone administered in the absence of a dominant

not be acceptable to the farmer. Simple modifications

follicle is ineffective, so overall blind-treatment success

such as improved supervision of calving and better

rates are variable.

housing may be judged to be too expensive. Selecting

an easy calving breed of bull or individual bull within a

Progesterone: Administration of progesterone, using

breed may not produce calves with the desired confor-

an intravaginal device or norgestamet by injection and

mation or genetic potential.

implantation, is an alternative treatment for the

The consequences of several synergistic adverse

anoestrus cow. In the absence of severe NEB, once the effects tend to be particularly serious. Therefore, if a progesterone treatment is removed, there is an induced client chooses to use a bull that is liable to produce a ovulation and such treatment overcomes the absence of high rate of dystokia it is particularly important to avoid behavioural oestrous that often occurs at the first post-any other factor that predisposes to dystokia.

partum ovulation. However, if the NEB is severe, prog- Application of the following precautions will reduce esterone treatment requires supplementation with the incidence of dystokia in a herd.

400–700 iu equine chorionic gonadotrophin (eCG) by intramuscular injection at the time of implant removal.

(1)

Selection of sires that produce calves of suitable

An alternative to eCG is the administration of 1 mg size and conformation for the dams concerned.

oestradiol benzoate 24 hours after a seven-day proges-

This refers to the breed and to the individual bull

terone treatment which increased oestrus expression

within the breed. Special care is needed for heifers and ovulation (MacMillan et al. , 1995).

(see Chapter 5).

(2)

Induction of premature calving, if the fetus is likely to be too large at full term for a normal

Cystic ovarian disease

calving (see p. 687).

Cystic ovarian disease can be a major problem in the

(3)

Control of energy intake and avoidance of postpartum dairy cow resulting in an increase of 6 to 11 macromineral and trace element imbalances.

days to first service and an increase in the calving-to-

(a)

Do not let the dams get too fat, as the conception interval of 20–30 days (Fourichon et al., perirectal and perivaginal fat will narrow the 2000). However, this subject is dealt with elsewhere in birth canal, the dam may be less capable of this book in Chapters 35 and 36.

forceful labour and the calf may also be larger. This is also an important way of avoiding necrotic vaginitis and other damage

Prevention of postpartum

to the vagina. At calving a condition score of

disease problems

2.5 (see p. 10) is advised.

(b)

Avoid undernutrition as cows in very poor

The key to controlling postpartum disease problems is condition also have problems of dystokia.

to recognize that they are interlinked (Peeler et al. , (c)

Regular body condition scoring and careful

1994) and that their control is related to breeding man-

attention to the late lactation, dry period and

agement, nutrition, the management of the transition

particularly the transition period ration are

cow and management at and immediately after calving.

required. This may be aided by the use of

The Postpartum Period • 527

metabolic profiles (see Chapter 47). Specific

tum problems. The preparation of cattle for breeding dietary manipulations such as the use of begins well before the expected service date. Preparing salts to manipulate the dietary cation–anion the cow for parturition and particularly dry cow and balance (DCAB) of the transition cow ration transition cow management in the previous gestation may also be useful in reducing the incidence period are crucial stages in this preparation. However, of periparturient problems (see p. 787).

parturition is the key event, and it must be managed
(4)

Provision of a suitable environment, especially for to minimize the development of abnormalities of the cows that calve inside or in a restricted area. This genital tract in the postpartum period, which may lead should be clean, well bedded, with a good water to problems of subsequent oestrous cyclicity, depression supply, properly lit and with enough space to get in conception rates, increased embryonic loss and at the cow if need be and to allow observation

subfertility.

without disturbance. There also need to be facilities to safely restrain the cow.

References

(5)

Avoid stress and disturbance of the calving cow

(Hindson, 1976).

Adams, G.P., Matteri, R.L., Kastelic, J.P., Ko, J.C.H. &

(6)

Suitable supervision and timely intervention at

Ginther, O.J. (1992) Association between surges of follicle-

calving will reduce the severity of those cases of

stimulating hormone and the emergence of follicular waves

dystokia that do occur (Hodge et al. , 1982).

in heifers. Journal of Reproduction and Fertility, 94, 177–88.

(7)

Proper training of the farmer and stockworkers in

Amiridis, G.S., Leontides, L., Tassos, E., Kostoulas, P. & how to assist at calving should be supplied by the

Fthenakis, G.C. (2001) Flunixin meglumine accelerates

veterinarian.

uterine involution and shortens the calving-to-first-oestrus

(8)

When veterinary assistance is needed, the farmer

interval in cows with puerperal metritis. Journal of Veteri-and his staff must decide at an early stage to call

nary Pharmacology and Therapeutics, 24, 365–7.

for help. It is important that the veterinarian and

Archbald, L.F., Risco, C., Chavatte, P., Constant, S., Tran, T., the farmer should agree before the start of the

Klapstein, E. & Elliot, J. (1993) Estrus and pregnancy rate calving season how these problems will be dealt

of dairy cows given one or two doses of prostaglandin F2

alpha 8 or 24 hours apart. Theriogenology, 40, 873–84.

with and at what stage the farmer will call for

Beam, S.W. & Butler, W.R. (1997) Energy balance and ovarian professional help.

follicle development prior to the first ovulation postpartum

in dairy cows receiving three levels of dietary fat. Biology of Reproduction, 56, 133–42.

Therapy immediately postpartum

Beam, S.W. & Butler, W.R. (1999) Effects of energy balance on follicular development and first ovulation in postpartum

In addition to specific therapies such as those described

dairy cows. Journal of Reproduction and Fertility Supple-

above for uterine prolapse, cows that have had obstet-
ment, **54**, 411–24.

rical interventions at calving may benefit from a course

Bon-Durant, R. (1997) Pathogenesis, diagnosis and manage-
of systemic antibiotic.

ment of trichomoniasis in cattle. *Veterinary Clinics of North America* Metabolic diseases,
such as milk fever, should be

American Journal of Veterinary Medicine, **13**, 345–61.

Borsberry, S. & Dobson, H. (1989) Periparturient diseases and dealt with
appropriately and if these cases are common,

their effect on reproductive performance in five dairy herds.

investigation into their causes is essential. This aspect

Veterinary Record, **124**, 217–19.

of bovine medicine is dealt with elsewhere in this book

Bretzlaff, K. (1987) Rationale for treatment of endometritis
(Chapter 46).

in the dairy cow. *Veterinary Clinics of North America*: Food The routine
administration of agents such as PGF_{2a}

Animal Practice, **3**, 593–607.

(see p. 524) and oxytocin (see p. 517) to postpartum

Brooks, G. (2001) Comparison of two treatments after
cows is not justified by improvements in subsequent fer-

retained fetal membranes on clinical signs in cattle. Veterinary; reports of the benefits in the published literature

nary Record, 148, 243–4.

are inconsistent. Their use postpartum should therefore

Bulman, D.C. & Lamming, G.E. (1978) Milk progesterone

be restricted to animals where a specific indication

levels in relation to conception, repeat breeding and factors

exists, as described earlier in this chapter.

influencing acyclicity in dairy cows. Journal of Reproduction and Fertility, 54, 447–58.

Burton, N.R. & Lean, I.J. (1995) Investigation by meta-analysis of the effect of prostaglandin F2a administered

Conclusions

post partum on the reproductive performance of dairy

cattle. Veterinary Record, 136, 90–4.

The key to good fertility in multiparous cattle is to

Caron, J.P. & Le Blanc, P.H. (1989) Caudal epidural analgesia maintain a high level of stockmanship at all times, and

in cattle using xylazine. Canadian Journal of Veterinary

to manage cattle in such a way as to minimize postpar-

Research, 53, 486–9.

Cester, C.C., Ganiere, J.P. & Toutain, P.L. (1993) Effect of Holden, D. (1998) Local analgesia for surgery of the head,

stage of oestrous cycle on tylosin disposition in genital

distal limbs and mammary gland. *Cattle Practice*, **6**, tract secretions of cows. *Research in Veterinary Science*, **54**, 233–6.

32–9.

Hooper, R.N., Crabill, M.R., Taylor, T.S. & Roussel, A.J. (1999) Cook, N. (1998) Bovine herpes virus 1: clinical manifestations Managing vaginal and cervical prolapses in cows. *Veterinary and an outbreak of infectious pustular vulvovaginitis in a*

Medicine, **94**, 375–89.

UK dairy herd. *Cattle Practice*, **6**, 341–4.

Jackson, P.S. (1977) Treatment of chronic post partum

Correa, M.T., Erb, H.N. & Scarlett, J.M. (1992) A nested case-endometritis in cattle with cloprostenol. *Veterinary Record*, control study of uterine prolapse. *Theriogenology*, **37**, **101**, 441–3.

939–45.

Joosten, I., Stelwagen, J. & Dijkhuizen, A.A. (1988) Economic Darwash, A.O., Lamming, G.E. & Woolliams, J.A. (1997) The

and reproductive consequences of retained placenta in

phenotypic association between the interval to post-partum

dairy cattle. *Veterinary Record*, **123**, 53–7.

ovulation and traditional measures of fertility in dairy

Kaidi, R., Brown, P.J. & David, J.S.E. (1991) Uterine involu-cattle. *Animal Science*, **65**, 9–16.

tion in cattle. In *The Veterinary Annual* (ed. by C.S.G.

Eckersall, P.D. (2000) *Acute phase proteins as markers of*

Grunsell & M.E. Raw), Vol. 31, pp. 38–50. Blackwell

infection and inflammation: monitoring animal health,

Scientific Publications, Oxford.

animal welfare and food safety. Irish Veterinary Journal, **53**, Ko, J.C.H.,

Althouse, G.C., Hopkins, S.M., Jackson, L.L., Evans, 307–11.

L.E. & Smith, R.P. (1989) *Effects of epidural administration* Eiler, H. & Hopkins,

F.M. (1993) *Successful treatment of*

of xylazine or lidocaine on bovine uterine motility and per-

retained placenta with umbilical cord injection of collage-

ineal analgesia. Theriogenology, **32**, 779–86.

nase in cows. Journal of the American Veterinary Medical

Kossaibati, M.A. & Esslemont, R.J. (1995) *Daisy. The DAIRY*

Association, **203**, 436–43.

Information System Report No. 4 – Wastage in Dairy Herds.

Elliot, L., McMahon, K.J., Gier, H.T. & Marion, G.B. (1968) *University of Reading.*

Uterus of the cow after parturition: bacterial content.

Kossaibati, M.A. & Esslemont, R.J. (1997) *The cost of pro-*

American Journal of Veterinary Research, **29**, 77–81.

duction diseases in dairy herds in England. The Veterinary Etherington, W.G.,

Christie, K.A., Walton, J.S., Leslie, K.E., *Journal*, **154**, 41–51.

Wickstrom, S. & Johnson, W.H. (1991) Progesterone profiles Laven, R.A. (1995)
The treatment of retained placenta: a

in postpartum holstein dairy cows as an aid in the study

survey of practitioners. Cattle Practice, 3, 267–79.

of retained fetal membranes, pyometra and anestrus.

Laven, R.A. & Peters, A.R. (1996) Bovine retained placenta: *Theriogenology*,
35, 731–46.

aetiology, pathogenesis and economic loss. Veterinary

Etherington, W.G., Martin, S.W., Bonnett, B., Johnson, W.H.,

Record, 139, 465–71.

Miller, R.B., Savage, N.C., Walton, J.S. & Montgomery, M.E.

Leslie, K.E., Doig, P.A., Bosu, W.T.K., Curtis, R.A. & Martin, (1988)
Reproductive performance of dairy cows following

S.W. (1984) Effects of gonadotrophin releasing hormone

treatment with cloprostenol 26 and/or 40 days postpartum:

on reproductive performance of dairy cows with retained

a field trial. Theriogenology, 29, 565–75.

placenta. Canadian Journal of Comparative Medicine, 48, Fourichon, C.,
Seegers, H. & Malher, X. (2000) Effect of

354–9.

disease on reproduction in the dairy cow: a meta-analysis.

López-Gatius, F., Labèrnia, J., Santolaria, P., López-Béjar, M.

Theriogenology, 53, 1729–59.

& Rutllant, J. (1996) *Effect of reproductive disorders*

Ginther, O.J., Wiltbank, M.C., Fricke, P.M., Gibbons, J.R. & previous to
conception on pregnancy attrition in dairy

Kot, K. (1996) *Selection of the dominant follicle in cattle.*

cows. *Theriogenology*, **46**, 643–8.

Biology of Reproduction, **55**, 1187–94.

McDougall, S. (2001) *Effects of periparturient diseases and*

Gross, T.S., Williams, W.F. & Moreland, T.W. (1986) *Prevention conditions on
the reproductive performance of New*

of the retained fetal membrane syndrome (retained pla-

Zealand dairy cows. New Zealand Veterinary Journal, **49**, centa) during induced
calving in dairy cattle. *Theriogenol*-60–7.

ogy, **26**, 365–70.

MacMillan, K.L., Taufa, V.K., Day, A.M. & McDougall, S.

Halpern, N.E., Erb, H.N. & Smith, R.D. (1985) *Duration of*

(1995) *Some effects of using progesterone and oestradiol*

retained fetal membranes and subsequent fertility in dairy

benzoate to stimulate oestrus and ovulation in dairy

cows. *Theriogenology*, **23**, 807–13.

cows with anovulatory anoestrus. Proceedings of the New

Hernandez, J., Risco, C.A. & Elliott, J.B. (1999) *Effect of oral Zealand Society
of Animal Production*, **55**, 239–41.

administration of a calcium chloride gel on blood mineral

Markusfeld, O. (1984) Factors responsible for post parturient concentrations, parturient disorders, reproductive performance, and milk production of dairy cows with retained fetal membranes. *Veterinary Record*, **114**, 539–42.

Mee, J.F. (1991) The incidence of retained fetal membranes on nine dairy research farms over a ten-year period. *Journal of the American Veterinary Medical Association*, **215**, 72–6.

(1978–1987). *Irish Veterinary Journal*, **44**, 48–52.

Hindson, J.C. (1976) Retention of the fetal membranes in cattle. *Veterinary Record*, **99**, 49–50.

Meursinge, J.A. & Cooper, M.G. (1967) Retained fetal membranes in dairy herds in the Huntly district. *New Zealand Veterinary Journal*, **15**, 111–16.

Hodge, P.B., Wood, S.J., Newman, R.D. & Shepherd, R.K. (1982) Effect of calving supervision upon the calving performance of Hereford heifers. *Australian Veterinary Journal*, **58**, 97–100.

partum intrauterine treatment with 2% polyvinylpyrrolid-

done-iodine solution on reproductive efficiency in cows.

Savio, J.D., Boland, M.P., Hynes, N. & Roche, J.F. (1990)

Theriogenology, **30**, 1033–43.

Resumption of follicular activity in the early postpartum

Noakes, D.E. (2001a) Injuries and diseases incidental to par-

period of dairy cows. Journal of Reproduction & Fertility, turition. In Arthur's Veterinary Reproduction and Obstetrics **88**, 569–79.

(ed. by D.E. Noakes, T.J. Parkinson, & G.C.W. England), 8th Savio, J.D., Keenan, L., Boland, M.P. & Roche, J.F. (1988) edn, pp. 319–32. W.B. Saunders, London.

Pattern of growth of dominant follicles during the oestrous

Noakes, D.E. (2001b) Postparturient prolapse of the uterus.

cycle of heifers. Journal of Reproduction and Fertility, **83**, In Arthur's Veterinary Reproduction and Obstetrics (ed. by 663–71.

D.E. Noakes, T.J. Parkinson, & G.C.W. England), 8th edn,

Sheldon, I.M. (1999) Bovine endometritis: a review. Journal of pp. 333–8. W.B. Saunders, London.

Animal Breeding, **2**, 2–14.

Noakes, D.E., Wallace, L. & Smith, G.R. (1991) Bacterial flora Sheldon, I.M. & Noakes, D.E. (1998) Comparison of three

of the uterus of cows after calving on two hygienically con-

treatments for bovine endometritis. Veterinary Record, **142**, trasting farms. *Veterinary Record*, **128**, 440–2.

575–9.

Oakley, G.E. (1992) *Survival and fertility of dairy cows fol-*

Sheldon, I.M., Noakes, D.E. & Dobson, H. (2000) The influ-

lowing uterine prolapse. New Zealand Veterinary Journal, ence of ovarian activity and uterine involution determined

40, 120–2.

by ultrasonography on subsequent reproductive perfor-

Olson, J.D., Ball, L., Mortimer, R.G., Farin, P.W., Adney, W.S.

mance. Theriogenology, 54, 409–19.

& Huffman, E.M. (1984) Aspects of bacteriology and

Sheldon, I.M., Noakes, D.E., Rycroft, A. & Dobson, H.

endocrinology of cows with pyometra and retained fetal

(2001) Acute phase protein responses to uterine bacterial

membranes. American Journal of Veterinary Research, 45, contamination in cattle after calving. Veterinary Record, 2251–5.

148, 172–5.

Parkinson, T.J. (2001) Infertility in the cow: structural and

Smith, M.C.A. & Wallace, J.M. (1998) Influence of early post functional abnormalities, management deficiencies and

partum ovulation on the re-establishment of pregnancy in

non-specific infections. In Arthur's Veterinary Reproduction multiparous and primiparous dairy cattle. Reproduction

and Obstetrics (ed. by D.E. Noakes, T.J. Parkinson, & Fertility Development, 10,

207–16.

G.C.W. England), 8th edn, pp. 383– 472. W.B. Saunders,

Steffan, J., Adriamanga, S. & Thibier, M. (1984) *Treatment of London.*

metritis with antibiotics or prostaglandin F2a and influence

Peeler, E.J., Otte, M.J. & Esslemont, R.J. (1994) *Inter-*

of ovarian cyclicity in dairy cows. American Journal of

relationships of periparturient disease in dairy cows.

Veterinary Research, **45**, 1090–94.

Veterinary Record, **134**, 129–32.

Stevens, R.D. & Dinsmore, R.P. (1997) *Treatment of*

Pepper, R.T. & Dobson, H. (1987) *Preliminary results of treat-dairy cows at parturition with prostaglandin F2a or oxy-*

ment and endocrinology of chronic endometritis in the

tocin for prevention of retained fetal membranes. Journal

dairy cow. Veterinary Record, **120**, 53–6.

of the American Veterinary Medical Association, **211**, Peters, A.R. & Laven, R.A. (1996) *Treatment of bovine*

1280–84.

retained placenta and its effects. Veterinary Record, **139**, Stevens, R.D., Dinsmore, R.P. & Cattell, M.B. (1995) *Evalua-*535–9.

tion of the use of intrauterine infusions of oxytetracycline,

Peters, A.R. & Riley, G.M. (1982) *Milk progesterone profiles subcutaneous injections of fenprostalene, or a combination*

and factors affecting post partum ovarian activity in beef of both, for the treatment of retained fetal membranes in cows. *Animal Production*, **34**, 145–53.

dairy cows. *Journal of the American Veterinary Medical*

Plenderleith, B. (1986) Prolapse of the uterus in the cow. In Association, 207, 1612–15.

Practice, 8, 14–15.

Stocker, H. & Waelchli, R.O. (1993) A clinical trial on the Roche, J.F. & Boland, M.P. (1991) Turnover of dominant fol-effect of prostaglandin F2a on placental expulsion in dairy

lices in cattle of different reproductive states. Theriogenol-cattle after a caesarean operation. Veterinary Record, 132, ogy, 35, 81–90.

507–508.

Roche, J.F., Crowe, M.A. & Boland, M.P. (1992) Postpartum

Vighio, G.H., Liptrap, R.M. & Etherington, W.G. (1991)

anoestrus in dairy and beef cows. Animal Reproduction

Oxytocin–prostaglandin interrelationships in the cow with

Science, 28, 371–8.

pyometra. Theriogenology, 35, 1121–9.

Rowlands, G.A. & Lucey, S. (1986) Changes in milk yield in Waelchli, R.O., Thun, R. & Stocker, H. (1999) Effect of flu-dairy cows associated with metabolic and reproductive

nixin meglumine on placental expulsion in dairy cattle after

diseases and lameness. Preventive Veterinary Medicine, 4, a caesarean.

Veterinary Record, **144**, 702–703.

205–21.

Williams, G.L. & Griffith, M.K. (1995) *Sensory and behav-*

Rowson, L.E.A., Lamming, G.E. & Fry, R.M. (1953) The rela-

ioural control of gonadotrophin secretion during suckling-

tionship between ovarian hormones and uterine infection.

mediated anovulation in cows. Journal of Reproduction and

Veterinary Record, **65**, 335–40.

Fertility Supplement, **49**, 463–75.

Sandals, W.C.D., Curtis, R.A., Cote, J.F. & Martin, S.W. (1979) Wischral, A.,
Verreschi, I.T.N., Lima, S.B., Hayashi, L.F. & The effect of retained placenta and
metritis complex on

Barnabe, R.C. (2001) *Preparturition profile of steroids and*

reproductive performance in dairy cattle – a case control

prostaglandin in cows with or without fetal membrane

study. Canadian Veterinary Journal, **20**, 131–5.

retention. Animal Reproduction Science, **67**, 181–8.

Chapter 35

Problems Associated with

Oestrous Cyclicity

H. Boyd, D.C. Barrett and M. Mihm

Manifestation

530

nancy causing very prolonged calving seasons results in

Aetiology

532

a similar loss; this is partly due to management problems,

Diagnosis

535

such as loss of the seasonal calving pattern, but also

Treatment

541

because at weaning the calf is younger and therefore

Prognosis

542

weighs less. The marketing advantage of being able to

Long interservice interval

544

sell uniform batches of calves may also be lost if the

Prevention

544

calving season is extended much beyond nine weeks.

Clients may present individual cases of cows that have

not been seen in oestrus when due or overdue for

Manifestation

service, but mostly the problem is seen on regular fertility visits when post service anoestrus is also detected in

Normal oestrous cycles, clear oestrous expression and animals diagnosed not pregnant. It is important to dif-

ferentiate as far as possible those cows that have not

predictable interval to ovulation are still essential for

been in oestrus from those that have simply not been the successful management of reproduction in cattle.

seen in oestrus either because of poor expression of heat

Failure to observe oestrus or lack of oestrus adversely or poor oestrous observation. This may require an ass-

ffects the efficiency of production and profitability by essment to be made of the efficiency of oestrous detec-

interfering with the reproductive markers shown in Box tion in the herd and careful examination of 'anoestrus'

35.1 (see also Chapter 41 (b) page 678). Inaccurate

cows on more than one occasion. Regular milk progesterone detection, i.e. identification of a cow as being

terone assays may also be useful when attempting to determine whether an individual cow is undergoing oestrus when she is not, leads to cows being inseminated when fertilization is not possible because there is oestrous cyclicity or is truly anoestrus. Suckler cows no ovulation at the expected time after insemination, running with the bull may be presented as failure to conceive and to pregnancies being lost when already pregnant towards the end of the breeding season. The cows are inseminated using artificial insemination (AI). The problem of abnormal oestrous cyclicity, which is most serious when it occurs on a herd or group basis, is often missed heats that result in longer than planned intervals from calving to first service cause considerable economic loss. A missed oestrus will cause a delay of about 21 days until the next chance for service and this loss in Heifers milk production time and delay in producing a calf. Delayed puberty may affect targets for breeding age or results in a loss of income. Assessment of the real loss

date. It occurs mainly as a group problem because (litres of milk/year; calves/100 cows per year) and the heifers are handled and fed in groups and the cause of economic loss is complex. Where possible, herd-specific anoestrus is nearly always related to the environment economic estimates should be undertaken to determine or to the management, particularly feeding (Webb et al., the cost–benefit of alterations in the various elements of 1997). Occasionally, anoestrus is manifested in a group herd fertility (Dijkhuizen et al., 1997). However, in herds of heifers after natural service, which the farmer with a conventional breeding strategy it may be useful to erroneously thought was successful because of lack of consider a rule-of-thumb value to estimate losses occurs returns to service.

sioned with every day the calving interval is extended beyond the target of 365 days. A figure that has remained

Cows after calving

remarkably constant over the last decade (to 2002) is £2.50 to £3.00/day for every day over the planned calving

An extended interval between calving and first service,

interval. In beef suckler cows, delay in achieving pregnancy calculated on a herd basis, is the most important

530

Problems Associated with Oestrous Cyclicity • 531

Box 35.1

Aspects of reproduction affected by failure to observe, or lack of, oestrus. different countries and demonstrates the seriousness of

the problem. Within herds the incidence of the problem is quite variable at different times and there may be

Heifers

periods of four to six weeks during the year when a high

Age at first calving

proportion of cows appear to stop cycling or exhibit

Season of first calving

poor oestrous signs. This may be a direct seasonal effect

Cows

in certain climates, but in temperate regions is probably

Interval from calving to first service (and thus calving to largely a nutritional effect (Butler, 1999).

conception)

*The number of cows treated by veterinarians for
Timely observation of returns to service
anoestrus and suboestrus was reported to be low in the
1980s. Saloniemi et al. (1986) gave the incidence as 5.2
per cent of cows, falling from first calvers (6.3 per cent)
to older cows (3.7 per cent). Markusfeld (1987) diag-*

Table 35.1

*Percentage of cows with various calving-to-first-
nosed inactive ovaries after two rectal examinations in
service intervals in days, based on 1595 cows in 15 herds.*

*8.5 per cent of 7751 lactations. However, more recently
20 per cent of high-yielding cows have been shown to*

Calving-to-first-service

interval (days)

*exhibit a delayed interval to first ovulation postpartum
based on progesterone profiles (Opsomer et al. , 1996).*

0–55

Target

78–99

> 100

Another manifestation of absence of oestrus is the

56–77

cow that starts cycling and then stops, a situation recorded in 5.2 per cent of cases in a survey based on

Percentage of cows

15.9

32.2

24.7

27.1

progesterone assays by Bulman and Lamming (1978).

Cumulative percentage

15.9

48.1

72.8

99.9

In a similar study (Kassa et al., 1986), cyclicity started and then stopped in four (3.2 per cent) of 125 lactations.

Once again this situation seems to have worsened over time; in a recent Dutch study, true return to anoestrus manifestation of dysfunction of cyclicity and oestrus or was seen in only 3 per cent of high-yielding cows, oestrous detection. A farmer with a serious problem

however, 20 per cent of cows showed a prolonged first may not be aware of it because of poor records or good luteal phase of more than 20 days based on their pro- ones that are not used properly. In the example given gesterone profiles (Opsomer et al. , 1996). Royal et al. in Table 35.1, in 15 herds that were not in fertility (2000) have shown an increase in the proportion of control programmes, the target for calving interval was animals with one or more atypical ovarian hormone 12 months and to achieve this target, cows should have patterns from 32 per cent of cows in the period 1975–82 had their first services 56–76 days after calving. The dis- to 44 per cent in the period 1995–98. During the same tribution of days from calving to first service for the period pregnancy rate to first service declined from 55.6 1595 cows served in these herds shows that about half per cent to 39.7 per cent, again suggesting that fertility of the cows were first served after the target date (Boyd in dairy cattle has declined over time.

& Munro, 1980). Recent data from the National Milk Records (NMR) representing approximately 1 million

Cows after service

dairy cows shows that between January 1997 and September 2000 the mean calving to first service interval rose from 84 to 88 days (Barrett, 2001), therefore Analysis of data from approximately 35 000 cows in 255 British herds (Warren, 1984) showed that 26 per cent of the majority of cows are probably now served after the interservice intervals were greater than 48 days, i.e. target 56–76-day window (assuming a target calving more than two cycles (Table 35.2). These results are index of 365 days). However, the voluntary waiting period, i.e. the number of days postpartum waited until first service, may be deliberately increased in high-yielding cows, where the decision is taken to extend greater than 48 days and in Boyd and Reed's (1961) study in smaller herds at a time when management was (Knight & Sorensen, 1998). On the best farm illustrated

less intensive, 23 per cent of returns were greater than in Table 35.1, 67 per cent of the cows were served within 48 days.

77 days of calving and on the worst farm only 21 per

In Warren's (1984) report the average interservice cent of cows reached that target.

interval was 39 days, possibly due to a delay in the

The situation in this group of herds is quite typical of return to service of 39 - 21 days = 18 days. In some other the results obtained in many surveys carried out in surveys there were rather fewer very long intervals,

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Table 35.2

Percentage of cows with various intervals between services in days (Warren, 1984)

Interservice interval (days)

1–17

18–24

25–35

36–48

49–90

91+

Percentage of cows

6

37

13

18

18

8

possibly due to different culling patterns or more effi-

Box 35.2

Aetiology of anoestrus.

cient heat detection. In New Zealand, Moller et al.

- *Physiological anoestrus: prepuberty, pregnancy, (1986) reported that only 8.6 per cent of return-to-puerperium*

service intervals were greater than 36 days. Nowadays

- *True anoestrus: no oestrous cycle and no ovulation or in New Zealand and other locations where dairy blocked cycle (prolonged luteal phase, such as with farming is highly seasonal, repeats are often resynchro-pyometra)*

nized which increases the submission rates for the first

- *Silent oestrus* : better termed silent ovulation, i.e. ovarian and second repeat cycles of the breeding season.

cycles occur but no oestrous behaviour is detected

While survey data do not explain why a cow has not

- *Weak oestrus*: poor oestrous expression (reduced length
*been served, discussion with farmers makes it clear that
or intensity)*

in the majority of cases the reason is that the cow has

- *Unobserved oestrus*: oestrus normal, human error
*not been seen in heat. In conclusion, failure to have
cows served because they have not been seen in season
is a widespread and important problem.*

first ovulation may be prolonged to 60 days in high-

*yielding cows (Opsomer et al., 1996). In cows that are **Aetiology***

suckling, usually beef cows, the duration of non-

cyclicity may be extended from 25 to beyond 100 days,

The aetiology of anoestrus in the cow and heifer is

depending on body condition score at calving and post-

complex because as presented to the veterinarian it

partum nutrition (Stagg et al., 1995). The effect of

includes several different conditions, as shown in Box
suckling of one or more calves is to inhibit the return
35.2. Because there is a wide range of types of anoestrus
to cyclicity via neurohormonal routes, which can be
there is also a wide range of causes, some unknown.
modified by nutritional factors. This is demonstrated by
Moreover, a variety of management and environmental
the observation that some well-fed, well-managed beef
factors contribute to poor oestrous detection rates.
suckler herds achieve a tight calving pattern at the same
time each year, which requires an early return to cyclic-
ity while the cows are still suckling their calves. For a
Physiological anoestrus
list of factors that affect the onset of puberty, see
It is important to bear in mind that during certain
Chapter 5.
phases of the reproductive cycle, the absence of oestrus
is physiological. These phases are prior to puberty,
True anoestrus
during pregnancy and for at least two weeks after
calving. It is not uncommon for a cow that is pregnant

The major causes of true anoestrus characterized by to be presented to the veterinarian as a case of lack of ovulation are summarized in Box 35.3.

anoestrus. This occurs if there is unknown access to a bull, or unrecorded insemination or misidentification of
(1)

In heifers, bilateral gonadal aplasia, which causes the cow inseminated.

permanent anoestrus and sterility, occurs in the

In dairy cows, from which the calves are removed a common condition of freemartinism, found in the day or so after birth, ovulation rarely occurs earlier than majority of female calves that are cotwins with males.

15 days after calving; in general first postpartum ovula-

It is also observed in bilateral ovarian hypoplasia, a rare tion occurs 17–21 days after calving in medium-yielding inherited condition, in which the genes are passed on dairy cows calving in appropriate body condition and through fertile individuals with unilateral gonadal experiencing no major loss in body condition subse-
hypoplasia. Poor nutrition is important and discussed

quently (Savio et al., 1990). This postpartum interval to below.

Problems Associated with Oestrous Cyclicity • 533

Box 35.3

Major causes of true anoestrus.

*easy to understand why the cow with the luteal cyst,
which is predominantly progesterone producing, does*

Heifers

not exhibit oestrus, though it is less clear why she fails

Freemartinism

to cycle. However, follicular cysts with oestrogen pro-

Ovarian hypoplasia

Inactive ovaries (poor nutrition)

*duction are also found in cases of anoestrus. This may
be due to the low level of the steroid produced in some*

Cows

cases, while in other cases it may result from the nega-

Dystokia and postpartum problems

tive effect on the behavioural centre of high levels of

Delayed involution (inactive ovaries)

oestrogen circulating more or less continuously.

Pyometra (persistent corpus luteum)

Another explanation for the lack of oestrus in cows

Cystic ovarian disease

*Poor nutrition prepartum, severe energy deficit postpartum,
with follicular cysts is that the behavioural centres in
especially first calvers*

the brain, which control the expression of oestrus,

Age-related factors

*respond better to oestrogens if they have been primed
by progesterone.*

Garverick (1997) in the USA and Dobson et al.

(2001a) in the UK have both reviewed cystic ovarian

(2)

*In cows a variety of abnormalities at or around
disease. Factors associated with the occurrence of cystic
calving, such as dystokia, retained fetal membranes,
ovarian disease are discussed further in Chapter 36.*

*hypocalcaemia, metritis, endometritis and other post-
partum abnormalities may result in delayed return to*

(4)

The role of nutritional deficiencies in anoestrus is

cyclicity (for a full discussion of this see also Chapter

relatively straightforward in heifers but is complex in 34).

adult dairy cows.

(3)

Ovarian cysts, traditionally defined as fluid-filled

(a)

In heifers it has been shown in many field trials structures >25 mm in diameter and present for more than 10 days in the absence of a corpus luteum (CL), associated with delayed puberty. Onset of puberty are quite commonly recorded in cases of anoestrus and tends to be related to body weight rather than age. The in fact the majority of cows with cystic ovarian disease mechanism is discussed in Chapter 5.

exhibit anoestrus. This traditional definition implied that cysts were static structures; however, this is now

(b)

In adult lactating dairy cows it is not clear how known not to be the case, and cysts should be con-feeding affects return to cyclicity after calving, even

sidered dynamic both in structure and function (Cook
when only energy is considered; however, evidence is
et al., 1990). The likely hormonal mechanism of cystic growing to suggest that
the effect may, at least in part,
ovarian disease is failure of the preovulatory luteiniz-
be mediated by insulin-like growth factor-1 (IGF-1)
ing hormone (LH) surge. In some cases there may be
levels (Taylor et al., 2001). It is clear that dairy cows failure of the ovary to
respond to LH at the start of
experience a metabolic crisis early postpartum, as
oestrus (Brown et al. , 1986). Cystic ovarian disease is a energy loss cannot be
compensated for by energy
very complex condition and in many cases the cyst pro-
intake and stored energy is utilized. Factors that exac-
duces a mixture of steroids, which are variably absorbed
erbate the negative energy balance including the
into the general circulation.
feeding levels before and after calving, particularly the
In the postpartum period, particularly during the first
energy density of the ration, excessive energy loss due
six weeks, some cows (approximately 20–25 per cent)
to high milk yield and increased body condition loss

experience ovulation failure followed by the development of palpable but functionally transient cystic structures (body condition score loss of more than 0.5) will prolong the interval to first ovulation, as will associated metabolic or other diseases postpartum. A clear relationship has been shown between the timing of the negative energy balance nadir (the lowest point presented for veterinary examination. Cystic ovaries were of the negative energy balance postpartum) and the interval to first ovulation, the LH pulse frequency and with 20 to 30 more days to conception in a meta-analysis of papers published between January 1987 and selected postpartum (Canfield & Butler, 1990; Beam & January 1999 (Fourichon et al., 2000). By about six weeks after calving an ovarian cystic structure should

energy balance nadir; thus, if the lowest point of the
be regarded as pathological. Cysts are often classified
negative energy balance is reached between the first to
as either follicular (about 70 per cent; single large cyst
second week postpartum, cows can be expected to
or multiple smaller cystic structures) or luteal and it is
ovulate by 21–30 days postpartum. However, in cows in
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which body condition score loss exceeds 1, the interval
for food. It is also presumably a stressful experience
to first ovulation may be prolonged to 60 days and
being low within the herd hierarchy.

beyond. One method to try to take into account these
(d)

In many cases first calvers may exhibit a high
many interacting factors is regularly to measure the
incidence of lameness. Lameness (see Chapters 31 and
body condition score of cows (Anon, 1984); particularly
32) is a cause of weight loss that can result in true
important times include drying off, calving and service
anoestrus and also, because of the associated pain, can

times.

cause poor oestrous expression in animals that are cycling. Various other forms of stress such as surgery,
(c)

In the beef suckler cow, prepartum nutritional changes in social grouping, transport and heat stress deficits leading to reduced body condition at calving may all affect fertility, thus stress on breeding cattle delay the interval to first ovulation by more than 40 should be minimized whenever possible (Dobson et al., days to 90 days and beyond (Stagg et al., 1995, 1998). 2001b).

The delay in resumption of ovulatory activity is associated with continued atresia of recurrently selected
(5)

Among miscellaneous causes of persistent corpus dominant follicles; instead of ovulation of the 2nd to luteum is the rare condition of uterus unicornis, in 4th dominant follicle postpartum, it is on average the which one horn of the uterus is absent. Ovulation on 9th dominant follicle which eventually secretes suffi-

the side without a uterine horn results in a corpus
cient oestradiol to induce the gonadotrophin surge and
luteum that is not exposed to locally produced
ovulation. As circulatory concentrations of FSH are not
prostaglandin and therefore does not regress after 16 or
affected by pre- or postpartum diet, the detrimental
17 days.

effects of low body condition score at calving on the
fate of dominant follicles selected early postpartum are
Silent ovulation and unobserved oestrus
attributed to reduced LH secretion, which reduces
dominant follicle oestradiol production.

First ovulation postpartum is generally silent (80 per
The relationship between nutrition and fertility is a
cent), but once cycles have been established, silent
complex one which is still not fully understood. There
oestrus is not a common phenomenon, as was clearly
is a vast body of literature on this subject which is too
demonstrated by Williamson et al. (1972), who carried
extensive to review here. However, the reader may find
out continuous observation of 107 cows for 21 days and

the following reviews of interest: Britt (1995); Webb et al. (1997, 1999); Wathes et al. (1998); O'Callaghan and others in field trials in herds in Boland (1999); Garnsworthy and Webb (2000) and which silent oestrus was thought to be a problem. Generally, when oestrous detection methods are improved, Several studies have shown that cows in their first lactation exhibit a disproportionately high incidence of cases regarded as silent oestrus. Many cases previously true anoestrus, with lack of ovulation. A variety of explanations for this have been put forward, in particular the inhibitory effects of lactation and growth on the recovery of the LH pulse frequency early postpartum, pre-able when based on serial progesterone assays, as in the venting dominant follicle maturation and ovulation.

study of 1400 cows by Ball and Jackson (1984) in which Another suggestion is that this is mainly the result of the

21 per cent of the cows had ovulated but had not been

high incidence of dystokia that occurs in first calvers.

seen in oestrus. This level of non-detected oestrus is in

However, the high incidence of anoestrus in first

agreement with other studies. McLeod et al. (1991)

calvers is often attributed to nutritional and environ-

reported 78 per cent of ovulations in their study were

mental factors. Excessive weight loss and very poor

correctly identified by conventional oestrous detection,

body condition result in cessation of ovulation and

confirming that approximately 20 per cent of those

cyclicity, presumably through hypothalamic–pituitary

cows ovulating are not seen in oestrus.

dysfunction (see Chapter 67). Cows continue to grow

To what extent delayed return to service is due to

until their second or third lactation and therefore young

unobserved oestrus is not clear, but Humbolt (1982),

animals have to carry out the tasks of milk production,

using milk progesterone assays, calculated that only

growth and reproduction simultaneously, at a time 29 per cent of delayed returns were due to embryonic when adult dentition has not been fully developed. In death and that 73 per cent were due to anoestrus dairy cows it is also the time when the young, smaller (including true, silent and unobserved oestrus).

cow is introduced to the highly competitive environ- However, ovulation without the accompanying signs ment of a herd, which, particularly during the housing of oestrus (that is, silent oestrus) does occur. The best period, makes it difficult for small animals to compete documented situation relates to the first ovulation after

Problems Associated with Oestrous Cyclicity • 535 sexual monitoring of the herd. However, a bull may not

Box 35.4

Causes of silent and weakly expressed oestrus.

choose to serve all cows in oestrus if several are in season at the same time.

- *Social*
- *Pain and fear*

(2)

Pain and fear. As oestrus is a behavioural phe-

- *Weight loss*

nomenon that is subject to psychological influences, it

- *Genetic factors*

can be inhibited by discomfort, pain or fear. Severe

- *Presence of calf and suckling*

adverse weather conditions such as driving rain and

cold reduce cows' interest in sexual behaviour. Slippery

flooring produces fear of falling and may inhibit

calving, particularly if it occurs within the first 20 days.

oestrous behaviour. The pain caused by lameness

Otherwise it is almost impossible to distinguish between

inhibits oestrous expression.

silent and unobserved oestrus. Some instances of oestrus

are so weak or last for such a short time that detection by

(3)

Weight loss. It has been suggested that weight loss

conventional observation is a matter of chance.

is associated with an increase in silent heats.

Standing oestrus lasts for about 8–12 hours in cows

(4)

Genetic factors are almost certainly involved. The and 6–8 hours in maiden heifers. However, 20 per cent subjective nature of the measurement of oestrous of heats last less than 6 hours (Esslemont et al. , 1985). expression makes it difficult to gather enough reliable Increasing herd sizes and decreasing availability of data to evaluate the hereditary influence. Reports skilled manpower on many farms is probably exacerbating the problem of poor oestrous detection. It may daughters from bulls under test were kept, showed also be the case that high-genetic-merit, high-yielding that different daughter groups exhibited oestrus in modern dairy cattle exhibit oestrous behaviour either characteristic ways. There may also be differences less intensely or for a shorter duration, making oestrous between breeds in expression of oestrus. Currently, detection more difficult.

there is much anecdotal evidence to suggest that the The causes of silent oestrus, weak oestrus and short modern high-genetic-merit Holstein exhibits oestrus

oestrus are not well understood, but those considered less well than the traditional British Friesian, or some to be the main factors are presented in Box 35.4.

other dairy breeds.

(1)

Social factors within the group of cows, the size of

(5)

Presence of calf and suckling. The presence of a the group and the reproductive status of the rest of the suckling calf may reduce expression of oestrous behaviour all play a role in oestrous behaviour and in the iour in some cows as well as affect oestrous cyclicity. frequency of mounting (Britt, 1987). Mounting requires that there is both a cow in oestrus and a cow that is willing to mount.

Epidemiological data

Cows that are usually bullied or are low in the In a study of 70 775 lactations in Finnish Ayrshire cows pecking order are less likely to stand to be ridden when in which only cases treated by veterinarians were they are in oestrus than more dominant cows. A small

analysed (Saloniemi et al. , 1986) it was noted that the group of cows, say fewer than four or five, may be less

chance of a cow being presented to the veterinarian as effective at stimulating mounting behaviour than a anoestrus was affected by parity (highest risk in first larger group of sexually active cows. Cows at different calvers), calving season (highest risk in cows that calved stages of the reproductive cycle vary in their likelihood from September to February), herd milk yield (highest of trying to mount a cow in oestrus. The most likely incidence in high-yielding herds) and the occurrence of to jump are those in or near oestrus, followed by non-metritis (Chapter 34), mastitis (Chapter 23) and ketosis pregnant cows in the early luteal phase followed by (Chapter 46).

pregnant cows. At the end of an intensive breeding season when most cows are pregnant the remaining cows to be served may be more difficult to detect because of the absence of cows willing to mount. In the

Diagnosis

main, oestrous detection is therefore easier in tight sea-

involving regular visits, diagnosis will be both easier and action, but this may be made up for by the bull's own more reliable because there will be historical informa-





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Box 35.5

The diagnosis of anoestrus.

- *History (including records)*
- *General clinical examination (including condition score)*
- *Special examination of the reproductive system*
- *Rectal palpation and transrectal ultrasound examination*
- *Vaginal specular examination*
- *Laboratory tests, e.g. progesterone assays*

Fig. 35.2

Cyclic solid CL.

Fig. 35.1

Cyclic CL with lacunae on right ovary.

tion available and it will be simpler to introduce routine sampling where this is indicated.

Fig. 35.3

Dominant follicle on right ovary bordering the CL.

There are two separate but overlapping aspects of diagnosis:

- *Stage 1: assessment of ovarian status to determine whether the case is true anoestrus, in which there is no ovulatory activity, or in which cyclicity is prevented by a persistent corpus luteum or other progesterone producing structure; or silent or unobserved oestrus, in which ovarian cycles are taking place.*

- *Stage 2: assessment of the causes of the condition.*

Stage 1: assessment of ovarian status

Since the word 'cycle' implies repetition, theory would

Fig. 35.4

Two corpora lutea in a 6-week pregnancy.

dictate that assessment of whether or not a cow is cycling necessitates repeated examinations over an interval that should allow the observation of two

oestrous periods. Under practical conditions it is may be functional before that time. However, positive usually assumed that the animal is cycling if it is possible to prove that ovulation has recently taken place. examination (size and echogenicity, Figs 35.1–35.4) is Another common assumption concerns a group of usually closely related to its progesterone secretion. animals. It is usually considered that the group as a Progesterone assay of milk or plasma will only produce whole is cycling if two-thirds of the cows are in the a definite, recognizable rise after a similar delay; to luteal phase, i.e. the same fraction of the oestrous confirm cyclicity a sequence of progesterone assays two cycle that the luteal phase occupies. Furthermore, the or three days apart showing a pattern consistent with a description of the ‘luteal phase’ given in Chapter 33 luteal phase followed by a period of low progesterone presents some difficulties in connection with diagnosis. level and another luteal phase should be seen. This is because a corpus luteum is unlikely to be pal-

Accordingly, for clinical purposes ‘luteal phase’ is pable until approximately seven days after oestrus, but defined as from day 5 to day 17 and the normal ‘period



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Box 35.6

History taking for anoestrus cows.

Table 35.3

The accuracy of corpora lutea detection by rectal palpation based on comparison with milk progesterone assay.

- *Date of calving*
- *Any abnormality at and after calving*

Corpus luteum

- *Has the cow been seen in oestrus since calving?*
- *Could the cow be pregnant?*

Present

Absent

- *Oestrous detection methods in the herd*
- *Are there other cows in the herd with similar problems?*

Number of cases

244

158

Accuracy (%)

82

70

of low progesterone' is expected to be from day 18 through oestrus (day 0) to day 4. This definition applies to the majority of cows in a normal population and is needed to form a basis for rational diagnosis, but its use should be tempered by the realization that no definition will fit all cases (Bloomfield et al. , 1986).

The first step is to take a history of the case. Relevant information, required for both stages of diagnosis, is shown in Box 35.6.

The next step is to find out if there is functional luteal tissue present in the ovaries. The traditional way to do this is to palpate the ovaries per rectum, but there are now several choices. One is to carry out a single or a series of milk progesterone assays, which gives a quick,

Fig. 35.5

Long-standing follicular cyst.

reliable result. Another is ultrasound scanning, which allows the user to observe functional luteal tissue in the ovary (Figs 35.1–35.4) (Omran et al. , 1988; Boyd & Omran 1991).

The choice of method will depend on circumstances as each method has advantages and disadvantages.

Rectal palpation is immediate, simple, requires little restraint of the cow and, if part of a regular visit, is relatively cheap. It also allows other observations to be made on other parts of the reproductive tract and on the animal's general condition. There is minimal risk of misidentification of the animal, a danger always present

when samples are taken for analysis. On the other hand, it is a less accurate indicator of luteal function than a progesterone assay. Real-time ultrasound scanning is a

Fig. 35.6

Follicles on right ovary.

valuable tool that, in experienced hands, is capable of giving a very accurate picture of ovaries and uterus (Figs 35.1–35.13).

Rectal palpation alone, when undertaken by an experienced clinician, can be relatively accurate. For example, Dawson (1975) compared the findings made by rectal palpation shortly before slaughter with post-mortem observations. He identified accurately 125 (89 per cent) of 141 ovaries with corpora lutea. The errors were mainly in cows with small corpora lutea and also some with cysts or follicles. However, others have suggested that rectal examinations alone are less accurate. On the basis of milk progesterone assays (which were assumed to be correct) Ott et al. (1986) summarized their own and other workers' results, as shown in Table

Fig. 35.7

phase.





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Fig. 35.8

Non-pregnant left uterine horn during the luteal phase

Fig. 35.11

Early PD 5.5 weeks.

with bladder ventrally.

Fig. 35.12

Early PD 6 weeks.

Fig. 35.9

Pyometra.

Fig. 35.13

Male fetus (3 months).

Fig. 35.10

Early PD 4.5 weeks.

The use of progesterone assays has the advantage

The accuracy of ultrasound scanning was investigated

that they can be incorporated into a fertility control

by Pieterse et al. (1990), who examined the ovaries of programme and used prior to a visit, so that the clini-59 cows by rectal palpation, ultrasound scanning

cian has information available before the examination

(5 MHz linear array transducer) and dissection on the

of the animal. They can also be used after the visit to

day of slaughter, and concluded that for the detection

give extra information about the case. There may be

of a mid-cycle corpus luteum the sensitivity of rectal

slight problems in deciding the level of circulating pro-

palpation was 83 per cent and for ultrasonography the

gesterone that can be taken as indicative of luteal func-

sensitivity was 80 per cent. For the detection of follicles,

tion and caution needs to be exercised in interpretation

ultrasonography was considered to be significantly

of absolute values, as these may change depending on

better than rectal palpation, with ultrasonography

time from milking (Waldmann et al., 1999).

detecting 95 per cent of follicles over 10 mm in

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diameter whereas rectal palpation only detected 71 per

Box 35.8

Likely reasons for no luteal tissue being present.

cent. These results obviously reflect in part the quality

- *True anoestrus*

of the ultrasound equipment used and the skill of the

- *Cycling (day 18 through oestrus to day 4)*

clinician palpating the ovaries and interpreting the

- *Ovarian pathology*

ultrasound images. In another study comparing the use

Freemartin: heifer

of ultrasound and rectal palpation (Fishwick, 1993), the

Cystic ovarian disease

accuracy of palpation and ultrasonography in detection

of luteal tissue was compared to contemporaneous

plasma progesterone concentrations. In this study the

overall accuracy of manual palpation was 62 per cent

compared with 83 per cent for ultrasound examination.

Booth (1988) has investigated the accuracy of diag-

In cows with high progesterone status, manual palpation of luteal cysts using rectal palpation, and the
tion (60 per cent) was significantly less accurate than
clinical finding of luteal cysts was confirmed by
ultrasound imaging (94 per cent), whilst in cows with a
progesterone assay in only 54 per cent of cases. This
low progesterone status manual palpation (65 per cent)
accuracy can be improved by transrectal ultrasound
and ultrasound imaging (70 per cent) were both equally
scanning. For example, Douthwaite and Dobson (2000)
accurate.

reported that diagnostic criteria for follicular cysts
In general it would be expected that imaging of
using ultrasound examination and plasma progesterone
the ovaries using modern ultrasound equipment, for
assays agreed in 92 per cent of cases; a similar compar-
example using a 7.5 MHz transducer, would be more
ison for luteal cyst diagnosis gave a 74 per cent agree-
accurate than manual palpation. However, individuals
ment. The rapid on-farm diagnostic method of choice
highly skilled in rectal palpation may be able to detect

should thus be transrectal ultrasonography, supported luteal tissue with an accuracy comparable to ultra- by milk progesterone assay where required.

sound, but will not be able to palpate small follicles that

If initial examination shows there is no functional are readily detected by ultrasound scanning. Reduced luteal tissue, there are also the various possibilities cost of equipment and improved image quality

shown in Box 35.8.

combined with practice and much increased use within

When the initial examination reveals no luteal func- veterinary medicine have made transrectal real-time tion and apparently normal ovaries and tract, and no ultrasound examination of the bovine reproductive evidence of mucus or mucohaemorrhagic discharge on organs the examination of choice in regular fertility the tail to indicate recent oestrus or ovulation, the cow's visits.

ovarian status is unresolved. If she is cycling normally

If there is evidence of functional luteal tissue, the and in the low progesterone phase of the cycle, a corpus

possibilities shown in Box 35.7 exist.

luteum will become palpable and visible by ultrasound

The simplest way to decide whether the animal is

examination in the ovary within approximately 7 days

pregnant or has a pathological condition is to carry

from the first examination. A second examination at

out rectal palpation or ultrasound examination of the

7–10 days after the first will detect this corpus luteum.

ovaries and genital tract, plus in some cases a vaginal

This second examination must not be delayed too long,

examination. This will, however, fail to reveal very early

as some cows (those at day 5) will go into the low pro-

pregnancy before 25–28 days after service using ultra-

gesterone phase again in about 12 days after the first

sound and below 35–40 days using manual palpation.

examination. These calculations can be upset by the

Figures 35.10 to 35.12 show examples of ultrasound

considerable variations in cycle lengths and proges-

images of early pregnancy. Figure 35.13 shows an image

terone patterns that occur in a normal population of

of fetal sexing in a three-month-old fetus. The most

cows, as detailed by Bloomfield et al. (1986). It is also common finding is that ovaries and uterus appear to be

very useful to carry out a vaginal examination at the normal and these cases are usually regarded as normal time of the first examination, as the presence of copious, cycling cows that have simply not been seen in oestrus.

clear mucus is strongly indicative of the peri-oestral stage of the cycle. In this way vaginal examination may allow a diagnosis to be made without the need for a

Box 35.7.

Likely reasons for the presence of functional second progesterone assay or a second rectal palpation. luteal tissue.

If a second examination or sampling is required it is

- Cycling (days 5–17 inclusive)

best done 7–10 days after the first. It is even better if

- Pregnancy

serial sampling for progesterone assays can be

- Ovarian pathology (luteal cyst)

arranged. It is necessary to work out a satisfactory

- Uterine pathology (pyometra with persisting corpus

regimen of sampling. This involves making a compromise between the biological ideal, which might be daily

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samples, and what is acceptable to the farmer because

Box 35.9

Causes of dysfunction of the ovarian cycle.

of the cost, labour and interference with the milking

- *Freemartinism (congenital)*

routine. For example, a sampling regimen of two to

- *Ovarian hypoplasia (inherited)*

three samples per week, which will give a useful picture

- *Uterus unicornis (congenital)*

of the oestrous cycle but is not frequent enough to time

- *Pyometra (periparturient problems)*

oestrus exactly, may be unacceptably frequent for a

- *Some cases of inactive ovaries*

farmer. This situation may change in future if accurate

- *Nutritional and metabolic stress*

and reliable automated systems to assess the cows'

- *Systemic disease*

physiological state using biosensors can be developed

- *Delayed involution*

(Koelsch et al., 1994; Mottram, 1997; Scully et al., 2000).

New developments in this area of technology may allow automatic milk progesterone assays, and other assays, involution and concomitant disease, present some problems to be performed while cows are being milked, allowing lems in diagnosis.

the timing of ovulation to be accurately predicted and

In cases of delayed involution, the enlarged uterus removing the need for oestrous detection.

and particularly the enlarged cervix, which involutes

Detection of antral follicles in the ovary, the observation more slowly, usually make the diagnosis of the immediate cause easy. However, on some occasions, by the of limited value because they do not give clear indication of exact timing of oestrus to predict ovulation.

returned to normal size and the veterinarian will have

There are a number of aids to detection of oestrus that

to depend on a history of periparturient problems and can aid diagnosis (Boyd, 1984).

possibly of abnormal vaginal discharges noted by the

The veterinarian now knows whether the cow or

farmer (see Chapter 34 page 523 for details of an invo-

*group of cows is exhibiting true anoestrus or is under-
lution scoring system).*

going ovarian cycles. If a conclusion of anoestrus has

The clinician should look for any other disease con-

been reached an important question is whether (i) the

dition in the cow examined for anoestrus. Especially

cow is in 'deep' anoestrus, which means that she is not

important is any disease that causes loss of body con-

*likely to start cycling soon and may revert to an anoe-
dition or pain.*

strous state following treatment, or (ii) whether she is

There is a complicated series of factors, such as actual

in 'shallow' anoestrus and liable to begin cycling

and potential milk yield, feeding before and after

soon and be responsive to therapy. Unfortunately, it is

calving and weight change after calving, which are very

almost impossible to predict accurately the 'depth' of difficult to assess. Body condition of the cow should be anoestrus. However, if the cow is in extremely poor condition at the time of examination for anoestrus, but this condition and if she has small hard ovaries she is probably deals with only part of the problem (Anon., 1984). If in deep anoestrus and may require a combination of body condition is poor or loss of body condition score treatments to induce first ovulation, with the possibility has been extreme, or if there are many cases of inactive of subsequently reverting to an anoestrous state.

ovaries, feeding should be investigated. Feeding analysis and metabolic profiles are discussed elsewhere in about to start cycling and are absent in those in deep this book (see Chapters 9 and 47).

anoestrus requires a series of samples taken at short On occasion there is no clear reason for the occurrence of inactive ovaries, either in the individual cow or in a practice laboratory but are routinely carried out in

when the condition affects a group of animals.

affiliated research laboratories.

When investigating silent oestrus and unobserved

oestrus, the veterinarian should find out about the

Stage 2: assessment of the cause of anoestrus or

following:

silent ovulation

- *Organization of oestrous detection.*

This section deals with the difficult question of how to

- *Ease of cow identification.*

find out what has caused the abnormal ovarian status

- *Method of recording.*

or the problem of oestrous expression or detection.

- *Layout of the buildings.*

To some extent procedures followed for diagnosis of

- *State of the floors and levels of lighting.*

ovarian status also give the clinician an understanding

- *The way the cows are handled, moved around and of the causes of the ovarian dysfunction. Examples of assigned to groups.*

these are listed in Box 35.9. Three of the causes of inac-

- Amount of knowledge of oestrus and the oestrous ovaries: nutritional deficiency causing metabolic cycle among the people who are detecting oestrus. stress and severe negative energy balance, delayed
- Motivation for oestrous detection.

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- Time made available for oestrous detection.

Administration of an adequate dose of GnRH

- Personalities of the people involved.

results, in the majority of cases, in luteinization or ovulation of healthy dominant follicles present at the time followed by a transient period of progesterone production that can first be identified about five days after

Treatment (see also Chapter 42)

treatment. The duration of this progesterone phase is very variable, ranging from one or two days to the life-
When using proprietary products, the clinician should span of a normal cyclic corpus luteum, and therefore consult the data sheets, produced by the manufacturers, the interval from treatment to oestrus is very variable, for details of use, contraindications and other guidance.

about 8–22 days. It is desirable to follow up these cases
Successful treatment, leading to oestrus, service and
to ensure that luteinization or ovulation has taken
pregnancy, depends not only on veterinary therapy but
place; this is best done by ultrasound scanning or by
on husbandry and breeding management, some aspects
progesterone assay.

of which are described in the data sheets and also on
If there is luteal tissue present at the follow-up exam-
page 525. Different manufacturers of apparently similar
ination 7 days later, it is useful to treat the animal with
products often give slightly different advice about their
PGF2a or an analogue, as this will make the resulting
use and users should be aware of the major differences
oestrus more predictable. Regular fertility control visits
between some of these products. Some of the thera-
at weekly intervals are ideal for this regimen; however,
peutics, for example Prostaglandin F2a (PGF2a) are dan-
visits are often at fortnightly intervals and this may be
gerous for humans and could cause return to service
a less suitable period between the initial and follow-up

and abortion in cows, and therefore the veterinarian treatment when using the GnRH–PGF2a regimen.

must handle them responsibly and with care.

If, on the follow-up examination, there is no luteal

Where drugs that result in oestrus are used, efficiency tissue the cow should be dealt with as a new case.

of oestrous detection will be enhanced as the clients are

The use of a progesterone or synthetic progestagen

told to concentrate on a specific time for oestrous detec-

slow release device also mimics the progesterone rise

tion; their attention is focused on the cows treated and

before first ovulation. This device is left in the vagina

aids to oestrous detection such as tail paint are often

or implanted subcutaneously at the base of the ear for

used.

8–12 days and the animal should be served at the

oestrus that occurs in many cases about two or three

days after withdrawal. Fixed-time insemination 48 and

True anoestrus: no functional luteal tissue

72 hours (or a single insemination at 56 hours) after

Two points should be remembered when dealing with

withdrawal may be carried out (follow data sheet these cases:

recommendations for whichever product is selected).

If oestrus is observed later another insemination

- *All healthy, suitably fed cows will start cycling in the should be carried out. Avoid stress and drastic change first two months after calving.*

of diet during and after treatment (see p. 683).

- *There must be an underlying reason for the occur-*

In those animals in which increased negative energy rence of this type of true anoestrus and an attempt

balance, poor body condition or very strong behav-

should always be made to find the cause and deal

ioural effects inhibit LH pulse frequency and thus

with it. Where the cause appears to be nutritional,

dominant follicle maturation, final differentiation of

improved feeding should be part of the treatment

the preovulatory dominant follicle or the preovulatory

of the affected individual and should also be

oestradiol rise may have to be enhanced. The addition

considered for other recently calved cows. Consi-

*of equine chorionic gonadotrophin (eCG) given at the
deration should also be given to the body condition
end of the progesterone or synthetic progestagen
changes and feeding regimen of the dry cows as well
treatment will induce oestrus 48 hours later. Similarly,
as the management and nutrition of the transition
treatment with a very low dose of oestradiol benzoate
cow. Particular care should be taken to ensure that
24 hours after device withdrawal will induce oestrus
high-genetic-merit dairy cows do not become
12–20 hours later, but consideration will have to be
too fat during the dry period (Rukkwamsuk et al.,
given to the national legislation relating to the use of
1999).*

these drugs in dairy or beef cattle.

At present there are two main hormonal treat-

*The treatment of follicular cysts involves either using
ments for inactive ovaries: (i) administration of
intravaginal progesterone releasing devices (PRID or
gonadotrophin-releasing hormone (GnRH) or ana-
CIDR) or establishment of endogenous luteal tissue*

logues with GnRH activity and (ii) insertion of a
with chorionic gonadotrophin or GnRH (Dobson et al.,
progesterone-releasing device (see p. 681).

2001a). There is evidence to suggest that the success of
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treatment of follicular cysts depends on whether the
with a view to treating with PGF_{2a} or an analogue, if
cyst is producing oestradiol or not, as reflected by the
appropriate.

presence or absence of other follicles >5 mm in diame-

Conservative treatment is preferred in the first
ter. Treated with either an intravaginal progesterone
instance by some clients who would rather leave the
releasing device or GnRH, cystic follicles in the
animal untreated, are content to know that she is
presence of other follicles had a pregnancy rate to all
cycling and hope to observe the next oestrus. In these
inseminations of 91 per cent compared with 69 per cent
cases aids to heat detection should be used and the
if no other follicles were present at the time of
animal re-examined if she is not seen in season within

treatment (Tebble et al., 2001).

the next two weeks.

True anoestrus: functional luteal tissue

Silent oestrus/unobserved oestrus: no corpus

luteum present

The therapeutic approach is to cause luteolysis by

parenteral administration of PGF2a, or an analogue,

These cases do not benefit from immediate drug

followed by intensive oestrous detection up to one

therapy as animals are showing ovarian cycles but have

week after injection and insemination at observed

either just regressed their CL or are in the early luteal

oestrus. In the case of pyometra (Fig. 35.9), the client

stage following ovulation. Improvements in husbandry,

should be advised to delay service until it appears that

i.e. oestrous detection, and, possibly, feeding may be

the infection has cleared up, as indicated by an oestrus

required to enhance detection and oestrous expression,

at which the mucus from the vulva is free of pus. It is

and it may be useful to enhance oestrous expression

advisable that the veterinary surgeon should examine

hormonally by using eCG or oestradiol benzoate following these cases at a follow-up visit to ensure the uterus has returned to normal (Figs 35.7 and 35.8).

above).

The treatment of cystic ovarian disease depends on the results of the first examination. Because diagnosis is uncertain until the second examination, largely on whether the structure is progesterone producing or not. If considered a luteal cyst with functional corpus luteum, whether these cows should be treated at the first visit. If there is no corpus luteum on the second examination, luteal tissue, as judged by ultrasound examination or milk progesterone assay, the logical treatment is to administer PGF_{2a}, although alternative therapies such as the use of intravaginal progesterone releasing devices may also be successful (Douthwaite & Dobson, 2000).

Prognosis

In considering prognosis, primary attention has to be

Silent oestrus/unobserved oestrus: corpus

paid to the return to cyclicity, which can be measured

luteum present

either by observation of oestrus or, more reliably, by

There are two standard approaches in these cases, the

one or more progesterone assays at planned times after

choice being between (i) the induction of luteolysis

treatment. The outcome of treatment is influenced by

using PGF2a or an analogue and (ii) leaving the animal

both the veterinarian and the client (and perhaps also

untreated to be observed when she comes in heat. The

by the cow), as presented in Box 35.10.

method chosen will depend on many variable factors,

Factors influenced by the veterinarian, which affect

such as the season, the time since calving, the herd

the outcome of treatment for anoestrus, are as follows:

calving pattern and the inclination of the client, and

therefore the decision has to be made individually for

Box 35.10

Contribution of farmer and veterinary surgeon to

each case.

influencing prognosis.

Treatment with PGF2a or an analogue should, in the majority of adult cows, result in oestrus in 2 to 7

Prognosis influenced by veterinarian

Accuracy of diagnosis

days, with the peak period being days 3 and 4 after

Selection of cases

treatment. Service should be at observed oestrus and

Choice of therapeutic intervention

aids to oestrous detection should be used. Enhanced

Follow-up examination

oestrous expression may be observed when combining

a progesterone or synthetic progestagen device with

Prognosis influenced by farmer

PGF

Oestrous detection

2a.

Herd pregnancy rate

Cows that are not seen in oestrus after treatment

Culling rate

should be re-examined after 14 days (Young, 1989)

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- The accuracy of diagnosis is the basis for correct Heifers*

treatment. There is good evidence that identification

Freemartins and cases of bilateral ovarian hypoplasia

of a corpus luteum by palpation by experienced

are sterile and will never cycle or become pregnant.

veterinarians is over 80 per cent accurate (see p. 537)

Groups of heifers with delayed puberty due to low

and that differentiation between follicular and luteal

energy intake will return to cyclicity after restoration of

cysts is much less accurate (see p. 539); however,

a suitable energy intake level and other correction of

such a diagnosis can now be more specific due to

the feeding. How long this will take depends on many

the routine use of ultrasound scanning, and can be

things. Under the best possible circumstances, i.e. where

enhanced via using milk progesterone assays.

the animals are in not excessively poor condition and

- Selection of cases for treatment has an important*

where there is no complicating disease factor, it will influence on the outcome. For example, the corpus take approximately two months of good feeding before luteum early in the cycle is less responsive to PGF2a most of the group start cycling (Imakawa et al. , 1986).

or an analogue than is the mid-cycle corpus luteum.

Hormonal treatments to induce and thus advance

Accurate assessment of the stage of the cycle will aid

first ovulation can be used if the management system

in the correct selection of animals for treatment.

is seasonally restricted; introduction of a bull may

Similarly, if treatment is confined to cases in which

also aid in the induction and synchronization of first

corpora lutea are positively diagnosed, better results

ovulation.

may be obtained than if less critical selection of cases

is undertaken. However, when PGF2a treatments

based on the presence of a corpus luteum were

True anoestrus after calving in cows

compared with 14-day fixed treatments, the fixed

treatments were still superior resulting in better

In cases of pyometra with a corpus luteum, the failure reproductive performance (Heuwieser et al., 1997). to cycle is very effectively dealt with by using PGF2a. Ott and Gustafsson (1981) reported that more than 80 Other factors that influence the success of luteolysis per cent of treated cows emptied the uterus and came and whether the cow comes in season after successful in season a few days after treatment. If one treatment luteolysis are not well documented. However, it is fails to produce luteolysis, then a second injection possible that nutritional factors are involved in the almost certainly will be successful. There is evidence to subsequent expression of oestrus. suggest that treatment of pyometra is improved by the administration of two doses of PGF2a, 8 hours apart

- *None of the luteolytic products commercially (Archbald et al. , 1993) (see p. 524). available achieve 100 per cent luteolysis (Ball & Inactive ovaries are treated with either GnRH to Jackson, 1984; Martinez & Thibier, 1984). cause ovulation and thus a progesterone rise, which*

- *Re-examination and retreatment in good time of all then primes the animal to express heat at the next cases that do not respond to the initial treatment ovulation, or a progesterone or progestagen releasing will affect the time from initial treatment to oestrus, device. Some indications of the results that can be service and conception.*

obtained were shown in a field study involving 62 The farmer also affects the outcome of the treatment acyclic cows (Ball & Lamming, 1983). There were two in important ways.

main differences between the two methods. Firstly, ovarian response to the progesterone intravaginal

- *The efficiency of detection of oestrus in the herd releasing device (PRID) was better, as 89.7 per cent of is crucial to the effectiveness of treatment when cows ovulated compared to 73.9 per cent of those measured by the number of cases that come into treated with GnRH. Secondly, after the PRID, fixed-season within a certain number of days.*

time insemination resulted in 59 per cent conceptions

- The first-service pregnancy rate in the herd which (measured by milk progesterone) whereas after GnRH, is to some extent under the control of the farmer, a number of cows that responded successfully were not not least because correctly timed insemination is seen in heat so that only 26 per cent of the cows treated essential for conception, will have a great effect on became pregnant at the ovulation after treatment. One the success rate as measured in time to conception. problem with GnRH treatment is that the luteal tissue
- High culling rate and willingness to cull may result produced has a very variable lifespan with the result in apparently good results because the failures are that the timing of heat and the next ovulation cannot culled, but depending on the economic factors per- be predicted and may occur from 8 to about 20 days taining at the time this may be a false economy after treatment. In order to cause more predictable (Esslemont & Kossaibati, 1997; Whitaker et al. , onset of oestrus and next ovulation following GnRH 2000).

treatment, a luteolytic dose of PGF2a can be given 6–7

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days after GnRH. Such a protocol can be further com-

Silent and unobserved oestrus after calving in cows

bined with a second injection of GnRH or an injection

Prognosis for unobserved oestrus is good and preg-

of human chorionic gonadotrophin (hCG) 48 hours

nancy rate is not greatly different from that for cycling

after PGF2a to time the next ovulation in all animals

cows in the same herd that have been observed in

and use fixed-time insemination (De Rensis & Peters,

oestrus. Seguin (1981) compiled results from six trials

1999). Occasionally, cows will come into oestrus quickly

in which 886 cows were treated; 67 per cent were in

after GnRH treatment and this may be due to treat-

oestrus after treatment and pregnancy rate to service at

ing an animal that was just about to start cycling

that oestrus was 54 per cent. Seguin (1981) also cited

spontaneously.

trials in which controls and treated cows had the same

In all reports on treatment of inactive ovaries there

pregnancy rate, whereas O'Farrell and Hartigan (1984) are some animals that do not respond to conventional obtained a statistically significantly poorer pregnancy treatment. It is obvious that cows that are still under the rate in the suboestrus cows treated with an analogue of influence of the original aetiological factors (such as PGF poor condition) are unlikely to respond well. Similarly, 2a than in controls. This latter finding is supported by an analysis of more than 10 000 cases of unobserved cows in deep anoestrus may not respond satisfactorily oestrus in Israel and 30 000 normal controls (Mayer et al. , 1987). First-service pregnancy rate for unobserved with inactive ovaries, lacking any detectable structures, oestrus cows was 35.2 per cent compared with 60.3 per respond poorly to treatment and may revert to an cent for the controls. anoestrous state.

In an interesting study (Ball & Jackson, 1984) in In considering the outcome of treatment of cows with

which non-detected oestrus cows were treated with an cystic ovarian disease associated with anoestrus there analogue of PGF

are a number of relevant points to note:

2a, and monitored by observation and progesterone assays, 84 per cent of the cows had complete luteolysis after a correctly timed injection. Of all

- With the important exception of cases seen within the cows treated 42 per cent were seen in oestrus within six weeks of calving, spontaneous resolution of 4 days of treatment and 60 per cent within 14 days.*

these cases is not to be relied upon (Garcia &

One factor that makes prognosis difficult is the effect Larsson, 1982).

of the age of the corpus luteum on the response to

- The condition tends to recur unless treatment*

PGF

results in ovulation of a healthy new dominant

2a or an analogue. Luteolysis is unlikely to occur in

cows treated before day 5 of the cycle and it may be day follicle and conception occurs.

7 before maximum effect results. Even when luteolysis

- To the extent that there is a familial predisposition occurs, the time from treatment to ovulation varies to cystic ovarian disease, the use of offspring from greatly depending, in a complex manner, on the stage affected dams for breeding purposes is not of dominant follicle development when the cow is advisable.

treated (MacMillan, 1983; Kastelic et al. , 1990). If there

- Results have to be looked at in two stages. The first is a healthy dominant follicle present, oestrus will stage is the resolution of the cystic condition and usually occur on the third day after treatment; should the induction of normal oestrus and ovulation. The there be no dominant follicle present at the time of second is getting the affected cow pregnant.

PGF2a injection, 5–7 days will pass until induction of oestrus occurs.

Where diagnosis is correct and appropriate treatment

Prognosis is improved if the veterinarian actively

is given the return to ovarian normality is good. The

follows up all cases, both those treated and those left majority of luteal cysts respond to PGF_{2a} or an untreated, within two weeks.

logue; in one study (Booth, 1988) 79 per cent of cows had low levels of circulating progesterone within 7 days.

In the same field trial, 73 per cent of cases with

Long interservice interval

follicular cysts that were treated with GnRH or hCG plus progesterone had functional luteal tissue by 14

Prognosis in these cases is similar to preservice cases, days after treatment.

but because of the longer time since calving the urgency

The pregnancy rate achieved again depends very to get the cow reserved is greater.

much on the ability of the farm staff to detect oestrus and have the cow served at the correct time. This, plus the difficulty in accurate diagnosis of the type of cyst

Prevention

present, means that on many farms an unacceptably high percentage of cows with cystic ovarian disease are

The economic importance of prevention of anoestrus in

eventually culled.

all its forms can hardly be overstated. The cost-effective

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application of preventive measures, however, calls for

and onset of puberty. The farmer should consult the

careful consideration, and a balance has to be achieved

veterinarian at an early stage if it looks as though not

between undue intervention in cows that would resume

enough animals are starting to cycle at the expected

normal cycles spontaneously and unnecessary loss of

time, and hormonal treatments to induce first ovulation

time due to leaving intervention too late. For example,

and synchronize oestrus and ovulation in a group of

the majority of cows that are not cycling by six weeks

heifers may be considered.

after calving will be coming in season by eight weeks,

Avoidance of disease conditions that will interfere

i.e. in time for service within the target period. On the

with growth is also the veterinarian's responsibility and

other hand, cows that are left without examination and

is described elsewhere in this book. This should not only

treatment until the end of the target period for first assist the attainment of the target for age at puberty but service, say 11 weeks after calving, will lose a great deal should also help to reduce the high mortality rate that of production time. However, with the increase in milk occurs between birth and puberty.

yield and genetic merit of dairy cows, more prolonged Most farmers are aware that female calves born twin voluntary waiting periods may be economically viable to a bull calf are very likely to be sterile freemartins. and of benefit to the individual animal.

Few realize that measurement of the vagina in the very young calf and comparison with a normal calf can be used to decide if a suspect calf is a freemartin.

Heifers

Alternatively, in valuable animals blood sampling both Prevention of delay in the onset of puberty depends twins for karyotyping will usually clarify the issue and on good nutrition and the avoidance of diseases that allow early culling if desired. However, this procedure interfere with growth rate (see also Chapter 5).

is too expensive for use in most commercial cattle.

The first step is establishment of clear targets. The farmer should decide at what age, or at what time of the year, the heifers should have their first calves. It is a simple calculation from that target to decide when the heifers should start their service period. The foundations for early return to normal cyclicity and service period should start, so that the majority of the heifers calve within the planned period. All these factors are very much under the farmer's control, timing for the start of the service period will depend on although the veterinarian has an important part to play in ensuring that the husbandry is up to standard, that calving period are held to be. If conditions are good, intervention, if required, at and after calving is timely such as a healthy, fertile young bull running with and appropriate and that cyclicity is monitored and well-grown, healthy, cycling heifers, more than 80 per aberrations are treated.

cent of heifers should be in calf after six weeks of the

- Prevention of dystokia and postpartum abnormali-

service period and practically all by nine weeks.

ties is a basic requirement for optimal cyclicity and To ensure that the majority of the heifers are cycling

fertility and has been discussed on p. 526.

at the beginning of the service period it is necessary to

- Routine veterinary postpartum examination and

aim for a growth rate that achieves the appropriate

treatment can effectively limit the adverse effect of body weight, taking account of the breed involved.

abnormalities around calving and delayed involu-

Puberty occurs around 280 kg in large Holsteins and

tion. It is essential to examine all cows that experi-

Holstein/Friesians, around 260 kg for Friesians and

enced dystokia or postpartum problems by not later

around 230 kg for Jersey heifers; these weights equate

than 28 days after calving. Many veterinary practi-

to approximately 40–45 per cent of the expected adult

tioners incorporate routine examination of all cows

weight. A rough rule-of-thumb for the rearing of heifers

two to four weeks after calving, as this allows them

to calve at 2 years of age is to aim for prepubertal to deal with problems early (see also Chapter 34). growth rates in g/day that equal the adult body weight in kg. For example, if the expected adult weight is 650 kg, rear at a rate of 650 g/day. Target growth rates

Box 35.11

Factors likely to prevent postpartum anoestrus. and the key target weights for dairy heifers in the UK are given in Chapter 5.

- *Appropriate body condition and nutrition in the dry period*

The veterinarian has a number of roles to play.

- *Normal calving*

Initially, it is necessary to make the farmer aware of

- *Disease-free puerperium, or adequate rapid treatment of the need to plan for a specific calving season or age diseases in the puerperium*

and what this implies in terms of growth rates. There-

- *Suitable transition period feeding and regular checking of body condition score postpartum*

after, the job is mainly to give advice and to make sure

- *Efficient oestrous detection*

that the client monitors the growth rate of the heifers

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Table 35.4

Effect of various treatments on interval from calving to first service.

Calving – service interval (days)

Reference

PGF2a

GnRH

Both

None

—

81

—

77

Langley & O’Farrell (1979)

—

88/89

—

83

Nash et al. (1980)

—

72/74

—

73

Nash et al. (1980)

—

—

61

63

Richardson et al. (1983)

92

108

85

84

Etherington et al. (1984)

61

67

64

58

Benmrad & Stevenson (1986)

73

—

—
69

Young & Anderson (1986)

81/51

—

—

91/58

Schofield et al. (1999)

*There is an attractive concept that by routinely
terms of reproductive efficiency, there is the danger
administering PGF2a or an analogue, GnRH or both in
that induction of a corpus luteum with GnRH in a cow
the early postpartum period it is possible to improve
that still has an infected uterus after calving can lead
return to cyclicity and pregnancy rate. It is clear that
to pyometra. Selected treatment of cows with uterine
GnRH given before day 20 after calving will initiate
or ovarian pathology is on the other hand entirely
early cycles and PGF2a or an analogue given a little later
justified.*

increases the number of cycles in the first six weeks

- *Nutrition. For normal reproductive function it is after calving (Benmrad & Stevenson, 1986). The effect vitally important that nutritional levels before and of these treatments on overall fertility is less well after calving are correct. It has been suggested that defined; Table 35.4 shows results obtained in several condition score at calving should not exceed 3.0 and field trials.*

2.5–3.0 should be the target at service.

Meta-analysis of data from 4052 cows in 24 trials published in 10 papers looking at the effect of injecting PGF2a in dairy cattle within 40 days of calving on the Oestrous detection rate of pregnancy at first service and the number of days open came to the following conclusions. Treatment with PGF2a during this early postpartum period had no effect on the first service pregnancy rate of cows with a normal or abnormal puerperium. An analysis of those

Before discussing how oestrous detection can be data for the number of days open showed that a significant percentage (54 per cent) of the treated cows had oestrous detection inefficient? After all, many stockworkers who carry out this task have worked with cows difference tended to be greater for cows with an abnormal puerperium. The weighted average reduction in in a recent study of stockworker's attitudes, oestrous days open between treated and control cows was 2.6 detection was recognized as being an important task, days for trials with abnormal cows and 3.3 days for trials but was generally a task that was disliked (Seabrook & including normal and abnormal cows (Burton & Lean, Wilkinson, 2000). This may be because it is time consuming and often requires the stockman to spend time in the submission rate; it is questionable whether watching cows at antisocial hours. The decreasing

any improvement in calving-to-first-service and calving-availability of labour on farms is resulting in reduced to-conception interval justifies the treatment costs and labour hours per cow per year and overworked staff the cost of the extra semen that would be used if PGF2a often do not have the necessary time to dedicate to were administered routinely to postpartum cows. Thus oestrous detection.

the routine administration of PGF2a (or other hormone Good oestrous detection requires regrouping and preparations) to cattle without a specific therapeutic possibly moving animals as well as accurate observation indication probably cannot be justified.

and recording of behavioural events several times a day

In addition to the routine application of these treat- over months on end and is therefore a very demanding ments to normal cows not conferring any benefit in task. It is made more difficult because of the short dura-

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tion of oestrus exhibited by many cows, the time of the A rational system of oestrous detection should be based

night or day when oestrus occurs, the weak and indefinite nature of signs shown by some cows when in season a good heat detection system:

and the fact that some animals not in season exhibit

(1)

The cows should be observed three times per day some of the signs of oestrus. Veterinarians who wish for 30 minutes each time. The periods of observation to educate dairy workers on heat detection would be when the herd is peaceful after well advised to attempt to carry out heat detection milking and feeding. Often these times are:

themselves, so that they may understand the practical

(a)

briefly before collecting the cows for difficulties.

morning milking;

If oestrous detection is not a priority for staff and

(b)

in mid-morning;

is impossible to organize even with the assistance of

(c)

before the evening milking; and

oestrous detection aids, then the use of hormonal treat-

(d)

late in the evening, a very good time for

ments to synchronize oestrus and ovulation should be

detection of oestrus.

contemplated and discussed (see p. 678). This will

(2)

The housing conditions must be suitable for cows

reduce oestrous detection periods to very specific times,

and observer. Good non-slippery floors on which

or allow the use of fixed-time artificial insemination

the cows feel secure are essential. The observer

with no heat detection. However, pregnancy rates to

must be able to see all the cows clearly, should be

single fixed-time insemination may be low, and this will

comfortable and should have a paper and pen to

also have to be discussed with the client. The details

write down details of the cows' behaviour. Good

of the various oestrous synchronization regimens for lighting is required.

cattle are discussed elsewhere in this book (see p. 684).

(3)

The cows must be clearly identifiable from a

The stockworker's dilemma occurs when a cow distance.

exhibits signs of oestrus just short of actually standing to be ridden and the decision is taken to have the

There are many aids to oestrous detection; however,

cow inseminated. If the cow is not in season this

there is at present no one substitute for close observa-

decision is criticised. However, if the cow is not

tion of the cows' behaviour (review by Boyd, 1984;

inseminated, a cow in season may have been missed. In

Stevenson, 2001). Aids to heat detection either detect

a large herd these critical decisions have to be made

unobserved standing to be ridden or depend on the fact

many times a year.

that cows in oestrus are likely to behave in ways that,

Good heat detection is based on the following facts.

although not unique to oestrus, occur with greater frequency during oestrus. Increased frequency of bellow-

- *The definitive sign of oestrus is standing freely in, increased restlessness, reduced milk yield, change to be ridden. It has been proposed that a cow in routine behaviour such as order of coming into the milking parlour for milking, and increased body temperature are non-specific, oestrus-related events. There other cows.*

are also a number of physical changes in the genitalia

- *There may be an interval of as much as 30 minutes related to oestrus that can be measured, such as change between bouts of riding.*

in electrical resistance and viscosity of the vaginal

- *Oestrus rarely lasts for more than 18 hours and in mucus.*

many cases lasts for less than 6 hours.

Marking systems fixed on the cow's pelvic region to

- *Oestrus often starts during the night.*

detect standing to be ridden when the cows are not

- *Oestrous expression is greatest during quiet times being observed can be valuable in suitable circumstances. Examples are tail paint, tail flags and detectors seen less often around milking times and when cows that contain dye that is squeezed out under sustained are fed.*

pressure from another cow. All of these suffer from the

- *As well as a willingness to stand on the part of the defect that rubbing or pressure that is not caused by the cow in oestrus, it is necessary to have a cow that cow standing freely to be ridden may trigger them. wants to and can mount as discussed above (see*

Television cameras with video recording have also aetiology, p. 532). This can cause particular problems when most cows in the herd are pregnant.

viewed later, at increased speed. There are problems of

- *When cows are loose housed there is often a parity identity (very big numbers on shoulders and rumps are*

ticular area that cows prefer to use for oestrous needed) and replaying the recordings is a tedious chore. expression.

Milk progesterone assays, which can be used either

- Stalled cows may have reduced physical expression as an aid to or as a replacement for oestrous detection, of oestrus (Claus et al. , 1983).

are discussed later (see p. 548).

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Pedometers that automatically monitor and record preferred sampling regimen was every second day from the cow's level of activity can also be used to detect days 18 to 24 after service. The financial benefit to cost behavioural changes that are consistent with oestrus in ratio was 7.5:1. Some veterinarians report improve-some management systems.

ments based on a single progesterone assay on day 19

It is probable that in future sophisticated decision after service. For good results it is necessary for the support software integrated into computerized milking stock personnel to understand the test and to be willing

parlour systems will be developed to detect cows likely to carry out the extra work carefully and conscientiously. Very poor pregnancy rates have been reported to be in oestrus and automatically shed them from the main herd for the attention of the stockman. These to result if progesterone assays are used as the only decision support tools will use various data automatically generated from a number of sources such as pedometers, milk yield, milk temperature, order of milking and milking frequency (fully automated confirm that cows are in oestrus before inseminating milking systems only), and data detected by integrated biosensors such as in-line milk progesterone assays and number of mis-timed inseminations (McLeod et al. , 1991; Watson, 1996). Strategic milk progesterone assays of other milk constituents. Cows will be identified by implanted microchips. These may also advance

monitoring as a diagnostic and management tool in
beyond the current static data-specific microchip that
dairy cattle is discussed by Darwash and Lamming
simply holds the data it was implanted containing, to a
(1997). Under experimental conditions milk progesterone
new generation of smart chips that record the animal's
monitoring has also been used in suckler cow
health and disease history along with other information
fertility investigation and management (Mann et al. ,
such as its movement history. It may even be possible
1998); however, logistical problems will almost certainly
in future to develop integrated implanted biosensors
prevent widespread use of this technology in the field.
that analyse and record the animal's physiological state,
Resynchronization of repeats following an initial
including data related to reproduction.
synchronizing treatment using a progesterone device is
used successfully in New Zealand and other locations
where the breeding period needs to be kept short; used
Anestrus after service
progesterone devices are re-inserted on day 16 to days

In the majority of cases, delayed return to service is due 21 or 22 following first service, and return oestrus to unobserved oestrus, as has been shown in the reduced on days 23 or 24 (Van Cleeff et al. , 1996; tion in the number of long return-to-service intervals McDougall, 2001). This increases submission rates in when oestrous detection is improved. One problem, in the first three weeks of the breeding season and focuses a case where the return-to-service heat signs are not heat detection periods to a specific time following first clear, is that the result of inseminating by AI a cow that service. A similar regimen has been successfully used in has been served, and could be pregnant, may be loss of the UK in beef suckler herds (Penny et al. , 2000). the conceptus.

Improved efficiency by the use of aids for heat detection is particularly useful in these cases because the

Acknowledgement

service date can be taken as the start of the oestrous cycle. Clearly, in those cases where insemination takes
The ultrasound images were produced at the University

*place in the luteal phase this starting point is erroneous,
of Glasgow Veterinary School using digital acquisitions
a fact that will become clear to the stockworker as heat
from Toshiba Medical Systems.*

detection aids and substitutes are used.

*Use of a three-week calendar and the application of
a marker such as tail paint shortly before expected*

References

return to service can help efficiency of observation.

Systematic use of milk progesterone assays at about

*Anon. (1984) Dairy Herd Fertility. Ministry of Agriculture, the expected time of
return to service can help in over-Fisheries and Food, Reference Book 259, pp.
1–80. HMSO,*

London.

coming problems of long interservice intervals. High

Archbald, L.F., Risco, C., Chavatte, P., Constant, S., Tran,

progesterone followed by two successive days of low

*T., Klapstein, E. & Elliot, J. (1993) Estrus and pregnancy progesterone is an
indication that service should be*

rate of dairy cows given one or two doses of prostaglandin

carried out on the next day. Eddy and Clark (1987)

F2 alpha 8 or 24 hours apart. Theriogenology, 40, 873–

demonstrated, in four large dairy herds, that the effi-

84.

ciency of heat detection after service could be improved

Ball, P.J.H. & Jackson, P.S. (1984) The use of milk progesterone greatly by the use of milk progesterone assays. The pre-profiles for assessing the response to cloprostenol treatment

Problems Associated with Oestrous Cyclicity • 549

of non-detected oestrus in dairy cattle. British Veterinary Journal, **140**, 543–9.

Rattenberger, E. (1983) Analysis of factors influencing

Ball, P.J.H. & Lamming, G.E. (1983) Diagnosis of ovarian

reproductive performance of the dairy cow by progesterone

acyclicity in lactating dairy cows and evaluation of treat-

ment with gonadotrophin-releasing hormone or a proges-

assay in milk fat. British Veterinary Journal, **139**, 29–37.

Cook, D.L., Smith, C.A., Parfet, J.R., Youngquist, R.S., Brown, terone releasing intravaginal device. British Veterinary

E.M. & Garverick, H.A. (1990) Fate and turnover rate of

Journal, **139**, 522–7.

ovarian follicular cysts in dairy cattle. Journal of Reproduc-Barrett, D.C. (2001) Cattle Fertility Management in the UK.

tion and Fertility, **90**, 37–46.

Cattle Practice, **9**, 59–68.

Darwash, A.O. & Lamming, G.E. (1997) Strategic milk prog-

Beam, S.W. & Butler, W.R. (1997) Energy balance and ovarian esterone monitoring. *Cattle Practice*, **5**, 353–5.

follicle development prior to the first ovulation postpartum

Dawson, F.L.M. (1975) Accuracy of rectal palpation in the

in dairy cows receiving three levels of dietary fat. *Biology diagnosis of ovarian function in the cow. Veterinary Record, of Reproduction*, **56**, 133–42.

96, 218–20.

Benmrad, M. & Stevenson, J.S. (1986) Gonadotrophin-

De Rensis F. & Peters A.R. (1999) The control of follicular releasing hormone and prostaglandin F2a for postpartum

dynamics by PGF2a, GnRH, hCG and oestrus synchroniza-

dairy cows: estrous, ovulation and fertility traits. *Journal of tion in cattle. Reproduction in Domestic Animals*, **34**, 49–59.

Dairy Science, **69**, 800–11.

Dijkhuizen, A.A., Huirne, R.B.M., Jalvingh, A.W.

&

Bloomfield, G.A., Morant, S.V. & Ducker, M.I. (1986) A

Stelwagen, J. (1997) Economic impact of common health

survey of reproductive performance in dairy herds. Char-

and fertility problems. In *Animal Health Economics –*

acteristics of the patterns of progesterone concentrations in

Principles and Applications. (ed. by A.A Dijkhuizen & R.S.

milk. *Animal Production*, **42**, 1–10.

Morris) pp. 41–58. *Post Graduate Foundation in Veterinary*

Booth, J.M. (1988) *The milk progesterone test as an aid to*

Science, University of Sydney.

diagnosis of cystic ovaries in dairy cows. Veterinary Record, Dobson, H.,
Douthwaite, R., Nobel, K.M., O'Donnell, M.J.,

123, 437–9.

Ribadu, A.Y., Tebble, J.E. & Ward, W.R. (2001a) *Cystic*

Boyd, H. (1984) *Aids to oestrus detection – a review. In Dairy ovaries in cattle.*
Cattle Practice, **9**, 185–9.

Cow Fertility (ed. by R.G. Eddy & M.J. Ducker), pp. 60–7.

Dobson, H., Tebble, J.E., Smith, R.F. & Ward, W.R. (2001b) *Is British Veterinary*
Association, London.

stress really all that important? Theriogenology, **55**, 65–73.

Boyd, H. & Munro, C.D. (1980) *Experience and results of a*

Douthwaite, R. & Dobson, H. (2000) *Comparison of different herd health*
programme – rectal palpation and progesterone

methods of diagnosis of cystic ovarian disease in cattle and

assays in fertility control. In IX International Congress on an assessment of its
treatment with a progesterone-Animal Reproduction and AI, Vol. 2, pp. 373–80.

releasing intravaginal device. Veterinary Record, **147**, 355–9.

Boyd, H. & Reed, H.C.B. (1961) *Investigations into the*

Eddy, R.G. & Clark, P.L (1987) *Oestrus prediction in dairy incidence and causes*
of infertility in dairy cattle. Fertility cows using an ELISA progesterone test.

Veterinary Record, variations. *British Veterinary Journal*, **117**, 18–35.

120, 31–4.

Boyd, J.S. & Omran, S.N. (1991) *Diagnostic ultrasonography* Esslemont, R.J.,
Bailie, J.H. & Cooper, M.J. (1985) *Fertility of the bovine female reproductive*
tract. In Practice, **3**, Management in Dairy Cattle, pp. 1–143. Collins, London.

109–18.

Esslemont, R.J. & Kossaibati, M.A. (1997) *Culling in 50 dairy* Britt, J.H. (1987)
Detection of oestrus in cattle. Veterinary herds in England. Veterinary Record,
140, 36–9.

Annual, **27**, 74–80.

Etherington, W.G., Bosu, W.T.K., Martin, S.W., Cote, J.F., Doig, Britt, J.H.
(1995) *Relationship between postpartum nutrition*,

P.A. & Leslie, K.E. (1984) *Reproductive performance*

weight loss and fertility. Cattle Practice, **3**, 79–83.

in dairy cows following postpartum treatment with

Brown, J.L., Schoenemann, H.M. & Reeves, J.J. (1986) *Effect gonadotrophin*
releasing hormone and/or prostaglandin: a

of FSH treatment on LH and FSH receptors in chronic

field trial. Canadian Journal of Comparative Medicine, **48**, cysticovarian-
diseased dairy cows. Journal of Animal

245–50.

Science, **62**, 1063–71.

Fishwick, J.C. (1993) *An assessment of the accuracy of manual*

Bulman, D.C. & Lamming, G.E. (1978) *Milk progesterone*

rectal palpation as compared with ultrasound imaging of the levels in relation to conception, repeat breeding and factors genital system, milk and plasma progesterone concentration influencing acyclicity in dairy cows. *Journal of Reproduction*, and the response to therapy in cows not observed in and Fertility, **54**, 447–58.

oestrus. *RCVS Diploma in Cattle Health and Production*
Burton, N.R. & Lean, I.J. (1995) Investigation by meta-Dissertation, pp. 1–47. Royal College of Veterinary Surgeons Library, London.

post partum on the reproductive performance of dairy

Foulkes, L.A. & Goodey, R.G. (1988) Fertility of Friesian cows cattle. *Veterinary Record*, **136**, 90–4.

on the second, third and fourth days of low milk progesterone

Butler, W.R. (1999) Nutritional effects on resumption of terone concentration. *Veterinary Record*, **122**, 135.

cyclicity and on conception rate. *Fertility in the High-*

Fourichon, C., Seegers, H. & Malher, X. (2000) Effect of Producing Dairy Cow, BSAS Occasional Meeting, Galway, disease on reproduction in the dairy cow: a meta-analysis. Ireland, September 1999, pp. 13–14.

Theriogenology, **53**, 1729–59.

Canfield, R.W. & Butler, W.R. (1990) Energy balance and postpartum anorexia in dairy cows. *Journal of Dairy Science*, **73**, 1000–1005.

satellite LH secretion in early postpartum dairy cattle. *Domestic Animal Medicine*, **34**, 255–260.

Animal Endocrinology, **7**, 323–30.

63.

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Garnsworthy, P.C. & Webb, R. (2000) Nutritional influences on fertility in dairy cows. *Cattle Practice*, **8**, 401–5.

longed service intervals in cattle. *New Zealand Veterinary Journal*, **50**, 10–15.

Journal, **34**, 128–32.

Journal of Dairy Science, **80**, 995–1004.

Mottram, T. (1997) Automatic monitoring of the health and welfare of dairy cows. *Proceedings of the 10th International Conference on Computerized Veterinary Medicine*, pp. 10–15.

Heuwieser W., Oltenacu P.A., Lednor A.J. & Foote R.H.

metabolic status of dairy cows. *Livestock Production Science*, **50**, 101–108.

(1997) Evaluation of different protocols for prostaglandin treatment of dairy cows. *Journal of Dairy Science*, **80**, 1000–1005.

Science, **48**, 209–17.

synchronization to improve reproductive performance in dairy cows. *Proceedings of the 10th International Conference on Computerized Veterinary Medicine*, pp. 10–15.

Nash, J.G., Ball, L. & Olson, J.D. (1980) Effects of GnRH on reproductive performance of dairy herds with low estrus detection efficiency. *Journal of Dairy Science*, **63**, 1000–1005.

partum dairy cows. *Journal of Animal Science*, **50**, 1017–21.

Humbolt, P. (1982) Respective incidence of late embryonic

O'Callaghan, D. & Boland, M.P. (1999) Nutritional effects on mortality and post insemination anoestrus in late returns to

ovulation, embryo development and the establishment of

oestrus in dairy cows. In *Factors Influencing Fertility in the pregnancy in ruminants*. *Animal Science*, **68**, 299–314.

Postpartum Cow (ed. by H. Karg, & E. Schallenberger), pp.

O'Callaghan, D., Lozanot, J.M., Fahey, J., Gath, V., Snijders, S.

298–304. Nijhoff, The Hague.

& Boland, M.P. (2000) Recent developments in the effect of Imakawa, K., Day, M.L., Garcia-Winder, M., Zalesky, D.D.,

nutrition on fertility in dairy cows. *Irish Veterinary Journal*, Kittok, R.J., Schanbacher, B.D. & Kinder, J.E. (1986)

53, 417–25.

Endocrine changes during restoration of oestrous cycles fol-

O'Farrell, K.J. & Hartigan, P.L (1984) The treatment of non-lowing induction of anestrus by restricted nutrient intake in

detected oestrus (NDO) in dairy cows with cloprostenol.

beef heifers. *Journal of Animal Science*, **63**, 565–71.

Irish Veterinary Journal, **38**, 23–8.

Kassa, T., Ahlin, K-A. & Larsson, K. (1986) Profiles of prog-Omran, S.N., Ayliffe, T.R. & Boyd, J.S. (1988) Preliminary esterone in milk and clinical ovarian findings in postpartum

observations of bovine ovarian structures using B-mode

cows with ovarian dysfunctions. Nordisk Veterinaer Medicin, real time ultrasound. Veterinary Record, 122, 465–6.

38, 360–9.

Opsomer G., Mijten P., Coryn M. & de Kruif A. (1996) Post-

Kastelic, J.P., Knopf, L. & Ginther, O.J. (1990) Effect of day of partum anoestrus in dairy cows: a review. Veterinary

prostaglandin F2a treatment on selection and development

Quarterly, 18, 68–75.

of the ovulatory follicle in heifers. Animal Reproduction

Ott, R.S., Breulaff, K.N. & Hixon, J.E. (1986) Comparison of Science, 23, 169–80.

palpable corpora lutea with serum progesterone concentra-

Knight, C.H. & Sorensen, A. (1998) Fertility parameters of tions in cows. Journal of the American Veterinary Medical

cows with extended lactations. Cattle Practice, 6, 379–82.

Association, 188, 1417–19.

Koelsch, R.K., Aneshansley, D.J. & Butler, W.R. (1994)

Ott, R.S. & Gustaffson, B.K. (1981) Therapeutic application of Milk progesterone sensor for application with dairy

prostaglandins for post partum infections. Acta Veterinaria cattle. Journal of Agricultural Engineering Research, 58, Scandinavica (Suppl.), 77, 363–90.

115–20.

Penny C.D., Lowman, B.G., Scott, N.A. & Scott, P.R. (2000) Langley, O.H. &

O'Farrell, K.J. (1979) *The use of Gn-RH to Repeated oestrus synchronisation of beef cows with*

stimulate early resumption of oestrous cycles in dairy cows.

progesterone implants and the effects of a gonadotrophin-

Irish Journal of Agricultural Research, 18, 157–65.

releasing hormone agonist at implant insertion. Veterinary McDougall S. (2001) Reproductive performance of anovula-Record, 146, 395–8.

tory anoestrous postpartum dairy cows following treatment

Pieterse, M.C., Taverne, M.A.M., Kruip, T.A.M. & Willemse, with two progesterone and oestradiol benzoate-based proA.H. (1990) Detection of corpora lutea and follicles in cows:

tocols, with or without resynchrony. New Zealand Veterinary a comparison of transvaginal ultrasonography and rectal

Journal, 49, 187–94.

palpation. Veterinary Record, 126, 552–4.

McLeod, B.J., Foulkes, J.A., Williams, M.E. & Weller, R.F.

Richardson, G.F., Archbald, L.F., Galton, D.M. & Godke, R.A.

(1991) Predicting the time of ovulation in dairy cows using

(1983) Effect of gonadotropin-releasing hormone and

on-farm progesterone kits. Animal Production, 52, 1–9.

prostaglandin F2a on reproduction in postpartum dairy

MacMillan, K.L. (1983) Prostaglandin responses in dairy herd

cows. Theriogenology, 19, 763–70.

breeding programmes. *New Zealand Veterinary Journal*, **31**, Royal, M.D., Darwash, A.O., Flint, A.P.F., Webb, R., Woolliams,

110–13.

J.A. & Lamming, G.E. (2000) Declining fertility in dairy

Mann, G.E., Keatinge, R., Hunter, M., Hedley, B.A. &

cattle: changes in traditional and endocrine parameters of

Lamming, G.E. (1998) Milk progesterone monitoring of

fertility. *Animal Science*, **70**, 487–501.

suckler cow fertility. *Cattle Practice*, **6**, 383–4.

Rukkwamsuk, T., Kruip, T.A.M. & Wensing, T. (1999) Rela-

Markusfeld, O. (1987) Inactive ovaries in high-yielding dairy

tionship between overfeeding and overconditioning in the

cows before service: aetiology and effect on conception.

dry period and the problems of high producing dairy cows

Veterinary Record, **121**, 149–53.

during the postparturient period. *The Veterinary Quarterly*, Martinez, J. &

Thibier, M. (1984) Fertility in anoestrous dairy **21**, 71–7.

cows following treatment with prostaglandin F_{2a} or the

Saloniemi, H., Groelm, Y. & Syvaejaervi, J. (1986) An epi-

synthetic analogue fenprostalene. *Veterinary Record*, **115**, demiological and genetic study on registered diseases in

57–9.

Finnish Ayrshire cattle. II. Reproductive disorders. Acta

Mayer, E., Francos, G. & Neria, A. (1987) Eierstocksbefunde Veterinaria Scandinavica, 27, 196–208.

und Fertilitäts-Parameter bei Kühen mit ‘unbeobachteter

Savio, J.D., Boland, M.P., Hynes, N. & Roche J.F. (1990)

Brunst’. Tierärztliche Umschau, 42, 506–9.

Resumption of follicular activity in the early post-partum

Problems Associated with Oestrous Cyclicity • 551

period of dairy cows. Journal of Reproduction and Fertility, terone at selected intervals after insemination of synchro-88, 569–79.

nized heifers on pregnancy rates and resynchronization of

Schofield, S.A., Kitwood, S.E. & Phillips, C.J.C. (1999) The returns to service. Theriogenology, 46, 1117–30.

effects of a post partum injection of prostaglandin F2a

Waldmann, A., Ropstad, E., Landsverk, K., Sørensen, K.,

on return to oestrus and pregnancy rate in dairy cows.

Sølverød, L. & Dahl, E. (1999) Level and distribution of

Veterinary Journal, 157, 172–7.

progesterone in bovine milk in relation to storage in the

Scully, A., Hunter, M., Gregson, K., Lamming, G.E. &

mammary gland. Animal Reproduction Science, 56, 79–91.

Darwash, A.O. (2000) The use of on-farm software for auto-

Warren, M.E. (1984) Biological targets for fertility and their matic interpretation of milk progesterone data: an aid to

effects on herd economics. In *Dairy Cow Fertility* (ed. by dairy fertility management. *Cattle Practice*, **8**, 407–10.

R.G. Eddy & M.J. Ducker), British Veterinary Association,

Seabrook, M.F. & Wilkinson, J.M. (2000) Stockpersons' atti-London. pp. 1–14.

tudes to the husbandry of dairy cows. *Veterinary Record*, Wathes, D.C., Robinson, R.S., Pushpakumara, A., Cheng, Z. & **147**, 157–60.

Abayasekara, D.R.E. (1998) Nutritional effects on repro-

Seguin, B.E. (1981) Use of prostaglandins in cows with unob-

ductive performance in dairy cows. *Cattle Practice*, **6**, 371–7.

served oestrus. *Acta Veterinaria Scandinavica* (Suppl.), **77**, Watson, C.L. (1996) Milk progesterone analysis – an under-343–53.

used asset in fertility control of the dairy cow. *Cattle Prac*-Stagg, K., Diskin, M.G., Sreenan, J.M. & Roche, J.F. (1995) *Fol-tice*, **4**, 277–80.

licular development in long-term anoestrous suckler beef

Webb, R., Garnsworthy, P.C., Gong, J.G., Gutierrez, C.G.,

cows fed 2 levels of energy postpartum. *Animal Reproduc-*

Logue, D., Crawshaw, W.M. & Robinson, J.J. (1997) *Nutri-*

tion Science, **38**, 49–61.

tional influence on subfertility in cattle. *Cattle Practice*, **5**, Stagg, K., Spicer, L.J., Sreenan, J.M., Roche, J.F. & Diskin, M.G.

361–7.

(1998) Effect of calf isolation on follicular wave dynamics,

Webb, R., Royal, M.D., Gong, J.G. & Garnsworthy, P.C. (1999) gonadotropin and metabolic hormone changes, and interval

The influence of nutrition on fertility. Cattle Practice, 7, to first ovulation in beef cows fed either of two energy levels 227–34.

postpartum. Biology of Reproduction, 59, 777–83.

Whitaker, D.A., Kelly, J.M. & Smith, S. (2000) Disposal and Stevenson J.S. (2001) A review of oestrous behaviour and

disease rates in 340 British dairy herds. Veterinary Record, detection in dairy cows. In Fertility in the High Producing 146, 363–7.

Dairy Cow. Occasional Publication No. 26, pp. 43–62.

Williamson, N.B., Morris, R.S., Blood, D.C. & Cannon, C.M.

British Society of Animal Science London.

(1972) A study of oestrous behaviour and oestrus detection

Taylor, V.J., Beever, D.E. & Wathes, D.C. (2001) Plasma IGF-methods in a large commercial dairy herd. 1. The relative

1 levels in relation to ovarian function in high producing

efficiency of methods of oestrus detection. Veterinary

dairy cows. Cattle Practice, 9, 197–201.

Record, 91, 50–8.

Tebble, J.E., O'Donnell, M.J. & Dobson, H. (2001) Ultrasound Young, I.M. (1989) Dinoprost 14-day oestrus synchronisation

diagnosis and treatment outcome of cystic ovaries in cattle.

schedule for dairy cows. Veterinary Record, 124, 587–8.

Veterinary Record, 148, 411–13.

Young, I.M. & Anderson, D.B. (1986) First service conception Van Cleeff, J., Macmillan, K.L., Drost, M., Lucy, M.C. &

rate in dairy cows treated with dinoprost tromethamine

Thatcher, W.W. (1996) Effects of administering proges-
early post partum. *Veterinary Record*, **118**, 212–13.

Chapter 36

Failure to Conceive and

Embryonic Loss

D.C. Barrett, H. Boyd and M. Mihm

Manifestation

552

of the pelvic ligaments and voice change. In general,

Aetiology

554

however, the cow that returns to service presents an

Stage 1 Failure of ovulation after oestrus

554

apparently normal oestrus as regards both behaviour

Stage 2 Fertilization failure

557

and visible signs.

Stage 3 Embryonic death

559

The way in which the client presents return to service

Stage of failure not known

562

cases affects the clinician's response to the problem. It

Diagnosis

563

is presented either as an individual cow problem or as

Treatment

566

Prognosis

569

a herd (or group) problem, and it is presented with or

Prevention

572

without evidence of past or present pathology.

Individual cows

Manifestation

Individual cases are presented in two quite different

Return to service or detection of non-pregnancy up to

ways, which influence how they are dealt with.

six weeks after insemination/natural service are the

In the first way, some cows are presented to the vet-

ways in which poor pregnancy rate due to failure to

erinarian that have been served two or three times and

conceive and embryonic losses are presented. This can

are still not pregnant. To understand these cases prop-

have a major economic impact in any herd, but is

erly they have to be considered within the context of

particularly noticeable where a tight seasonal calving

the entire population of cows and heifers in the herd.

pattern is sought.

In any group of apparently normal cows served cor-

rectly by a fertile bull or inseminated properly with

good semen, either for the first time after calving or at

Interservice intervals

a repeat service, a considerable number will return to

The interval in days that has elapsed between the

service depending on first-, second- and third-service

unsuccessful service and the next oestrus is part of

pregnancy rates in each particular herd. Most of these

the manifestation of the condition of return to service.

animals are only temporarily infertile and the majority

The concept of normal and abnormal interservice inter-

will become pregnant within an acceptable time.

vals has been discussed in connection with postservice

Although there is some understanding of the stage at

anoestrus (see p. 531). While the distribution of inter-

which these losses occur, i.e. whether there is fertiliza-

service intervals varies greatly between herds, reflecting

tion failure or early embryonic death, the aetiology is

in part the management efficiency in the herd, in many

known to only a limited extent. For this reason these

cattle populations fewer than 50 per cent have a normal

returns appear to happen at random.

interservice interval of 18–24 days.

Field studies show that about 12 per cent of cows are

still not pregnant after three services as is demonstrated

in Table 36.1, which is taken from a population of 5744

Observable abnormality

cows with a first insemination pregnancy rate of 60.6

Another manifestation of return to service is the nor-

per cent (Boyd & Reed, 1961a). 'Actual results' are
 mality or otherwise of the cow at the return oestrus. The
 compared with the 'expected results' that would have
 farmer can observe certain deviations from normality
 occurred if all subsequent inseminations had had a
 in these cows, in particular the presence of a purulent
 pregnancy rate of 60.6 per cent. However, in recent
 discharge from the vulva. The farmer may also report
 years a drop in pregnancy rates of approximately one
 aberrant sexual behaviour. In relevant cases, the obser-
 per cent per annum has occurred in high-genetic-merit
 vant client may notice some of the changes associated
 dairy cattle populations (Royal et al., 2000; Barrett, with chronic cystic ovarian
 disease, such as slackening
 2001). This has resulted in expected pregnancy rates of
 552

Failure to Conceive and Embryonic Loss • 553

Table 36.1

The percentage of cows that required various numbers of inseminations for pregnancy with a first insemination pregnancy rate of 60.6 per cent.

Number of

Actual results

Expected results

inseminations

required for

Percentage

Cumulative

Percentage

Cumulative

pregnancy

percentage

percentage

1

60.6

60.6

60.6

60.6

2

20.4

81.0

23.9

84.5

3

7.2

88.2

9.4

93.9

>3 or culled

11.8

100.0

6.1

100.0

50 per cent or less to a single service, thus increasing the

There is no realistic incidence figure for chronic

proportion of animals within a herd not pregnant after

repeat breeders because their retention in the breeding

three services.

herd is dependent on extraneous factors but clearly is

Farmers often wait until a cow has had three

unlikely to be more than 6 per cent of the breeding

unsuccessful services before presenting her to the

population, although it may be higher in individual

veterinarian and Table 36.1 shows that (with

herds. The number in any given herd will be determined

certain reservations) about half of these cows, 6.1% by various economic factors which dictate at what point versus 11.8%, are not pregnant because of the same it becomes more economic to cull a cow and replace her (unknown) factors that establish the level of the first rather than persist in attempting to get her in calf. insemination pregnancy rate. It is therefore to be Overall culling rates in UK dairy herds are about 20–25 expected that half of these cows will not exhibit any per cent per annum (Esslemont & Kossaibati, 1997; diagnosable cause of return to service and that their Whitaker et al., 2000). Poor fertility is the single most expectation of pregnancy at the next service is about important reason for involuntary culling. With 30–40 the same as that for first insemination. However, per cent of cull cows being culled for infertility, this pregnancy rates to three or more services are usually represents almost 10 per cent of dairy cows calving each reduced due to an accumulation of animals with repro-year (Kossaibati & Esslemont, 1995). The exact cost to ductive problems. Warren (1984) cited published data

the farmer of an involuntary cull will vary from farm to farm and from time to time, depending on the cost of a pregnancy rates to first service ranged from 47–57%. Within replacement, the value of a cull cow, the milk price and these populations, however, the herd pregnancy rates availability (and cost) of milk quota, loss of genetic merit and many other factors. For this reason it is

In the second instance, other cases, for which the term 'chronic repeat breeder' might be used, are presented commercial dairy farm it is probably between £600 and £800 per cull cow at the present time (2003).

a long period of time. These cases have been differentiated into apparently normal repeat breeders and

Herd or group problems

those with abnormalities on the basis of cycle length, sexual behaviour and detectable abnormality, a differ-

Return to service may affect many animals in the herd

entiation of limited clinical value. These chronic repeat in which case the client is very worried about the extent breeders require a different approach from the cases of the problem and the serious effect it will have on production and profits. For an assessment of the condition the owners regard as particularly valuable and therefore it is necessary to analyse the breeding records to fore the clinician can devote more time and money to determine pregnancy rates to first, second and further their diagnosis and treatment. The prognosis in chronic inseminations (or other measure of the success rate of repeat breeders is inevitably poor because they have services) and interservice intervals (see also Chapter 41(b)).

without success. It is animals of this type that may be hospitalized in teaching and research institutions where The range of incidence of return to service is very wide. In a population of herds with any given mean they are given expensive diagnostic and therapeutic

herd pregnancy rate there is a tendency for the herd treatment, although this is becoming more and more averages to be distributed at random round this mean, uneconomic within the UK.

as demonstrated in Table 36.2. Just over 40 years ago,

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Table 36.2

Distribution (%) of herd average first insemination pregnancy rates in 191 dairy herds (Boyd & Reed, 1961a).

Herd first insemination pregnancy rate (herd average)

Total

<31

31–40

41–50

51–60

61–70

71–80

>80

3.1%

3.7%

16.2%

25.1%

27.2%

19.4%

5.2%

99.9%

the overall herd first insemination pregnancy rate in

Box 36.1

Reasons for unsuccessful services.

191 herds was 59.9 per cent (Boyd & Reed, 1961a).

Stage 1

Ovulation failure

Although not demonstrated here, it is usual for the dis-

Stage 2

Fertilization failure

tribution to be skewed towards the lower end of the

Stage 3

Loss of the conceptus, due to early (3 week return

scale. The skewed distribution of these data tends to be

intervals) or late embryonic death (3–6 week return

seen in all studies, however in more recent surveys the

intervals) or fetal death (>6 week return intervals)

Stage of failure unknown

first insemination pregnancy rates are lower than those reported by Boyd and Reed (1961a). For example, the mean first service pregnancy rate in 90 herds in the UK was reported as 47.3 per cent in the 1992/1993 calving season (Kossaibati & Esslemont, 1995). National Milk such as bovine virus diarrhoea virus infection (see Records (NMR) data from approximately one million p. 578) or leptospirosis (see pp. 580, 735).

dairy cattle in 9000 herds showed that the pregnancy

- The incidence of chronic repeat breeders, which to rate to first service (based on available pregnancy diagnosis data and non-return rates at 48 days) was 52.5 per cent in September 2000 (Barrett, 2001). This will be an overestimate of the true pregnancy rate as a proportion*

some extent relates to the background effect.

Aetiology

of the data are based on non-return rates which will include some cows culled that were not rebred after

There are three main stages at which failure to achieve failing to conceive. Royal et al. (2000) reported that 265

or maintain a pregnancy can occur. Although it is often first services out of 667 resulted in pregnancy in their not possible to assign an individual case to one of these study of Holstein Friesian dairy cows between October 1995 and June 1998. This equates to a first service pregnancy rate of 39.7 per cent. Given these recent data it seems reasonable to accept a target first service pregnancy rate of 50 per cent in modern dairy herds, while accepting that in some high yielding herds this may be difficult to achieve.

Box 36.1.

seems reasonable to accept a target first service pregnancy rate of 50 per cent in modern dairy herds, while accepting that in some high yielding herds this may be

Stage 1 Failure of ovulation after oestrus

difficult to achieve.

The most important condition in which there is oestrus

It is helpful to think of the pregnancy rate for any

without ovulation is cystic ovarian disease. It is im-

individual herd as dependent on the combined effects

important to define 'cystic ovarian disease' because not all

of the following factors:

cases of ovarian cysts are pathological (see Box 36.2).

- *The random variation around the mean of the*

This traditional definition implied that cysts were population of herds.

static structures; however, cysts should be considered

- *The background effect, which is dependent on man-dynamic both in structure and function (Cook et al., 1990). When studying the aetiology of ovulation failure to be constant over a long period. Examples, any of the conclusions reached are affected by which animals which individually would tend to result in low herd are included as clinical cases. For example, cows pregnancy rates, are inaccurate oestrous detected as having cystic ovarian disease at routine, unsatisfactory feeding, a high incidence of preservice veterinary examination will form a different endometritis (see p. 521) or poor semen handling population from cystic ovarian disease cases detected in and insemination techniques (see Chapter 39). repeat breeder cows.*

- *Specific pathogenic causes, such as a subfertile bull*

Histological and biochemical examination of ovarian or venereal diseases such as campylobacteriosis, or cysts demonstrates that cysts are complex structures. other infectious conditions known to affect fertility

On clinical examination and gross anatomical inspection

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Box 36.2

Definition of cystic ovarian disease (see also

Box 36.4

Factors that predispose to or cause cystic ovarian

Chapter 35).

disease.

- *Persisting, steroid-secreting follicles >2.5cm in diameter*
- *Heredity*
- *No functional corpus luteum present for more than 10*
- *High yield*

days

- *Age*
- *Abnormal sexual cycle (anoestrus, nymphomania, viril-*
- *Exogenous oestrogens*

ism)

- *Nutrition and season of the year*
- *More than six weeks after calving*
- *Dystokia and periparturient disease*

Heredity: A familial predisposition to cystic ovarian

Box 36.3

The possible pathogenesis of cystic ovarian

disease through sire or dam has been recognized for disease.

many years (Bane, 1964). Other studies have produced

- *Insufficient luteinizing hormone (LH) release:*

less clearcut results or even failed to detect a hereditary

hypothalamic–pituitary dysfunction causing lack of GnRH

effect at all (Dohoo & Martin, 1984a). It is possible that release (commonest cause)

this difference is partly due to the stage at which

- *Insufficient production of LH by the pituitary (rare)*

diagnosis is carried out. If the incidence is assessed at

- *Failure of the dominant follicle to respond to LH*

routine preservice checks most of the detected cysts are

likely to be attributed to environmental factors rather

than heredity; on the other hand, one might expect con-

tion, however, there appear to be two main types of
siderably more evidence of hereditary predisposition if
cysts: follicular cysts, which are like large follicles, and
the assessment is made on a sample of repeat breeders.
luteal cysts, in which obvious luteal tissue produces a
While most veterinarians are generally aware that
thicker cyst wall. These may be difficult to differentiate
there is a hereditary component in cystic ovarian
from a corpus luteum with a central lacuna, which
disease, their clients want to know to what extent breed-
frequently occurs during the normal oestrous cycle
ing a replacement heifer from a cow with cystic ovarian
(Okuda et al., 1988).

disease will increase the risk of that daughter having the
It is well known that follicular cysts can occur and
disease. It is not easy to answer this because cystic
spontaneously resolve during the first few weeks after
ovarian disease may not occur in a daughter until she
calving in some cows that then return to normal cyclic
has had several calves and so field studies take many
ovarian function, albeit later than normal counterparts

years and involve complex statistical analysis.

(Savio et al., 1990). It is often suggested that cows with Early studies carried out by Henricson (1957) helped

cystic follicles up to six weeks after calving should be to answer the question. He analysed data from two regarded as normal and not be interfered with unless artificial insemination (AI) centres of cases presented they are nymphomaniac or show other signs of at the time of insemination and classified cows as: abnormality.

- *Having cystic ovarian disease on more than one*

Box 36.3 is to some extent hypothetical but should occasion in a service period;

act as a reminder that there are several possible routes

- *Having only one observation of cystic ovarian*

that can result in cystic ovarian disease, among them disease; and

long-term exposure to oestradiol or subnormal concen-

- *Without cystic ovarian disease.*

trations of progesterone (Dobson et al., 2001a) and

cortisol release following stress (Dobson et al., 2001b).

The effect of age was taken into account and the results

*There have also been suggestions that energy im-
expressed as an average frequency of the condition,
balance due to high milk yields may be contributory,
where 1.00 is equivalent to an incidence of 100 per cent
although this has not been substantiated by correlation
(Table 36.3).*

with either b-hydroxybutyrate concentrations or body

*Henricson (1957) also studied the effect of the sire
condition score (Dobson & Nanda, 1992; Tebble et al., and noted that the
average frequency of cystic ovarian
2001).*

disease in individual bulls' daughters ranged from 0.00

*There have been many studies of the causes of cystic
to 0.258. Whether the low incidence of cystic ovarian
ovarian disease and factors that predispose to the con-
disease in beef breeds is directly due to hereditary
dition are listed in Box 36.4. There is lack of unanimity
factors, to the lower milk production or to some other
about causes, which may reflect differences between
factor is not known.*

the populations of cattle selected for investigation as well as variations in the definition of cystic ovarian
High milk yield: Cystic ovarian disease may be related disease.
to high milk yield. It occurs most frequently at the peak
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Table 36.3

Dam effect on incidence of cystic ovarian disease
arise when oestrogenic substances, such as those found
(COD) in daughters (Henricson, 1957).
in mouldy brewers' grains, are eaten.

Number of times COD

Average frequency of

Seasonal effect: A very definite seasonal effect has
diagnosed in dam in

COD in daughters

been reported by many authors, with most reports indi-
one service period

cating that the incidence is highest in winter (Dohoo et

Centre 1

Centre 2

al., 1984a,b; Saloniemi et al., 1986). The effect can be so great that Bane (1964)

parous cows than in first calvers. There is a secondary tional effects, and could also include the hours of peak of cases late in lactation; presumably these are daylight, temperature extremes and stressors such as cases that have been picked up on the examination housing.

of repeat breeders. Bartlett et al. (1986b) discussed, without coming to a conclusion, whether high milk yield

Dystokia and periparturient disease: Some information

causes cystic ovarian disease, cystic ovarian disease

suggests that dystokia and periparturient diseases may

causes high yield or there is a common cause of both!

predispose to cystic ovarian disease. It is possible that

Saloniemi et al. (1986) thought that the relationship

preparturient factors that cause the postpartum condi-

between cystic ovarian disease and ketosis was due

tions also affect the incidence of cystic ovarian disease,

primarily to high milk yield, leading to greater energy

for example overfeeding leading to fatty liver (Reid et

demands that in turn resulted in both ketosis and inter-

al., 1979). It has been demonstrated experimentally that ference with LH production or release.

damage to the endometrium can prolong the existence

Some workers have been unable to find an adverse effect of cystic follicles and so inhibit spontaneous recovery of high milk yield on the occurrence of cystic (Fathalla et al., 1978) (see also Chapter 34).

ovarian disease (Dohoo & Martin, 1984a).

Other causes: It is possible that gross adhesions can bind the ovary so tightly that ovulation does not take

Age: It is widely accepted that cystic ovarian disease is place, but this must be a rare phenomenon. Of much

rare in heifers before their first calving, not common in greater importance is the concept that in cows which first-lactation cows and then increasingly observed in had other reproductive disorders there were possibly mature cows, with possibly some slight fall off in old more than the expected cases of cystic ovarian disease animals.

(Jasko et al., 1984).

Reviews of the literature show that incidence ranges

Exogenous oestrogens: Administration of oestrogens

from about 6 to nearly 20 per cent (Bartlett et al., 1986b; in the follicular phase will cause a premature LH peak.

Garverick, 1997). However, diagnostic methods affect
This will not cause ovulation if no dominant follicle
the reported incidence figure, for example Coleman et
is present at the time. If oestrogens are administered
al. (1985) found that the single most important factor shortly before dominant
follicle selection, the cow may
that influenced the incidence was the veterinarian who
not be able to produce a second LH peak due to down
made the diagnosis! Selection of cases is also important,
regulation of the hypothalamic response to oestradiol
as has been discussed already.
and consequently the dominant follicle will continue to
It is generally agreed that follicular cysts make up
grow and not ovulate. This may be one explanation why
about two-thirds of recognized cases of cystic ovarian
a single large dose of an oestrogen for therapeutic pur-
disease (Booth, 1988; Garverick, 1997) although again
poses may be followed later by cystic ovaries. Recent
this figure depends a little on the diagnostic method and
transitions to organic farming may involve grazing
criterion used.

periods on pastures with a high component of oestro-
While the most important reason for failure to
genic plant material; however, a much increased inci-
ovulate is considered to be cystic ovarian disease, there
dence of cystic ovarian disease in such herds has not
are also a proportion of cows who have a delayed ovu-
been reported to date. An analogous situation may
lation after oestrus, resulting in an extended period
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Box 36.5

Causes of fertilization failure.

Box 36.6

Causes of ovarian and bursal adhesions.

- Rectal palpation just before ovulation (bursting pre-
- Trauma

ovulatory follicles, interference with egg flow into the

- Rectal palpation

uterine tubes)

- (Enucleation of corpus luteum – no longer indicated as a
- Interference with egg transport

therapeutic practice in cattle)

- *Interference with sperm transport*
- *Caesarean section – inducing (localized) peritonitis*
- *Delayed ovulation*
- *Ascending infection*
- *Insemination at wrong time in relation to ovulation*

Postpartum metritis

- *Service too soon after calving*

Therapeutic infusion of uterus

- *Poor quality spermatozoa*
- *Descending infection*
- *Poor quality oocyte (e.g. from persistent follicles)*

from peritoneum

- *Failure to serve the cow*
- *Specific infections, e.g.*
- *Metritis and salpingitis*

Ureaplasma sp.

- *Vaginitis and cervicitis*

Mycoplasma sp.

from insemination to ovulation. The incidence of

As transport of the egg depends on secretory and

delayed ovulation can be as high as 11 to 13 per cent,

ciliary activity in the uterine tube, salpingitis will interfere with transport of both eggs and spermatozoa, even (1998).

when the lumen remains patent.

Interference with transport of spermatozoa: Adhesions

Stage 2 Fertilization failure

in the female tract that block egg transport, and have

To achieve fertilization a number of events in the cow

been discussed above, will generally interfere with

(oestrus, ovulation, gamete transport) and in the bull

transport of spermatozoa. There are rare anatomical

(spermatozoa production, insemination) have to take

abnormalities such as white heifer disease (segmental

place and must synchronize with each other. Failure of

aplasia of the paramesonephric ducts), which results in

any one of these events to occur correctly or at the right

blockage of the tubular genitalia at various levels.

time will almost certainly result in fertilization failure.

Poor AI technique, such as insemination in the ante-

The causes of fertilization failure will be discussed,

rior vagina or caudal half of the cervix, will result in the mainly based on the headings shown in Box 36.5. The majority of cases in the loss of much of the inseminate first two causes, interference with gamete transport, are by reflux through the vulva. In contrast, in natural relatively rare occurrences.

service the ejaculate is deposited in the anterior vagina and on the external os of the cervix, but the volume of

Interference with egg transport: After ovulation the egg the ejaculate and the enormous number of spermato-has to be transported to the site of fertilization in the

zoa allow loss by reflux while sufficient spermatozoa are uterine tube. Adhesions affecting the ovary and bursa transported to the uterine tube. Returning to AI, a and blockage of the uterine tube will prevent this hap-rough operator could damage the uterus with the pening. Factors that cause adhesions, which are fairly insemination pipette. While it is unlikely that bad rare, are listed in Box 36.6. The ease with which fluids insemination technique is much of a problem with a and small objects can be transported spontaneously up technician inseminator service, it is very likely that the

or down the uterine tube is demonstrated in two techniques used to investigate uterine tube patency. In one of these tests, phenolsulphonphthalein (PSP) dye is introduced into the uterus and ascends spontaneously through the uterine tube into the peritoneal cavity and becomes absorbed and secreted in urine. In the other test, starch granules deposited in the bursa descend through the uterine tube into the uterus and vagina errors may be made.

Hunter (1999) discusses possible modifications of the technique used for AI involving deep intrauterine insemination and the effects this may have on the transport and storage of spermatozoa within the female reproductive tract.

(Kessy & Noakes, 1979).

Transport in the uterine tube is under hormonal

Delayed ovulation: Delayed ovulation is a condition in control and can be affected by abnormal variations in

which the interval from the onset of oestrus to ovula-

*steroid hormone production and by oxytocin, but the
tion is so much longer than normal that service at the
role of these factors in field conditions is not known.
recommended time does not result in fertilization*

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*because of the ageing of the spermatozoa. While many
of milk or blood taken on the day of insemination will
authors are of the opinion that the condition is rare,
detect insemination during the luteal phase of the cycle
there are a number of papers that suggest that it occurs
but will fail to pick up inseminations that are one or two
quite commonly (Hancock, 1948; Nakao et al., 1984;
days too early or too late. The reported incidence of
Lamming & Darwash, 1998). Watson and MacDonald
luteal phase progesterone levels on the day of insemi-
(1984) studied events around insemination using rectal
nation is variable. The incidence of 5.2 per cent in well-
palpation and assays for progesterone and oestradiol-
run herds reported by Claus et al. (1983), with over 20
17b. They concluded that in cows with a follicle in the
per cent in problem herds, is typical of several other*

ovaries on the day after insemination (which may indicate delayed ovulation if onset of heat occurred more than 30 hours before) this was often due to erroneous inseminations occurred in the summer. Oltner and Edqvist (1981) observed that in herds with a high incidence of wrongly timed inseminations, even inseminations apparently at the correct time resulted in low pregnancy rates, presumably because of other aspects of poor management. It is possible that there is an abnormality of the LH surge or response of the preovulatory dominant follicle to the LH surge. It has been hypothesized that deficiency of energy intake may be involved in the aetiology.

The reasons are misidentification of a cow that actually is in season or misinterpretation of sexual behaviour.

ious, such as jumping another cow, which is thought to be a sign of oestrus. The role of stress is also speculative, but it is often incorrectly used to indicate oestrus. The stockworkers are often under considerable pressure to get all cows served by a target date and are criticised by veterinarians and others, that cows in oestrus are missed too frequently. interesting that corticosteroids can block LH release (Wagner & Li, 1982); also the fact that the adrenal cortex under the influence of adrenocorticotrophic hormone (ACTH) can produce some progesterone may be significant in suppressing the LH surge (Watson & Munro, 1984). This can result in error through an overenthusiastic determination to increase the oestrous detection rate. The importance of stress in bovine reproduction is reviewed by Dobson et al. (2001b). The importance of inseminating only cows in oestrus was demonstrated by Sturman et al. (2000), who reported that in a herd they studied 19 per cent of suprabasal progesterone was described by Jackson et al. A little understood condition called prolonged low or insemuations were performed when progesterone

(1979) as affecting 18 per cent of cows treated with an levels were high in the oestrous cycle or while cows analogue of prostaglandin F2a (PGF2a) and also about were pregnant. Insemination of pregnant cows led to an 18 per cent of control cows. After oestrus there was an estimated 17 per cent induced embryonic death or excessively long period of low progesterone, followed abortion!

by a rise which seemed to indicate that ovulation took

Incorrect timing of insemination in relation to onset

place eventually. The herd incidence ranged from 7 to

of oestrus also occurs. Watson et al. (1987) confirmed 33 per cent and appeared to be low in herds with ade-that insemination 24 hours or more after the cow is first

quate nutrition. Other surveys have not revealed such

seen in oestrus results in a marked reduction in preg-

a high incidence. Some workers think prolonged low

nancy rate, possibly due to ageing of the oocyte before

progesterone is most commonly associated with treat-

fertilization. It is likely that insemination very early in

ment with PGF2a or an analogue causing incomplete

oestrus also causes reduced fertility, possibly due to

luteolysis and subsequent recovery of some luteal function. Independent of its cause, it will lead to persistence is a long-established, successful rule of thumb about the ideal time for insemination (Trimberger, 1948). Cows showing its ovulation. This does not appear to be due to first seen in the morning should be inseminated late that poor fertilization rates, but to more losses during early afternoon. Cows first seen in the afternoon or evening embryonic development (Mihm et al., 1994).

should be inseminated next morning. Cows still in oestrus 24 hours after first being seen should receive a

Insemination at the wrong time in relation to ovulation: second insemination. This pragmatic recommendation

Service during the luteal phase is only a minor problem takes account of the relatively short lifespan of both with natural service but a major one with AI (Sturman spermatozoa and ova within the female tubular tract et al., 2000). However, on farms using hand mated (about 24 hours), the need for capacitation of bull

natural service, a cow restrained in a service crate may spermatozoa (about 6 hours) and the interval from the be mated when she is not in oestrus.

onset of oestrus to ovulation (about 24–30 hours).

Artificial insemination at the wrong time is an impor-

Incorrect timing within oestrus is of little significance

tant cause of fertilization failure. Progesterone assays

with natural service in which the service is usually early

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in oestrus. The large quantity of the ejaculate and the

served. Bulls can develop abnormalities of the penis

great numbers of spermatozoa deposited in the anterior

that prevent normal service, and lack of libido is not

vagina ensure that there is a long-lasting supply of

uncommon (see p. 610). When animals are running

fertile spermatozoa in the uterine tube at the site of

freely, such as heifers or beef cattle, it is essential to

fertilization.

include an adequate number of bulls in the group. If

there are too few bulls for the number of cows, some

Service too soon after calving: It has been known for

cows in oestrus may not be served. The required cow to many decades that service within six weeks of calving bull ratio depends on the type of terrain, the age and results in reduced pregnancy rates. The expected libido of individual bulls (see p. 610).

ception rate increases from approximately 20 per cent, at 20 days post partum, to reach normal rates (at the

Metritis, salpingitis, vaginitis and cervicitis: Infection of time the studies were carried out these were 50 per cent

the tubular genitalia may interfere with the survival of calving rates) from 50 days post partum. The reasons the spermatozoa in transit and so reduce the chance are likely to be both ovarian and uterine in origin so of fertilization. Endometritis and metritis are fairly in some cases the cause is most probably fertilization common (see p. 524) but in addition a number of failure and in others embryonic death. Elimination of species of organisms have been recovered from the infection and restoration of endometrial structure and uterine tubes and may cause salpingitis or directly function after a normal calving requires 5–6 weeks in

affect fertilization, as for example reported by Grahn et al. (1984), in connection with fertilization failure with BVDV infection (see pp. 578, 853). Infectious agents in most animals.

Poor-quality semen or poor insemination technique:

the uterine tube include *Leptospira hardjo* (see p. 735), Poor semen quality may result in either low pregnancy

Ureaplasma spp. and *Mycoplasma bovis*.

rate or no pregnancies at all, mainly through fertilization failure but also to some extent through embryonic

Stage 3 Embryonic death

death. Again there are marked differences between natural service and AI. Because of laboratory control

The stage of the embryo lasts from fertilization of the egg until about day 42 after conception, by which time the organ systems have been laid down and placental AI organization will give poor pregnancy rates.

tion has been established. Failure of maternal recogni-

The exception to this is semen that has been poorly tion of pregnancy will cause early embryonic loss, and

stored or incorrectly handled on farm for use in DIY the animal returns to oestrus at normal intervals (<25 AI systems. Schermerhorn et al. (1986) found that a days). Late embryonic loss occurs after maternal recognition of pregnancy, and animals may show their next results than farmer DIY insemination, although this heat 25 to 40 days after the previous heat.

may depend on the level of training received by DIY In any group of breeding cattle, however normal they inseminators before they begin inseminating their own appear, there will occur a considerable amount of cows. Howells et al. (1999) showed that the amount of embryonic death, most of it within the first three weeks training received with live cows significantly affected after fertilization (Sreenan & Diskin, 1986). Ayalon future pregnancy rates; for those who spent up to three (1978) produced data to show that in repeat breeders days training in an abattoir using live cows there was an the majority of the losses occurred approximately 5–7 increase of 5.9 per cent in the calving rate they achieved

days after insemination, around the time the early in their first year for every day they spent training. If embryo enters the uterus and begins to synthesize its AI technique is satisfactory, poor results with DIY own proteins (Table 36.4). However, the exact timing of inseminations could possibly be related to mishandling early pregnancy losses in the modern high-yielding of the semen. By contrast a bull used for natural service dairy cow is largely unknown. Knowledge of the aetiology may be of reduced or low fertility or even sterile and ology of this important condition is still very limited but serve a number of cows before the owner realizes that it is possible to produce a list of factors that have been something is wrong. Farmers are often not aware that shown to cause embryonic death or are at least very a bull that has been fertile may become infertile or lose likely to be involved (Box 36.7). In the vast majority of libido (see Chapter 38).

cases the actual cause is never established.

Where fresh diluted semen is used, semen that is stored too long will give poor results.

Factors that may contribute to embryonic death

Failure to serve the cow: In certain circumstances when Extreme environmental temperatures: Controlled expera bull is used to serve the cows, a cow in oestrus is not

iments have demonstrated that cattle that are mated

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Table 36.4

Timing of embryonic death, determined by slaughter and dissection of the cow (number and percentage containing viable embryos) (from Ayalon, 1978).

Time of slaughter (days)

Normal cows

Repeat breeders

Number

Percentage

Number

Percentage

2–3

10/12

83

12/17

71

4–5

22/25

88

20/25

80

6–7

10/12

83

5/12

42

8–10

13/18

72

9/18

50

11–13

16/18

89

9/18

50

14–16

16/20

80

10/20

50

17–19

12/21

57

9/21

43

35–42

9/13

69

8/24

35

Box 36.7

Factors that may contribute to embryonic death.

Crosses between indigenous heat tolerant breeds and

European breeds are more heat tolerant than the

- *Extreme environmental temperatures, particularly heat*

imported animals and they are more fertile (see
stress

Chapter 6). The improved fertility appears to be the

- *Specific and non-specific endometritis or metritis*
result of the dam's enhanced ability to control body
- *Specific infections of follicles, gametes and/or embryo*
temperature rather than an inherited ability of the
- *Maternal pre- and postovulatory endocrine environment*
embryo itself to tolerate high intrauterine tempera-
- *Aged gametes*
- *Local trauma*
- tures. The position is less clear with extreme cold, but*
- *Genetic factors, resulting in non-viable genetic defects*
there are indications that a corresponding adverse
- *Nutrition*
effect occurs. The problem could arise during unusually
- *Possible fetal/maternal incompatibility*
cold periods in temperate climates where housing tends
to provide cover rather than warmth.

Metritis/endometritis: Metritis or endometritis in
in a high environmental temperature and kept there
varying degrees of intensity is a common condition
after service exhibit a high rate of embryonic death.
causing infertility in cattle (see Chapter 34). Where

The dominance of the large preovulatory follicle is caused by infection it can be divided into non-specific, suppressed by heat stress, and the steroidogenic capacity of theca and granulosa cells is compromised. Progesterone secretion by luteal cells is lowered during the summer in hot climates, and in cows subjected to

There are also a number of infections that are difficult to classify such as bovine herpes virus-1 (BHV-1), Ureaplasma spp. and Haemophilus somnus.

also been shown to impair oocyte quality and embryo

Non-specific metritis is the result of either massive development, and increase embryo mortality. In addition to the immediate effects of heat stress, delayed infection or of the infective organisms taking advantage of a deficient uterine defence mechanism, usually effects have also been detected. These include altered

caused by damage at and after calving. Non-specific follicular dynamics, suppressed production of follicular infection can be facilitated by the synergistic action of steroids and lower quality of oocytes and developing different organisms, for example A. pyogenes and embryos. This may explain why poor fertility may Fusobacterium necrophorum.

persist for some time after periods of heat stress Specific infections colonize the undamaged uterus. (Wolfenson et al., 2000).

Two important specific infective agents are C. fetus and Poor pregnancy rate is a problem when European

T. fetus. Campylobacteriosis is spread venereally and cattle are introduced into hot countries where they are causes a mild endometritis in infected females that have exposed to ambient temperatures above 30°C; it is quite not had previous experience of the condition. It has likely that increased embryonic death is part of the been shown in slaughter experiments that in infected reason. There is little doubt that genetic factors are animals fertilization rate is normal and that the infer-

involved in this as in other aspects of heat tolerance.

tility is due in the main to embryonic death within three

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weeks of conception. Loss of the embryo is likely to be

Maternal endocrine environment: Mann and Lamming

due to interference with the uterine environment.

(1995) showed that low plasma concentrations of prog-

Venereal campylobacter infection and its control in the

esterone resulted in the development of a stronger

UK has been reviewed by Taylor (2002). Trichomoniasis,

luteolytic signal. This was taken as an explanation for

sis, a parasitic venereal disease that still occurs in some

the fact that cows with lower plasma concentrations

*countries (Clark et al., 1986), is clinically similar to of progesterone
postinsemination are more prone to*

campylobacteriosis with one major difference, the

embryo loss than those with higher progesterone levels.

occurrence of pyometra in a number of cases (Bon-

Interferon tau (IFN-t) is a protein produced by the

Durant, 1997).

embryo that acts locally within the uterus to block lute-

*Another infectious agent that is introduced from
olysis and maintain the corpus luteum; it prevents
the vagina into the uterus at insemination, but not
PGF2a secretion by inhibiting the development of oxy-
at natural service, is Ureaplasma (Doig et al., 1979), toxin receptors in the
endometrium (Robinson et al.,
which causes a purulent metritis and infertility.*

1999). More recently, further work has shown that

*Haemophilus/ Histophilus also causes vaginitis and successful maternal
recognition of pregnancy in cows*

reduced fertility. For detailed discussion of a wide range

depends on the presence of a sufficiently well devel-

of infectious agents see Morrow (1986). For more

oped embryo producing sufficient quantities of IFN- γ ,

details on both endometritis and metritis see also

which is, in turn, dependent on an appropriate pattern

Chapter 34.

of maternal progesterone secretion (Mann, 1997; Mann

Infectious conditions can cause infertility in at least

& Lamming, 2001). Thus the maternal endocrine envi-

four ways:

ronment, and particularly maternal progesterone levels

within the first one to two weeks after insemination,

- *The febrile reaction raises the temperature of the uterus. Bluetongue (Chapter 43a) is an example of whether an embryo signals its presence to the dam and a disease that causes a high temperature resulting survives or is lost as the cow returns to oestrus.*

in loss of the embryo at about the time of hatching from the zona pellucida, about day 10–12 after

Aged gametes: Fresh chilled semen ages after several service.

days and the inseminated cows have a lower pregnancy

- *The organism infects the uterus and causes metritis, which presumably interferes with embryo nutrition and increased loss of embryos. There is no evidence of adverse effects from ageing of frozen semen stored BHV-1 virus (see p. 289) and Chlamydiales infections. It is likely that, in general, mild endometritis*

ovulation of persistent dominant follicles (Mihm et al., causes embryonic death whereas in cases of puru-1994; Austin et al., 1999) is also likely to result in an lent metritis there may be interference with sper-increased amount of early embryonic death.

matozoa survival and thus fertilization failure.

- Infection of the conceptus can cause its death. The

Local trauma: This cause of loss affects the late embryo thought that embryo transfer could transmit infec-and early fetus. One source of local trauma to the tious diseases from the sire or the dam is worrying.

pregnant uterus is the hand of a person carrying out

In theory, bacterial and fungal infections are less

manual pregnancy diagnosis, or some other palpation

likely than viral infections to be carried by embryos.

of the uterus. In one study the average loss was 2.82 per

From experimental studies with many different

cent of cows diagnosed pregnant (Beghelli et al., 1986).

viruses it appears that if the zona pellucida is intact

Franco et al. (1987) reported a fetal loss rate of 9.5 per and the embryo is washed properly, there is little

cent in cows diagnosed pregnant on days 42–46. The

danger of the transmission of viral infections by

technique, which was carried out on two days, involved

embryo transfer (Singh, 1987; Wrathall, 1995).

palpation of fetal fluid, identification of the amniotic

However, the advent of in vitro technology may

vesicle and slipping of the chorioallantoic membranes.

increase the risk of disease transmission due to dif-

However, with good transrectal ultrasound examina-

ferences in the zona pellucida of in vitro derived

tion technique such losses should now be largely

embryos, enabling easier adsorption of pathogens,

preventable.

and due to the use of biological products for cul-

In a very large field study in which they used milk

ture which may be contaminated with pathogens

progesterone assays, Laitinen et al. (1985) estimated

(Stringfellow & Wrathall, 1995; Guerin, et al., 1997).

that 1.8 per cent of cows that returned to service were

- Endotoxins produced by Gram-negative infections*

pregnant at the time of re-insemination. When a preg-

can increase PGF2a production and cause prema-

nant cow is inseminated, the conceptus must be at risk

ture luteolysis (Fredriksson, 1984).

either through direct trauma or by the introduction of

infection into a progesterone-dominated uterus. An
To complicate matters further, additive effects of two
experienced inseminator may be able to feel the differ-
or more types will produce low pregnancy rates. Two or
ence in the cervix and uterine horns and avoid insemi-
more independent minor adverse factors that occur at
nation into the body of the uterus, electing instead to
the same time will produce a poor result. For example,
deposit the semen in the anterior cervix or not to insem-
Boyd and Reed (1961b) observed that variations in
inate the cow at all.

three factors (calving-to-first-service interval, age of
cow and age of fresh semen) caused a range of preg-

Genetic factors: Mention has been made of the variable nancy rates from 22 to
70 per cent. As mentioned

breed susceptibility to high environmental temperature
above, synergistic action of two or more adverse aetio-
that causes embryo death.

logical factors, for example *A. pyogenes* and *F.*

In some cattle, in the process of cell division, trans-

necrophorum, can result in a more severe pathological location of parts of certain chromosomes without loss condition than either alone.

of genetic material has taken place, a condition that is

Many of the putative causes of return to service are passed on to future generations. These individuals can far from being proven but they cannot be simply dis- be identified by cytogenetic examination of leukocytes.

missed for that reason. Amongst these possibly adverse

When semen from bulls with a translocation is used for factors are the following:

insemination there is a slight increase in the incidence

- *Short-term change in feeding and environment, of return to service, which is believed to be the result particularly at turnout in the spring and at housing of embryonic death, presumably because of lack or in the autumn. This has been postulated to be due excess of some genetic material due to abnormal division at meiosis.*

uterine tube and uterine environment (pH and urea

It is also probable that many genetically abnormal concentrations) and thus affecting normal early embryos are lost early in development, with the advanced embryonic development.

tage that the dam can return to normal breeding at the

- *High milk yield leading to excessive metabolic earliest opportunity. Early embryo development and stress.*

non-infectious causes of embryo loss are reviewed by

- *Stressful effect of disputes about dominance among Kastelic (1994) and Sreenan et al. (1999).*

cows that are subject to frequent changes of groups in dairy herds.

Stage of failure not known

- *Non-reproductive systemic illness.*

It should be clear from the limited number of factors

- *Deficiencies and imbalances in minerals and vitamins listed as known to cause embryonic death, that in the majority of cases the cause is unknown. Some factors very local effect of feeding practices, local soil*

that result in return to service can affect different deficiencies and many other variables).

stages, for example late insemination can result in fer-

- Other conditions that stress the animal or affect its tilization failure in some cases and embryonic death in dry matter intake such as lameness.

others. There is a very large grey area concerning the

There are various reasons why there is so much aetiology of return to service, particularly where groups uncertainty in a field where numerous workers have of affected animals are concerned. The difficulty con-gathered observations and data for several decades.

cerning the relationship between (possible) aetiological Because expected pregnancy rate is about 50 per cent, factors and return to service is twofold.

the random variation in fertility in groups is great and

Some factors almost certainly cause an increased this necessitates large, properly controlled groups in return to service via an unknown mechanism. An

trials in order to give reliable results. The possibility of example is the aetiological relationship between loss

erroneous conclusions from any single trial is so great in body condition from calving to service and associated that only results that are consistently repeated in different populations can be regarded as reliable.

quate nutrition may have detrimental effects on reproduction.

In recent years there have been a number of epidemiological studies of a highly statistical nature on the

• The hypothalamus/pituitary gland to impair

relationships between various aspects of management gonadotrophin release.

and production, disease and reproduction. Because of

• The ovaries, possibly resulting in altered follicular

the complexity of interrelationships very large numbers

growth patterns and reduced quality of oocytes and

of observations are needed and these reports are based subsequent reduced embryo survival.

on data from populations that range from about 2000

• Inadequate uterine environment resulting in

to >70 000 lactation records. For the practitioner this

impaired embryo survival (Webb et al., 1997).

means that evaluation of fertility in small herds is

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difficult due to the large impact of small numbers of

tion, weight loss and fertility; Loeffler et al. (1999): time observations on overall herd results.

of disease, milk yield and body condition; Dohoo et al.

While there are considerable differences between the

(1984): disease, production and culling; Rowlands et al.

findings certain concepts have emerged:

(1986) and Peeler et al. (1994): interrelationship of

diseases; Saloniemi et al. (1986) and Fourichon et al.

- *Reproductive diseases at and after calving tend to*

(2000): reproductive diseases.

occur as an interrelated complex (see Chapter 34).

- *Some diseases that are not specific infections of*

the reproductive tract have an adverse effect on

Diagnosis

reproduction, e.g. Leptospira hardjo, BVD virus

(McGowan & Kirkland, 1995) and BHV-1.

Although a herd consists of many individuals, diagno-

- *There is disagreement on the effect of high yield on the possibility of a return-to-service problem that affects a large proportion of fertility.*

part of a herd requires a different approach from that

- *There is disagreement on the effect of hereditary factors taken to investigate the cause in an individual animal.*

For this reason the individual will be dealt with first, followed by the herd fertility problem.

Although the relative importance of the factors listed above, along with other management factors, is still poorly understood, what does seem to be beyond doubt

Diagnosis of the individual repeat breeder cow now is that fertility, at least in high producing dairy cows in the UK, is declining over time (Barrett, 2001).

One problem with diagnosis is that by the time the cow is recognized as being a repeat breeder the situation is complex one that is still not fully understood. The relationship between nutrition and fertility is a situation that prevailed at the time of reproductive failure may be different. The reader may find the following reviews of interest: Britt

well have changed.

(1995); Webb et al. (1997); Wathes et al. (1998); Webb et al. The clinician should take a history and carry out a

al. (1999); O'Callaghan and Boland (1999); Garnswor-

systematic clinical examination to find out whether any

thy and Webb (2000) and O'Callaghan et al. (2000).

of the factors that are likely to cause return to service

As an example of the complex interactions affecting

are present. The fullness and, therefore, costs of the

fertility, data from the very large study by Saloniemi et

investigation will need to be discussed with the farmer

al. (1986) showed the consequential effects of specific in advance and a diagnosis may only be pursued in value-causes described in Table 36.5.

able animals.

For details of other complex interactions, the reader

A systematic approach to diagnosis is essential and

is referred to some original texts: Andersson et al.

the following procedure is suggested (Box 36.8).

(1991) and Gustafsson and Emanuelson (1996): hyper-

ketonaemia and fertility; Bartlett et al. (1986b) and

History: The history should cover various points as

Garverick (1997): cystic ovarian disease; Bartlett et al.

shown in Box 36.9.

(1986a) and Sheldon (1999): metritis/endometritis;

*Curtis et al. (1985): metabolic disease, mastitis; Dohoo General examination:
The clinician should carry out*

and Martin (1984a): age, season and sire; Dohoo &

a brief general examination looking at the following

*Martin (1984b): mastitis and ketosis; Britt (1995): nutri-
points:*

Table 36.5

*The consequential effects of management and disease on problems of fertility
(reproduced from Saloniemi et al. , 1986*

with permission of the publisher).

Cause

Consequential effect

Retained fetal membranes

Metritis

Anoestrus/suboestrus

Ovarian dysfunction

Winter calving

No

Yes

Yes

Yes

Highest herd milk yield

Yes

Yes

Yes

Yes

Parturient paresis

Yes

Yes

Mastitis

Yes

Yes

Ketosis

Yes

Yes

Yes

Retained fetal membranes

Yes

Metritis

Yes

Yes

Box 36.8

The systematic diagnosis of return to service.

are discussed more fully in Chapter 35 (see p. 536). The use of milk progesterone assays may also improve diag-

- *History*

nostic accuracy (see p. 548).

- *Analysis of records*

Accuracy of diagnosis of cystic ovarian disease by

- *General clinical examination and body condition*

rectal examination: *When undertaking rectal palpation*

- *Examination of the reproductive tract (rectal, vaginal and suitable aids including ultrasound examination)*

there are obvious difficulties in trying to assess the nature of a cyst without rupturing it. A large, soft corpus

- *Appropriate laboratory tests*

luteum with no palpable ovulation papillum can be mistaken for a cyst. Some authors are very sceptical about the accuracy of diagnosis of cystic structures by rectal

Box 36.9

The main factors to be covered in history taking.

palpation alone, for example Stolla et al. (1980) and Guenzler and Schallenberger (1981). However, accu-

- Date of calving.

racy is immensely improved with transrectal ultrasound

- Dystokia and puerperal diseases

examination (Douthwaite & Dobson, 2000), and once

- Postpartum reproductive disease

again milk progesterone assays have their place in con-

- Other diseases (e.g. lameness, metabolic diseases)

firming whether functional luteal tissue is or is not

- Service details

Dates

present (Chapter 35).

Bull used

Accuracy of diagnosis of endometritis: Clinical diag-

Natural or AI

nosis of endometritis is discussed in some detail in

- Inseminator service or DIY AI

Chapter 34 (see p. 521).

- Major management or environmental changes

- Is this considered to be a herd or individual cow problem?

Vaginal examination: There is a choice of two methods of vaginal examination on the farm: manual or visual using a vaginal speculum. In both cases the perineal region and vulva are thoroughly cleaned and the vet-

- Body condition and conformation;*

erinarian uses fresh plastic gloves. With manual exami-

- Signs of non-reproductive disease;*

nation mucus in the anterior vagina is gathered and on

- Signs of vaginal discharge or dried pus or mucus on withdrawal of the hand examined visually and olfacto-
the tail or hindquarters; and*

rily. Minimum lubrication is used to avoid confusion

- Other signs, for example a raised tailhead.*

between clear mucus and the lubricant. The cervix and vaginal wall are palpated for lesions and abnormalities.

Rectal examination: The technique of rectal palpation

The vaginal speculum should be introduced with care

of the genital tract is fully described in various text-

and, when fully inserted, will give a clear view of the

books, as are the characteristics of the various normal

cervix. With both techniques the clinician may have

and abnormal structures in the ovaries and tract. The difficulty in passing the vulvo–vaginal junction, which is purpose of carrying out a rectal examination is to the narrowest part of the tract at this level. When using assess:

a speculum great care should be taken to ensure that the speculum does not transfer infection between cows.

- *The state of the ovaries;*

This is best achieved by using disposable, single-use

- *The condition of the bursae and uterine tubes;*

specula or autoclaving equipment between cows. If a

- *Uterine abnormality or status (e.g. metritis, adhesions, pregnancy);*

single speculum is to be used on a number of cows at a

- *Internal slackness of the pelvic ligaments;*

cows. Box 36.10 shows the factors that should be

- *Absence of fat inside the pelvis (body condition);*
- checked in a vaginal examination.*

- *Vulval discharge (stimulated by palpation);*

- *Evidence of other conditions detectable on rectal*

Selection of samples for laboratory examination: Selec-examination (e.g. cystitis,

pyelonephritis,

fat

tion of the appropriate laboratory examinations and the necrosis).

frequency of sampling depend very much on circum-

Nowadays it is general practice to supplement, or stances, including the value of the cow. The items listed even replace, the manual rectal examination with a in Box 36.11 may be considered.

transrectal ultrasound examination. This has the same

To extend the physical examination beyond what is aims as listed above, but will on the whole give more possible by palpation and transrectal ultrasound, the accurate results. The use of transrectal ultrasound patency of the uterine tubes can be investigated using examination as a diagnostic tool and the comparative the phenosulphonphthalein (PSP) dye test and/or starch levels of accuracy of rectal examination and ultrasound grain test as described by Kessy and Noakes (1979).

Box 36.10

Factors to be checked by vaginal examination.

Box 36.12.

Steps required to diagnose a herd fertility problem.

- *Anatomical abnormalities.*

Heifers

- *History*
- *Damage*
- *Analysis of records*

Caused by dystokia:

Breeding

Rectovaginal fistula

Health

Pneumovagina/urovagina (see p. 519)

Production

Damage to the cervix

- *Selected clinical examinations and sampling*

Serving injuries caused by the bull

- *Conclusion*

Sadistic human interference

Other acquired conditions

Vaginitis (see p. 519)

Type and quantity of mucus

- *Quick, cheap and, in some cases, automated*

Purulent

hormone analysis;

Bloody

- *Non-invasive (or acceptably invasive) examination*

Smelly

of internal organs: hysteroscopy, endoscopy (trans-

Clear oestrous mucus

rectal ultrasound is already routinely used during

on farm fertility examinations);

- *Automatic or semiautomatic recording of physical, behavioural and biochemical changes that are*

Box 36.11

Types of samples required for various laboratory related to the animal's reproductive status, such as tests.

body temperature, restlessness, changes in milk

- *Single milk progesterone assay: an adjunct to rectal palpation and ultrasound examination*
- *Computer analysis of data: compilation over time*
- *Serial milk progesterone assays: monitor events around of various measurements in the individual and service, confirm diagnosis, monitor treatment analysis of herd records (see Chapter 41b).*
- *Oestrone sulphate from milk, blood (specifically from the placenta: indicates live calf)*
- *Heparinized blood for cytogenetic analysis*

Diagnosis of a herd problem

- *Clotted blood for serology (e.g. BHV-1, BVDV, Leptospira It is necessary to approach the diagnosis of a herd fer-hardjo, Neospora caninum) tility problem systematically. It is not practical to lay out*
 - *Purulent material from the uterus: aerobic and anaerobic culture, antibiotic sensitivity*
- in any detail a series of steps to be followed because as the investigation develops the information obtained*
- *Uterine biopsy for histology*
 - *Samples from the cervix and uterus for serology and*

guides the continued course of the investigation.

cytology

The four main causes of herd problems are poor fer-

- Vaginal mucus for campylobacter diagnosis using the
tility management, an infertile 'male', nutritional errors
vaginal mucus agglutination test (VMAT)*

*and deficiencies and infectious conditions. In general
terms the steps required are shown in Box 36.12.*

*History: The object of history taking is to acquire infor-New technological aids
to diagnosis: In recent years*

mation on the factors shown in Box 36.13.

*advances in biochemistry, electronics and fibre optics,
along with skills and materials developed for embryo*

*Analysis of the records: It is useful, if possible, to transfer, have opened up new
diagnostic possibilities.*

examine the breeding records before visiting the herd.

There is every reason to believe that new equipment

*If there are no records then establish a recording
and concepts will continue to be produced, which pres-
system. The objectives of record analysis in connection
ents problems as well as opportunities for the veteri-
with a herd fertility problem are the following:*

narian. Most of the new techniques do not require specifically veterinary skills and so may be used by a

(1)

To assess the current level of fertility in the herd wide range of operators, although for best effect knowl- and to establish whether a problem exists, and if edgeable interpretation is needed. Choices have to be so to determine the seriousness of the situation.

made about the lasting value of each new step forward

(2)

To look for clues to aetiology, by looking at the as investing capital and training in inappropriate tech- fertility of subgroups within the herd:

nology is unproductive.

-

Bull, semen or inseminator;

The relatively new aids to diagnosis can be classified

-

Age (lactation) group;

as:

-

Yield group;

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Box 36.13

History taking in determining herd fertility

cases there may be no comparative data on the bull's problems.

fertility; the problem could be due to poor insemination technique or to improper semen handling.

- *General aspects of the herd and farm*

Number of breeding stock (male and female)

Stocking density

Examination of the animals: It is very useful to examine Type of housing as many cows as possible to obtain a current picture of

Details of grazing

the herd, to observe the condition of the cows and to

Targets for reproduction and production

detect or confirm the incidence of obvious reproductive

Other enterprises

abnormalities. It also reveals the (in)accuracy of the

Information about the stockworkers

information supplied by the farmer or farm staff.

- *Definition of the perceived problem*

Advice should be given about culling of individuals that

Anoestrus

have a very poor prognosis.

Return to service

By this stage the clinician should have a good idea of

Vaginal discharges

the type of problem; how the investigation then devel-

Other manifestations

ops is a matter of clinical judgement which cannot be

- *Duration of problem*

detailed further.

- *Proportion of herd affected*

- *Reproductive management*

Calving management

Service management: cows and heifers (heat detec-

Treatment (see Chapter 42)

tion, timing of AI, technician or DIY AI)

Bulls

The veterinarian is faced with some difficulties when

- *Nutrition*

Ration formulation

having to decide on appropriate and effective treat-

Feeding system

ments for cows that are presented as infertile. This is

Assessment of adequacy of nutrition e.g. yield, milk

partly because of the complex aetiology of return to

composition, condition score, metabolic profiles

service, but also because of the time that has passed

- *Details of 'herd health plan'*

since the factors that initiated the problem were

Disease monitoring

present. When using pharmaceutical products the

Intervention levels

reader is advised to consult the manufacturers' data

Preventive medicine initiatives

sheets for details of treatment, withdrawal periods,

Vaccination protocols

dangers and contraindications. Wherever possible the

Biosecurity

treatment should be followed up to see whether it has

- *Disease status of herd*

*Details of herd status for infectious diseases known to
been successful or not and, if need be, repeated or
affect fertility, e.g. BVDV, BHV-1, leptospirosis
changed.*

*Incidence of conditions that have a direct effect on fer-
tility, e.g. retained fetal membranes, endometritis*

Other diseases and conditions in the herd, e.g.

Ovulation failure

lameness

*The most important cause of ovulation failure is cystic
ovarian disease. Treatment is based on whether the cyst
has been diagnosed as follicular or luteal. In most cases
the condition is probably caused by hypothalamic–*

•

Seasonal effect;

pituitary dysfunction and it is, therefore, logical to treat

•

Familial effect;

the condition systemically. There is little or no benefit

•

Relevant infectious disease: e.g. non-specific

in rupturing the cyst manually, which also involves a risk

endometritis after calving, Campylobacter

of causing haemorrhage and ovarian adhesions. Nor

fetus, Leptospira hardjo, Neospora caninum, does there appear to be any benefit from administra-BHV-1, BVDV, etc.

tion of drugs directly into the cyst.

Analysis and interpretation of herd fertility records

Many cystic structures found less than 42 days after

are discussed in much more detail in Chapter 41a,b.

calving are transient and benign, and in general these

Once a cause of the infertility is suspected, a valuable

do not require to be treated. Only if abnormal behav-

technique is to group the records of all animals that are

your indicates that they are pathological or if there

influenced by the suspect factor and compare these with

is some pressing management need should they be

the records from all other cows. It can be difficult to

treated.

identify the cause of a problem where farmer insemin-

For follicular cysts the three therapeutics of choice

nation is associated with poor pregnancy rate. In these

are human chorionic gonadotrophin (hCG),

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gonadotrophin-releasing hormone (GnRH) or GnRH

up to three days before, can improve predictability of

analogues, such as buserelin, and progesterone-

the oestrous response. If fixed time insemination is used

releasing intravaginal devices (PRIDs) and controlled

and oestrus is observed later the animal should be

internal drug release (CIDR) (Dobson et al., 2001a).

inseminated again.

They all work in different ways to achieve the same end,

i.e. to put the animal under the influence of proges-

Fertilization failure

terone. One effect of this is to allow a build-up of LH in

the pituitary so that enough endogenous LH is available

Delayed ovulation: For many years one of the standard

after removal of the progesterone devices or regression

ways of treating apparently normal repeat breeders on

of the (induced) corpus luteum. This leads to normal

the farm has been to administer hCG or GnRH on the

preovulatory follicle development and ovulation, and in

day of service. The theory behind this was that many all cases the cow should be served at the first heat after of these cases were thought to be due to lack of an treatment. The duration of exposure to exogenous or adequate gonadotrophin surge delaying ovulation, endogenous progesterone is thought to be important in although controlled trials of this type of treatment are the non-recurrence of the cystic condition, with 14 days lacking and a better approach may be to inseminate the being preferable to seven (Nanda et al., 1989). There is cow again 24 hours after the first insemination.

also evidence to suggest that the success of treatment of follicular cysts depends on whether the cyst is producing

Interference with transport of egg or spermatozoa:

oestradiol or not, as reflected by the presence or absence

When this is due to bilateral blockage of the uterine

of other follicles >5 mm in diameter. Following treat-

tubes (or other parts of the female tract), there is no

ment with an intravaginal progesterone releasing device

simple treatment. In some cases the diagnostic dye test

or GnRH, animals had a pregnancy rate to all insemina-

may remove a minor blockage and attempts have been made to achieve this by adding antibiotics and corticosteroids to the dye, which is introduced into the uterine tube end of the uterus under gentle pressure (Tebble et al., 2001).

using a cuffed catheter.

Human chorionic gonadotrophin has an LH-like

In valuable animals the use of ovum pick-up techniques using transvaginal ultrasound-guided oocyte aspiration can be used to harvest oocytes for in vitro production of embryos. One or more of these embryos could then be implanted back into the uterus of the donor, although it is probably more advisable to

oestrus can be expected about 8–22 days after treatment. Implant valuable embryos derived in this way into other cows. It is desirable to examine the treated cows about 7 days after treatment for the presence of luteal tissue. If luteal tissue is found, treatment with PGF2a or donors, but which the farmer simply wishes to get back an analogue is a suitable way of increasing the pre-in calf so as to induce another lactation, the implanting dictability of the following oestrus. Service should be at of a low cost embryo may be used as a treatment. This that oestrus and aids to oestrous detection should be has now become commercially feasible and technically used. If there is no satisfactory response to the first achievable for repeat breeders with no uterine or treatment, the cow should be re-examined and treated endocrine abnormalities; single step (in straw) embryo again.

thawing, using embryos frozen in ethylene glycol, is

For cysts containing functional luteal tissue as judged

used, which allows embryo transfer with little more by ultrasound examination or milk progesterone assay, equipment and skill than that required for AI (May, injection of PGF2a or an analogue followed by insemination 1996).

nation at observed oestrus is the most suitable

Other factors that can cause failure of fertilization treatment.

are:

An alternative treatment, suitable for both types of cystic ovarian disease, is the insertion of a PRID/CIDR,

- *Service at the wrong time;*

which can be left in position for 12 days, and after with-

- *Poor-quality semen;*

drawal the cow should be served, either at a fixed time

- *Infertile bull;*

or preferably at observed oestrus (Douthwaite &

- *Problems with artificial insemination.*

Dobson, 2000). If the type of cyst cannot be diagnosed accurately, then administration of PGF2a or an ana-

These are dealt with elsewhere under Prevention, in

logue, either on the day of PRID/CIDR withdrawal or

Chapter 38 on the bull, or by common sense.

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Embryonic death

further 30 days later. Taylor (2002) describes in some detail the technique for collecting samples by preputial

Although the aetiology of early embryonic death is washing.

listed under nine headings (Box 36.7) only non-specific

Although there is no natural immune response in the endometritis (already dealt with in Chapter 34), specific

bull, one successful form of treatment depends on vac-

metritis caused by C. fetus (see p. 582) and a variety of cination (Clark & Dufty, 1982). The vaccine is licensed

other infections and possibly maternal endocrine envi-

for use in a number of countries but not in the UK,

ronment are suitable for conventional treatment. All

although one can be made under licence in the UK for

the other conditions should be corrected by manage-specific herds.

ment improvements or are dealt with in different chap-

*Treatment of cows with local or systemic antibiotics
ters of this book.*

is unreliable and because cows develop resistance to the

*A number of different Campylobacter spp. are asso-
infection it is usually best to wait for this to develop.*

ciated with infertility and abortion in cattle, the specia-

*Whatever course is followed an infected cow should be
tion and subspeciation of which is complex and*

*regarded as potentially infective for at least two gesta-
currently under review using modern molecular tech-*

tions after initial infection. Even after this time a few

niques. However, it is clear that the most common iso-

cows may remain infected. As it takes only one cow to

lates associated with fertility problems in the UK are C.

infect a bull when natural service is practised, it is clear

*fetus subsp. venerealis and C. fetus subsp. fetus (Newell that it is hazardous to
allow natural service of any cows*

*et al., 2000). Infection with either of these subspecies that have ever been
infected. Vaccination of cows*

should be thought of as a herd problem. In the case of

(where permitted) is widely and fairly effectively prac-

C. fetus subsp. venerealis the natural habitat of the tised in range conditions.

organism is the bovine reproductive tract and it does

On a herd basis, when campylobacteriosis is diagnosed the best advice is that natural service should stop only as a venereal disease (Taylor, 2002; see also p. 582). and all services should be by AI using semen from non-

In the bull, in which infection with C. fetus subsp.

infected bulls. Recently infected cows will continue to

venerealis is limited to the surface of the prepuce and return to service for some time. In suckler herds where

penis, spontaneous cure does occur but is erratic and

the use of AI to observed natural oestrus may be prob-

unreliable. Moreover, if the bull continues natural

lematic this approach can be facilitated by the use of

service of infected cows he will be re-infected as the

repeated oestrous synchronization and fixed-time arti-

superficial nature of the infection does not stimulate an

ficial insemination as described by Penny et al. (2000).

immune response in the bull. The organism colonizes

There is a management alternative, which is

the crypts of the preputial epithelium which increase in

extremely difficult to carry out successfully over a long both size and number as the bull ages. This means that period and should be advocated only under special the prepuce of the older bull provides favourable circumstances. This is to segregate the herd and use the conditions for the persistence of the organism, giving a infected bull(s) on infected cows and AI or non-higher incidence of carrier bulls among those over 5 infected bulls on non-infected cows and heifers. How-years of age. Diagnosis and treatment are also more ever, this depends on the accurate identification of the difficult in the older bull (Taylor, 2002).

infection status of all animals.

The usual treatment of the bull involves preputial Manipulation of the maternal endocrine environment lavage with streptomycin in a viscous oily medium and ment post insemination by the exogenous administration of progesterone or progestagens has been tried as (2002) recommends daily systemic (e.g. intramuscular) a treatment of repeat breeder cows or where herd con-

treatment with a combination of streptomycin and ception rates are particularly poor. However, the results dihydrostreptomycin, each at 150 mg per ml as sul- of such studies are variable and seem to be affected by phates at a dose rate of 10 mg per kg body weight. the time of progesterone administration and the initial Taylor (2002) gives a detailed description of the tech- fertility levels. A meta-analysis of 17 progesterone sup- nique of preputial lavage. It is important that both sys- plementation studies showed that treatment during the temic and local treatments are carried out thoroughly first week of pregnancy resulted in an increase in preg- and are repeated daily for three consecutive days. This nancy rate, especially on farms with poor fertility, while needs to be combined with further sampling for the treatment during the second and third weeks of preg- organism not less than 30 days after treatment has fin- nancy gave no overall significant increase (Mann & ished to confirm success of treatment. In a confirmed Lamming, 1999). At the present time treatments of this outbreak of campylobacteriosis, the bulls should be

type cannot be recommended as a means of improving sampled twice, not less than three days and not more conception rates, and more research is needed to identify suitable hormonal treatment strategies.

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An alternative post-insemination treatment involving the administration of GnRH (or GnRH analogues are rarely comparable because different criteria for such as buserelin) to the cow around 11 days after selection of cases and assessment of success may have insemination has become popular in recent years. In some studies this has been shown to give very good and because controlled studies are rare, interpretation of results is problematic. Accordingly it is suggested achieved an improved pregnancy rate to first service of that, as well as reading published reports, clinicians approximately 10 per cent. A recent meta-analysis of

should analyse their own practice records of diagnosis, studies of the effect of GnRH 11–14 days after insemination treatment and outcome.

nation concluded that while results were not consistent

The most common situation is the cow presented

across all studies, there was a significant improvement

after a few unsuccessful inseminations (or services),

in pregnancy rate amongst 2541 cows across six studies

which on examination appears to be normal. These

(Peters et al., 2000). However, post insemination hor-

apparently normal repeat breeders have an expected

monal treatments remain an unpredictable means of

pregnancy rate at the first service after examination that

improving pregnancy rate on any one given farm, and

is about the same as the first service pregnancy rate in

in most instances resources would probably be better

the herd, as indicated in Table 36.6.

targeted at improving the management of the herd

This is not to say that the factors influencing fertility

rather than instigating such treatment regimens.

in first service and repeat breeder cows are the same.

In the first service cows, fertility may be affected by closeness to calving and the stress of peak yield. The Stage of failure unknown: non-specific endometritis population returning to service will have overcome

The two most common therapeutic approaches are (i) these problems, but will include a higher proportion of promotion of the cow's normal resistance to infection cows that will never become pregnant. Unfortunately by inducing oestrus and (ii) the use of antibiotics. There before their ultimate disposal, cows in the last group is a tendency for spontaneous elimination of the infection will have had two or three different treatments and tious agents from the uterus, most likely due to the cow several examinations at regular visits. In most cases going through successive periods of oestrus. The current there is no reliable way of identifying these problem practical approach to treatment is discussed in detail in cows.

Chapter 34 (see p. 521).

Where economics allow the hospitalization and intensive investigation and therapy of repeat breeder

cows in referral centres (now very uncommon in the

Prognosis

UK), the owner should be given a definite prognosis

as soon as possible to minimize the cost involved in

From the client's point of view successful treatment

keeping and treating cows over several weeks. Much of

means that the cow becomes pregnant; this is a two-

this cost is due to the fact that once an animal has been

stage process. Firstly, the cow has to return to normal

served there is little positive that can be done except to

reproductive function and secondly she has to conceive

monitor changes until pregnancy or non-pregnancy is

and remain pregnant. While the first stage is mainly the

confirmed. With modern diagnostic methods such as

responsibility of the veterinarian, the second stage

ultrasound scanning it is now possible to reduce the

often depends on the farmer and farm staff. Where poor

waiting time from service to diagnosis of non-

husbandry (i.e. feeding, housing or breeding manage-

pregnancy to a minimum of 25–30 days, and milk pro-

ment) is thought to be causing return to service, prog-

*gesterone analysis can be used to give an indication of
nosis must be guarded because it is often a difficult
pregnancy status as early as 19–20 days post service.
and slow task for the veterinarian to effect a marked
Other methods of early pregnancy diagnosis such as
improvement.*

assays for bovine pregnancy-associated glycoprotein

Table 36.6

Response of apparently normal repeat breeders served without treatment.

Number of cows

Treatment

Percentage pregnant at first

Reference

AI after examination

191

None

59.7

de Kruif (1975)

141

None

60.0

Refsdal (1979)

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(bPAG) may also be used to speed up pregnancy diag-

In general, three factors affect the outcome:

nosis (Skinner et al., 1996). If the prognosis is

- The time that has elapsed since calving. Cases of favourable, costs are reduced if the cow is returned to cystic ovaries that occur up to six weeks after the owner's farm where the local veterinarian can treat calving have a good chance of spontaneous recovery.*

ery. Cases that are first seen six months after calving

Accurate prognosis with long-term repeat breeder

have a very poor prognosis.

cows is also difficult. The prognosis for natural concep-

- The accuracy of diagnosis. If the type of cyst is tion must always be regarded as poor, but particularly misdiagnosed, selection of inappropriate treatment so in cases with blocked uterine tubes, persisting may give poor results.*

endometritis and anomalous steroid hormone produc-

- *The aetiology of the condition affects the outcome.*

tion from the ovaries. For example, of 33 chronic repeat

If the cyst is caused by a temporary environmental

breeders treated by Boyd et al. (1984) only eight

influence such as poor nutrition, then the prognosis

became pregnant. However, the use of embryo transfer

is good even without treatment, once this environ-

techniques to maintain animals for subsequent lacta-

mental influence is corrected. At the other extreme,

tions may offer some hope and has been shown in some

if it is one of the minority of cases caused by defi-

studies to give reasonable results. May (1996) reports

ciency of LH receptors in preovulatory dominant

pregnancy rates in the region of 50 per cent using-single

follicles, the prognosis may be poor in the short

step (in straw) embryo thawing, with embryos frozen in

term as this may be the consequence of events

ethylene glycol. Furthermore, nowadays it is also pos-

occurring up to months previously.

sible to harvest oocytes from valuable animals for in

vitro production of embryos, and thus offspring, using The results presented in

Table 36.7, in most of which transvaginal ultrasound-guided oocyte aspiration of diagnosis and outcome were checked by progesterone ovarian follicles.

assays, are typical of earlier published field trials.

The two most commonly diagnosed specific patho-

However, these data do not illustrate the ultimate logical causes of return to service are cystic ovarian benefit of treatment, which should be measured in disease and purulent endometritis.

terms of calving or treatment to conception intervals and culling rates (see Table 36.8).

Guenzler and Schallenberger (1981) treated 66

Cystic ovarian disease (see p. 526)

cows in which luteal cysts had been diagnosed by

In data collected from a number of veterinary practices

rectal palpation. After treatment with an analogue of

Bartlett et al. (1986b) recorded that culling rate for PGF_{2a}, complete luteolysis occurred in the 35 cows

cows that had cystic ovarian disease was 26.6 per cent,

with mid-cycle levels of progesterone and all but three

compared with 21.6 per cent for other cows. Cows with of these started normal cycles. The first service cystic ovarian disease that conceived had an interval pregnancy rate for these cows was 40 per cent. The two from calving to conception 33.5 days longer than cows groups of cows with lower levels of progesterone without cystic ovarian disease. More recently cystic cycled erratically after treatment and had first service ovaries were associated with 6 to 11 more days to first pregnancy rates of 20 and 24 per cent. Nanda et al. service and with 20 to 30 more days to conception in a (1988) treated 77 luteal cyst cases with an analogue of meta-analysis of papers published between January PGF2a and 65 per cent of these exhibited initial recovery, i.e. the cyst regressed and a corpus luteum formed;

Table 36.7

The treatment of follicular cystic ovarian disease with GnRH.

Number of

Type of

Confirmed by

Treatment

Successful result:

Reference

cows

cyst

progesterone

progesterone rose

assay

within 14 days

104

Follicular

Yes

GnRH

73 (70%)

Nakao et al. (1983)

30

Follicular

Yes

GnRH

25 (83%)

Ax et al. (1986)

12

Follicular

Yes

None

3 (25%)

Ax et al. (1986)

116

Follicular

No

GnRH

61 (53%)

Nanda et al. (1988)

55

Follicular

Yes

GnRH

40 (73%)

Booth (1988)

44

Follicular

Yes

hCG +

32 (73%)

Booth (1988)

progesterone

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Table 36.8

Fertility parameters of cows treated for follicular and luteal ovarian cysts (mean \pm SE).

Treatment

Follicular

Matched

Treatment

Luteal cysts

Matched

(n cows)

cysts

controls

(n cows)

controls

Calving to

PRID

93 ± 27

90 ± 37

PRID

132 ± 49

93 ± 52

conception (days)^a

(*n* = 22)

(*n* = 14)

Treatment to

PRID

24 ± 19

PRID

60 ± 67

conception (days)^a

(*n* = 22)

(*n* = 14)

Culling rate (%)^a

PRID

41

4

PRID

11

7

(n = 22)

(n = 14)

Calving to

GnRH

82 ± 7

70 ± 6

PGF2a

141 ± 15

68 ± 9

conception (days)b

(n = 16)

(n = 22)

Treatment to

GnRH

48 ± 9

PGF2a

20 ± 5

conception (days)b

(n = 16)

(n = 22)

Culling rate (%)^b

GnRH

44

PGF_{2a}

9

(n = 16)

(n = 22)

Calving to

PRID

125 ± 17

70 ± 6

conception (days)^b

(n = 21)

Treatment to

PRID

33 ± 8

conception (days)^b

(n = 21)

Culling rate (%)^b

PRID

($n = 21$)

a From Douthwaite and Dobson (2000).

b From Tebble et al. (2001).

*not all became pregnant and in 18 per cent the cyst
than in normal cows. Time from treatment to concep-
recurred.*

tion was about 75 days.

More recent results from Douthwaite and Dobson

Factors that affect prognosis have been quantified by

*(2000) and Tebble et al. (2001) are summarized in Table Anderson (1985) who
modified a system proposed by*

36.8. In these studies the overall interval from calving

Studer and Morrow (1978), which was based on a

to conception was significantly greater than that of

careful analysis of clinical and laboratory observations.

matched controls irrespective of cyst type or treatment.

Anderson took into account (i) the time since calving,

Cows treated for luteal cysts took significantly longer to

(ii) whether oestrus had occurred since calving, (iii) the

get in calf than those treated for follicular cysts, again

amount of pus, (iv) the size of the cervix and (v) the
irrespective of treatment. Cows treated for follicular
diameter of the larger affected horn. By giving points
cysts had a higher culling rate than those with luteal
for these factors a cumulative score was calculated that
cysts. It would seem that at least in the herds used in
gave a useful prediction about the eventual outcome of
these studies, farmers and veterinarians were prepared
the case. Cows that had been in oestrus had a poor
to persevere with cows diagnosed with luteal cysts, even
prognosis, presumably because they had not responded
at the expense of an extended calving to conception
to the normal defence mechanism.

interval, whereas cows with follicular cysts tended to be
Pepper and Dobson (1987) found that the relative
culled earlier.

amount of pus in the discharge gave a (non-significant)
indication of the pregnancy rate after treatment and
that time from calving to treatment was significantly
Purulent endometritis/metritis (see pp. 519, 521)
related to pregnancy rate after treatment. Cows treated

Ott and Gustafsson (1981) reviewed reports on over 600 cases and showed that in 85 per cent the uterus was emptied within a few days of treatment with PGF2a or a significantly poorer result for cows treated later.

an analogue. In most reports the authors stated that From Anderson's (1985) data it appeared that in successfully treated cows there was a high incidence of

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periparturient problems at the resultant calving and a

(d)

checking the fertility of the bulls;

very high culling rate in this group of animals in the year

(e)

checking the efficiency of the inseminators,

after treatment, observations that warrant further

and the quality of the semen used;

study. The treatment and control of endometritis and

(f)

understanding the causes of fertility variations in a specific herd.

As regards the outlook for herds with a return-to-

(5)

Avoidance of service contact outside the herd and service problem prognosis varies and obviously is ensuring veterinary examination, isolation and related to the diagnosis. Some episodes of herd infertility of bought-in breeding stock, especially bulls will reduce the risk of introducing venereal spontaneously.

diseases and other diseases affecting fertility. (See In cases where a definite infectious cause is identified, such as campylobacteriosis, the outcome can be biosecurity.)

completely satisfactory with the elimination of the

(6)

Good housing environment and cattle handling

infection and the return to a normal herd pregnancy will help to avoid stress, lameness, discomfort and rate. This depends on treatment being carried out care-fear in the herd.

fully and the subsequent control of breeding manage-
(7)

Consideration should be given to reproductive ment being optimized.

dysfunction with a hereditary component, e.g.

Where the problem is related to poor husbandry cystic ovarian disease.

caution should be expressed until clear signs of
(8)

Interventions that may cause embryonic death improved management are noted.

should be avoided, such as drastic changes in feeding and environment, herd medication and the administration of vaccines in the weeks after

Prevention

service. In the case of vaccines the manufacturer's recommendations should always be followed.

Improvement in pregnancy rates is achieved by encouraging good husbandry and avoiding factors that have an adverse effect on fertility. While it is possible to achieve a very high fertilization rate (up to 100 per cent), some doubtful value of this is discussed below.

(9)

Drug therapy, using either antibiotics or hormones, in the post partum phase has been advocated as a way of improving pregnancy rates. The embryonic death is inevitable, which is why it is rare for a herd to achieve a pregnancy rate of greater than 70 per cent.

Where specific drug therapy is applied selectively in cows identified as likely to have poor fertility this is obviously a suitable approach. However, it has been proposed that routine treatments should be applied to these is likely to produce poor pregnancy rates.

all cows, normal and abnormal, and this is much more questionable. For example in one study post partum

(1)

Accurate heat detection.

administration of PGF2a 21 days after calving reduced

(2)

Proper nutrition and maintenance of suitable

the interval to first oestrus and first service by about 10

body condition before and after calving.

days. Pregnancy rates to first service were increased and

(3)

Avoidance of dystokia and post partum abnor-

the number of services to conceptions was reduced

malities and timely veterinary intervention where

from 2.0 to 1.3 (Schofield et al., 1999). However, a

indicated. This should reduce the incidence of

number of trials have given conflicting results. Meta-

delayed involution, non-specific endometritis

analysis of 24 trials where PGF2a was administered to

(Chapter 34), blocked uterine tubes, cystic ovarian

cattle within 40 days of calving reported no beneficial

disease, etc. (see Chapter 34).

effect on first service pregnancy rate, although there

(4)

The proper use of records can help to achieve a small reduction in days open for treated cows, good pregnancy rate and to avoid long intervals between calving (Burton & Lean, 1995). It is now becoming socially unacceptable to administer hormonal products to cattle

(a)

not serving cows too soon after calving (not as 'routine' treatments, and some sectors of the industry before 42 days); try such as organic milk producers and many consumers

(b)

being aware of the expected return-to-service date (for example, by using a three-week calendar); would rather that hormone preparations were not used at all, or that their use were restricted to individual cows with particular problems that cannot be treated by any

(c)

ensuring early pregnancy diagnosis via trans-

other means.

rectal ultrasound and milk progesterone

The routine use of antibiotics in both normal and assays;

abnormal cows after calving or at the time of

Failure to Conceive and Embryonic Loss • 573

insemination should be strongly discouraged; results

Ax, R.L., Bellin, M.E., Scheinder, D.X. & Haase-Hardie, J.A.

have shown no benefit to normal cows. Antibiotic usage

(1986) Reproductive performance of dairy cows with cystic

should be restricted to those animals where there is a

ovaries following administration of Procystin TM1. Journal specific therapeutic indication, and where failure to

of Dairy Science, 69, 542–5.

treat may compromise the animal's welfare.

Ayalon, N. (1978) A review of embryonic mortality in cattle.

Journal of Reproduction and Fertility, 54, 483–93.

In order to overcome problems with oestrous detec-

Bane, A. (1964) Fertility and reproductive disorders in

tion, oestrous synchronization and fixed time AI may

Swedish cattle. British Veterinary Journal, 120, 430–41.

be employed; Lane et al. (2001) have reviewed methods

Barrett, D.C. (2001) Cattle fertility management in the UK.

of synchronization. The technique is best suited for

Cattle Practice, 9, 59–68.

dairy heifers and beef suckler cows. For best results

Bartlett, P.C., Kirk, J.H., Wilke, M.A., Kaneene, L.B. & Mather, the farmer, the veterinary surgeon and a member of

E.C. (1986a) Metritis complex in Michigan Holstein–

the insemination organization should discuss the

Friesian cattle: incidence, descriptive epidemiology and esti-

whole operation well beforehand. The best results are

mated economic impact. Preventive Veterinary Medicine, 4, obtained when only reproductively normal cows at least

235–48.

six weeks after calving (preferably longer) or maiden

Bartlett, P.C., Ngategize, P.K., Kancene, J.B., Kirk, J.H., Anderson, S.M. & Mather, E.C. (1986b) Cystic follicular disease heifers are included, although it is possible to synchro-in Michigan Holstein–Friesian cattle: incidence, descriptive

nize cows that have had a shortened post partum period

epidemiology and estimated economic impact. Preventive

(Penny et al., 2000; Penny & Lowman, 2002). First

Veterinary Medicine, 4, 15–33.

calvers and animals in poor body condition tend to give

Beghelli, V., Boiti, C., Parmigiani, E. & Barbacini, S. (1986) poor results and may need additional hormones such as

Pregnancy diagnosis and embryonic mortality in the cow.

equine chorionic gonadotrophin and nutritional treat-

In Embryonic Mortality in Farm Animals (ed. by L.M.

ments. Results following synchronization of dairy cows

Sreenan & M.G.

Diskin),

pp.

159–67.

Nijhoff,

can be satisfactory (Biggadike & Mawhinney, 1996;

Dordrecht/Boston/Lancaster.

Jobst et al., 2000) and cost-effective (Esslemont & Biggadike, H. & Mawhinney, I. (1996) Planned breeding

Mawhinney, 1996), but currently still need insemina-

routine in dairy cows using a treatment regimen involving

tions at observed oestrus for optimal results. Stress and

GnRH and PGF2a. A multi site study (interim report).

Cattle Practice, 4, 289–91.

any change in management and diet should be avoided

Bon-Durant, R. (1997) Pathogenesis, diagnosis and manage-

during preparation and for three to four weeks after

ment of trichomoniasis in cattle. *Veterinary Clinics of North America*. As far as possible other interventions

America: *Food Animal Practice*, **13**, 345–61.

such as vaccination should be avoided during the same

Booth, J.M. (1988) The milk progesterone test as an aid to the period. Some of the oestrous synchronization regimens

diagnosis of cystic ovaries in dairy cows. *Veterinary Record*, may be used for the repeat synchronization of cows not

123, 437–9.

in calf to the first service (Penny et al., 2000; McDougall, Boyd, H. & Reed, H.C.B. (1961a) Investigations into the

2001; Penny & Lowman, 2002); this allows repeated

incidence and causes of infertility in dairy cattle – Fertility fixed time insemination. However, if repeat synchro-variations. *British Veterinary Journal*, **117**, 18–35.

nization is not to be used it is essential that preparation

Boyd, H. & Reed, H.C.B. (1961b) Investigations into the incidence and causes of infertility in dairy cattle – influence of some management factors affecting the semen and insemination – such as an intensive period of heat detection approximation conditions. *British Veterinary Journal*, **117**, 74–86.

mately 19–24 days after the initial service. If natural

Boyd, H., Renton, J., Munro, C., Harvey, M., Isbister, J. & Kelly, service is to be used on these returns it is essential that

E. (1984) *Clinical studies of a series of long (term) repeat the 'sweeper bull' is not presented with an excessive breeder cows and heifers. Vlaams Diergeneeskundig Tijdschrift, 53, 165–9.*
time (see Chapter 38).

Britt, J.H. (1995) *Relationship between postpartum nutrition, weight loss and fertility. Cattle Practice, 3, 79–83.*

Burton, N.R. & Lean, I.J. (1995) *Investigation by meta-*

References

analysis of the effect of prostaglandin F2a administered post partum on the reproductive performance of dairy

Anderson, D.B. (1985) *A clinical study of chronic endometri- cattle. Veterinary Record, 136, 90–4.*

tis in dairy cows. MVM thesis, Glasgow University.

Clark, B.L. & Dufty, J.H. (1982) *The duration of protection Andersson, L., Gustafsson, A.H. & Emanuelson, U. (1991)*

against infection with Campylobacter fetus subsp. venerealis Effect of hyperketonaemia and feeding on fertility in dairy

in immunised bulls. Australian Veterinary Journal, 58, cows. Theriogenology, 36, 521–36.

Austin, E.J., Mihm, M., Ryan, M.P., Williams, D.H. & Roche, Clark, B.L., Dufty, J.H. & Parsonson, I.M. (1986) The fre-J.F. (1999) Effect of duration of dominance of the ovulatory

quency of infertility and abortion in cows infected with

follicle on onset of estrus and fertility in heifers. *Journal of Trichomonas fetus var. brisbane. Australian Veterinary Animal Science*, **77**, 2219–26.

Journal, **63**, 31–2.

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Claus, R., Karg, H., Zwiauer, D., von Butler, I., Pirchner, F. & Fredriksson, G. (1984) Some reproductive and clinical aspects

Rattenberger, E. (1983) Analysis of factors influencing
of endotoxins in cows with special emphasis on the role
reproductive performance of the dairy cow by progesterone
of prostaglandins. *Acta Veterinaria Scandinavica*, **25**, 365–
assay in milk fat. *British Veterinary Journal*, **139**, 29–37.
77.

Coleman, D.A., Thayne, W.V. & Dailey, R.A. (1985) Factors

Garnsworthy, P.C. & Webb, R. (2000) Nutritional influences affecting
reproductive performance of dairy cows. *Journal*

on fertility in dairy cows. *Cattle Practice*, **8**, 401–405.

of Dairy Science, **68**, 1793–803.

Garverick, H.A. (1997) Ovarian follicular cysts in dairy cows.

Cook, D.L., Smith, C.A., Parfet, J.R., Youngquist, R.S., Brown, *Journal of Dairy Science*, **80**, 995–1004.

E.M. & Garverick, H.A. (1990) Fate and turnover rate of

Grahn, T.C., Fahning, M.L. & Zeinjanis, R. (1984) Nature of ovarian follicular cysts in dairy cattle. *Journal of Reproduc-early reproductive failure caused by bovine viral diarrhea*

tion and Fertility, **90**, 37–46.

virus. *Journal of the American Veterinary Medical Associa-*

Curtis, C.R., Erb, H.N., Sniffen, C.J., Smith, R.D. & Kronfield, *tion*, **185**, 429–32.

D.S. (1985) Path analysis of dry period nutrition, postpar-

Guenzler, O. & Schallenberger, E. (1981) The treatment of

tum metabolic and reproductive disorders, and mastitis in

ovarian cysts in cattle with prostaglandins – possibilities

Holstein cows. *Journal of Dairy Science*, **68**, 2347–60.

and limitations. *Acta Veterinaria Scandinavica*, **77** (Suppl.), Dobson, H., Douthwaite, R., Nobel, K.M., O'Donnell, M.J.,

327–41.

Ribadu, A.Y., Tebble, J.E. & Ward, W.R. (2001a) Cystic

Guerin, B., Nibart, M., Marquant-Le Guienne, B. & Humblot

ovaries in cattle. *Cattle Practice*, **9**, 185–9.

P. (1997) Sanitary risks related to embryo transfer in domes-

Dobson, H. & Nanda, A.S. (1992) Reliability of cyst diagnosis tic species. *Theriogenology*, **47**, 33–42.

and effect of energy status on LH release by oestradiol

Gustafsson, A.H. & Emanuelson, U. (1996) Milk acetone or GnRH in cows with ovarian cysts. *Theriogenology*, **37**, concentrations as an indicator of hyperketonaemia in dairy 465–72.

cows: the critical value revised. *Animal Science*, **63**, 183–8.

Dobson, H., Tebble, J.E., Smith, R.F. & Ward, W.R. (2001b) Hancock, L.L. (1948) The clinical analysis of reproductive

Is stress really all that important? *Theriogenology*, **55**, failure in cattle. *Veterinary Record*, **60**, 513–17.

65–73.

Henricson, B. (1957) Genetical and statistical investigations

Dohoo, I.R. & Martin, S.W. (1984a) Disease, production and into so-called cystic ovaries in cattle. *Acta Agriculturae culling in Holstein–Friesian cows. III. Disease and produc-Scandinavica*, **7**, 3–93.

tion as determinates of disease. *Preventive Veterinary*

Howells, H.M.J., Davies, D.A.R. & Dobson, H. (1999) *Influ-Medicine*, **2**, 671–90.

ence of the number of days spent training in an abattoir with

Dohoo, I.R. & Martin, S.W. (1984b) Disease, production and access to live cows on the efficiency of do-it-yourself artifi-culling in Holstein–Friesian cows. IV. Effects of disease on

cial insemination. *Veterinary Record*, **144**, 310–14.

production. *Preventive Veterinary Medicine*, **2**, 755–70.

Hunter, R.H.F. (1999) Pre-fertilisation events and the fate

Dohoo, I.R., Martin, S.W., McMillan, I. & Kennedy, B.W.

of bovine spermatozoa in the female genital tract. Cattle

(1984) Disease, production and culling in Holstein–Friesian

Practice, 7, 261–6.

cows. II. Age, season and sire effects. Preventive Veterinary Jackson, P.S., Johnson, C.T., Bulman, D.C. & Holdsworth, R.J.

Medicine, 2, 655–70.

(1979) A study of cloprostenol-induced oestrus and sponta-

Doig, P.A., Rulinke, H.L., Mackay, A.L. & Palmer, N.C. (1979) neous oestrus by means of the milk progesterone assay.

Bovine granular vulvitis associated with Ureaplasma infec-British Veterinary Journal, 135, 578–90.

tion. Canadian Veterinary Journal, 20, 89–94.

Jasko, D.J., Erb, H.N., White, M.E. & Smith, R.D. (1984)

Douthwaite, R. & Dobson, H. (2000) Comparison of different Prostaglandin treatment and subsequent cystic ovarian

methods of diagnosis of cystic ovarian disease in cattle

disease in Holstein cows. Journal of the American Veterinary and an assessment of its treatment with a progesterone-Medical Association, 185, 212–13.

releasing intravaginal device. Veterinary Record, 147, 355–

Jobst, S.M., Nebel, R.L., McGilliard, M.L. & Pelzer, K.D.

9.

(2000) Evaluation of reproductive performance in lactating

Esslemont, R.J. & Kossaibati, M.A. (1997) Culling in 50 dairy dairy cows with prostaglandin F2a, gonadotrophin-releasing

herds in England. *Veterinary Record*, **140**, 36–9.

hormone, and timed artificial insemination. *Journal of Dairy* Esslemont, R.J. & Mawhinney, I. (1996) The cost benefit of a Science, **83**, 2366–72.

planned breeding routine for dairy cows (Ovsync/Inter-

Kastelic, J.P. (1994) Noninfectious embryonic loss in cattle.

cept). *Cattle Practice*, **4**, 293–300.

Veterinary Medicine, **89**, 584–9.

Fathalla, M.A., Geissinger, H.D. & Liptrap, R.M. (1978) Effect Kessy, B.M. & Noakes, D.E. (1979) Determination of patency of endometrial damage and prostaglandin F2a on experi-of fallopian tubes in the cow by means of phenosulphon-

mental cystic ovarian follicles in the cow. *Research in*

phthalein and starch grain tests. *Veterinary Record*, **105**, *Veterinary Science*, **25**, 269–79.

414–20.

Fourichon, C., Seegers, H. & Malher, X. (2000) Effect of

Kossaibati, M.A. & Esslemont, R.J. (1995) Wastage in Dairy disease on reproduction in the dairy cow: a meta-analysis.

Herds. Report No. 4 DAISY – The Dairy Information

Theriogenology, **53**, 1729–59.

System. Department of Agriculture, University of Reading.

Franco, O.L., Drost, M., Thatcher, M.J., Shille, V.M. &

Kruif A. de (1975) *Fertiliteit en subfertiliteit Nj het vrouwelijk Thatcher*, W.W. (1987) *Fetal survival in the cow after preg-rand*. Thesis, Utrecht.

nancy diagnosis by palpation per rectum. *Theriogenology*, Laitinen, E.R., Tenhunen, M., Haenninen, O. & Alanko, M.

27, 631–43.

(1985) *Milk progesterone in Finnish dairy cows: a field study*

Failure to Conceive and Embryonic Loss • 575

on the control of artificial insemination and early preg-

Newell, D.G., Duim, B., van Bergen, M.A.P., Grogono-

nancy. *British Veterinary Journal*, **141**, 297–307.

Thomas, R. & Wagenaar, J.A. (2000) *Speciation, subspecia-*

Lamming, G.E. & Darwash, A.O. (1998) *The use of milk prog-*

tion and subtyping of Campylobacter spp. associated with esterone profiles to characterise components of subfertility

bovine infection and abortion. *Cattle Practice*, **8**, 421–5.

in milked dairy cows. *Animal Reproduction Science*, **52**, O’Callaghan, D. & Boland, M.P. (1999) *Nutritional effects on* 175–90.

ovulation, embryo development and the establishment of

Lane, E.A., Austin, E.J. & Crowe, M.A. (2001) *Oestrous*

pregnancy in ruminants. *Animal Science*, **68**, 299–314.

synchronisation in cattle. *Cattle Practice*, **9**, 211–15.

O’Callaghan, D., Lozanot, J.M., Fahey, J., Gath, V., Snijders, S.

Loeffler, S.H., Vries, M.J. de. & Schukken, Y.H. (1999) *The*

& Boland, M.P. (2000) Recent developments in the effect of effect of time of disease occurrence, milk yield, and body

nutrition on fertility in dairy cows. *Irish Veterinary Journal*, condition on fertility of dairy cows. *Journal of Dairy Science*, **53**, 417–25.

82, 2589–604.

Okuda, K., Kito, S., Sumi, N. & Sato, K. (1988) A study of McDougall S. (2001) Reproductive performance of anovula-the central cavity in the bovine corpus luteum. *Veterinary tory anoestrus postpartum dairy cows following treatment*

Record, **123**, 180–3.

with two progesterone and oestradiol benzoate-based pro-

Oltner, R. & Edqvist, L.E. (1981) Progesterone in defatted tocols, with or without resynchrony. *New Zealand Veterinary milk: its relation to insemination and pregnancy in normal*

Journal, **49**, 187–94.

cows as compared with cows on problem farms and indi-

McGowan, M.R. & Kirkland, P.D. (1995) Early reproductive

vidual problem animals. *British Veterinary Journal*, **137**, loss due to bovine pestivirus infection. *British Veterinary* 78–87.

Journal, **151**, 263–70.

Ott, R.S. & Gustafsson, B.K. (1981) Therapeutic application Mann, G.E. (1997) Early pregnancy in the cow. *Cattle*

of prostaglandins for post partum infections. *Acta Veteri-*

Practice, **5**, 349–51.

naria Scandinavica, **77** (Suppl), 363–9.

Mann, G.E. & Lamming, G.E. (1995) Progesterone inhibition

Peeler, E.J., Otte, M.J. & Esslemont, R.J. (1994) Inter-
of the development of the luteolytic signal in cows. *Journal relationships of
periparturient diseases in dairy cows.*

of Reproduction and Fertility, **104**, 1–5.

Veterinary Record, **134**, 129–32.

Mann, G.E. & Lamming, G.E. (1999) *The influence of prog-*

Penny, C.D. & Lowman, B.G. (2002) *Mating beef cows without esterone during
early pregnancy in cattle. Reproduction in natural service – a triple
synchronisation system. Cattle*

Domestic Animals, **34**, 269–74.

Practice, **10**, 23–6.

Mann, G.E. & Lamming, G.E. (2001) *Relationship between*

Penny, C.D., Lowman, B.G., Scott, N.A. & Scott, P.R. (2000) *maternal endocrine
environment, early embryo develop-Repeated oestrus synchronisation of beef
cows with prog-*

ment and inhibition of the luteolytic mechanism in cows.

esterone implants and the effects of a gonadotrophin-

Reproduction, **121**, 175–80.

*releasing hormone agonist at implant insertion. Veterinary May, P.J. (1996) In
straw thaw bovine embryos: new opportu-Record*, **146**, 395–8.

nities. Cattle Practice, **4**, 237–40.

Pepper, R.T. & Dobson, H. (1987) *Preliminary results of treat-Mihm, M.,*

Baguisi, A., Boland, M.P. & Roche, J.F. (1994) *Asso-ment and endocrinology of
chronic endometritis in the*

ciation between the duration of dominance of the ovulatory

dairy cow. *Veterinary Record*, **120**, 53–6.

follicle and pregnancy rate in beef heifers. *Journal of Repro-Peters*, A.R., Martinez, T.A. & Cook, A.J.C. (2000) A meta-

duction and Fertility, **102**, 123–30.

analysis of studies of the effect of GnRH 11–14 days after

Morrow, D.A. (1986) *Current Therapy in Theriogenology* 2.

insemination on pregnancy rates in cattle. *Theriogenology, Diagnosis, Treatment and Prevention of Reproductive Dis-54*, 1317–26.

eases in Small and Large Animals, pp. 1–143. W.B. Saunders, Refsdal, A.O. (1979) *Undersøkelse av kviger og kyr utsjaltet*

Philadelphia.

paa grunn av ufruktbarhet. Thesis, Oslo.

Nakao, T., Shirakawa, J., Tsurubayashi, M., Oboshi, K., Abe, T., Reid, I.M., Roberts, C.J. & Manston, R. (1979) *Fatty liver and Sawarmikai*, Y., Sago, N., Tsunoda, N. & Kawata, K. (1984)

infertility in high-yielding dairy cows. *Veterinary Record*, A preliminary report on the treatment of ovulation failure

104, 75–6.

in cows with gonadotrophin-releasing hormone analog or

Robinson, R.S., Mann, G.E., Lamming, G.E. & Wathes, D.C.

human chorionic gonadotrophin combined with insemina-

(1999) *The effect of pregnancy on the expression of uterine*

tion. *Animal Reproduction Science*, **7**, 489–95.

oxytocin, oestrogen and progesterone receptors during

Nakao, T., Sugiliashi, A., Saga, N., Tsunoda, N. & Kawata, K.

early pregnancy in the cow. *Journal of Endocrinology*, **160**, (1983) Use of milk progesterone enzyme immuno-assay for

21–33.

differential diagnosis of follicular cyst, luteal cyst and corpus Rowlands, G.J., Lucey, S. & Russell, A.M. (1986) Susceptibil-luteum in cows. *American Journal of Veterinary Research*, ity to disease in the dairy cow and its relationship with

44, 888–90.

occurrences of other diseases in the current or preceding

Nanda, A.S., Ward, W.R. & Dobson, H. (1989) Treatment

lactation. *Preventive Veterinary Medicine*, **4**, 223–34.

of cystic ovarian disease in cattle – an update. *Veterinary Royal*, M.D., Darwash, A.O., Flint, A.P.F., Webb, R., Woolliams,

Bulletin, **59**, 537–56.

J.A. & Lamming, G.E. (2000) Declining fertility in dairy

Nanda, A.S., Ward, W.R., Williams, P.C.W. & Dobson, H.

cattle: changes in traditional and endocrine parameters of

(1988) Retrospective analysis of the efficacy of different

fertility. *Animal Science*, **70**, 487–501.

hormone treatments of cystic ovarian disease in cattle.

Saloniemi, H., Groehn, Y. & Syvaejaervi, J. (1986) An epi-

Veterinary Record, **122**, 155–8.

demiological and genetic study on registered diseases in

Finnish Ayrshire cattle. II. Reproductive disorders. Acta

Taylor, A.J. (2002) Venereal Campylobacter infection in cattle.

Veterinaria Scandinavica, **27**, 196–208.

Cattle Practice, **10**, 35–42.

Savio, J.D., Boland, M.P., Hynes, N. & Roche J.F. (1990)

Tebble, J.E., O'Donnell, M.J. & Dobson, H. (2001) Ultrasound Resumption of follicular activity in the early post-partum

diagnosis and treatment outcome of cystic ovaries in cattle.

period of dairy cows. Journal of Reproduction and Fertility, Veterinary Record, **148**, 411–13.

88, 569–79.

Trimberger, G.W. (1948) Breeding efficiency in dairy cattle

Schermerhorn, E.C., Foote, R.H., Newman, S.K. & Smith R.D.

from artificial insemination at various intervals before and

(1986) Reproductive practices and results in dairies using

after ovulation. University of Nebraska College of Agricul-

owners or professional inseminators. Journal of Dairy

ture, Agricultural Experimental Station, Research Bulletin

Science, **69**, 1673–85.

153.

Schofield, S.A., Kitwood, S.E. & Phillips, C.J.C. (1999) The Wagner, W.C. & Li,

P.S. (1982) Influence of adrenal corticoids-effects of a post partum injection of prostaglandin F2a on steroids on postpartum pituitary and ovarian function. In return to oestrus and pregnancy rates in dairy cows. The Vet-Factors Influencing Fertility in the Postpartum Cow (ed. by H. Veterinary Journal, 157, 172–7.

Karg & E. Schallenberger), pp. 197–219. Nijhoff, The Hague.

Sheldon, I.M. (1999) Bovine endometritis: a review. Journal of Warren, M.E. (1984) Biological targets for fertility and their Animal Breeding, 2, 2–14.

effects on herd economics. In Dairy Cow Fertility (ed. by Sheldon, I.M. & Dobson, H. (1993) Effects of gonadotrophin R.G. Eddy & MA. Ducker), pp. 1–14. British Veterinary

releasing hormone administered 11 days after insemination

Association, London.

on the pregnancy rate of cattle to the first and later serv-

Wathes, D.C., Robinson, R.S., Pushpakumara, A., Cheng, Z. & ices. Veterinary Record, 133, 160–3.

Abayasekara, D.R.E. (1998) Nutritional effects on repro-

Singh, E.L. (1987) The disease control potential of embryos.

ductive performance in dairy cows. Cattle Practice, 6, 371–7.

Theriogenology, 27, 9–20.

Watson, E.D., Jones, P.C. & Saunders, R.W. (1987) Effect of Skinner, J.G., Gray, D., Gebbie, F.E., Beckers, J-F. & Sulon, J.

factors associated with insemination on calving rate in dairy

(1996) Field evaluation of pregnancy diagnosis using bovine

cows. *Veterinary Record*, **121**, 256–8.

pregnancy-associated glycoprotein (bPAG). *Cattle Practice*, Watson, E.D. & MacDonald, B.J. (1984) *Failure of conception* **4**, 281–4.

in dairy cattle: progesterone and oestradiol-17 β concentra-

Sreenan, J.M. & Diskin, M.G. (1986) *The extent and timing of tions and the presence of ovarian follicles in relation to the embryonic mortality in cattle. In Embryonic Mortality in*

timing of artificial insemination. British Veterinary Journal, Farm Animals (ed. by J.M. Sreenan & M.G. Diskin). Nijhoff, **140**, 398–406.

Dordrecht/Boston/Lancaster.

Watson, E.D. & Munro, C.D. (1984) *Adrenal progesterone*

Sreenan, J.M., Leese, H.J., Morris, D.G. & Dunne, L. (1999) *production in the cow. British Veterinary Journal*, **140**, *Early embryo development and survival in cattle. Cattle*

300–306.

Practice, **7**, 267–70.

Webb, R., Garnsworthy, P.C., Gong, J.G., Gutierrez, C.G.,

Stolla, R., Bostedt, H., Wendt, V. & Leidl, W. (1980) *Zur*

Logue, D., Crawshaw, W.M. & Robinson, J.J. (1997) *Nutri-*

Ovarialzyste des Rindes. III. Vergleichende Wertung von

tional influence on subfertility in cattle. Cattle Practice, **5**, *Therapieverfahren. Berliner und Muenchner Tieraerztliche*

361–7.

Wochenschrift, **93**, 4–10.

Webb, R., Royal, M.D., Gong, J.G. & Garnsworthy, P.C. (1999) Stringfellow, D.A. & Wrathall, A.E. (1995) Epidemiological The influence of nutrition on fertility. *Cattle Practice*, **7**, implications of the production and transfer of IVF embryos.

227–34.

Theriogenology, **43**, 89–96.

Whitaker, D.A., Kelly, J.M. & Smith, S. (2000) Disposal and Studer, E. & Morrow, D.A. (1978) Postpartum evaluation of

disease rates in 340 British dairy herds. *Veterinary Record*, bovine reproductive potential: comparison of findings from

146, 363–7.

genital tract examination per rectum, uterine culture and

Wolfenson, D., Roth, Z. & Meidan, R. (2000) Impaired repro-endometrial biopsy. *Journal of the American Veterinary*

duction in heat-stressed cattle: basic and applied aspects.

Medical Association, **172**, 489–94.

Animal Reproduction Science, **60/61**, 535–47.

Sturman, H., Oltenacu, E.A.B. & Foote, R.H. (2000) Impor-

Wrathall, A.E. (1995) Embryo transfer and disease transmis-

tance of inseminating only cows in estrus. *Theriogenology*, sion in livestock: a review of recent research. *Theriogenol*-53, 1657–67.

ogy, **43**, 81–8.

Chapter 37

Fetal Loss

G. Caldow and D. Gray

Introduction

577

(Bolin & Alt, 1998) can cause embryonic death or death

Pathophysiology

577

that occurs shortly after birth. Such embryonic loss or

Placentitis

578

early abortion is difficult to detect and increased neona-

Causes of abortion

578

tal mortality may not be immediately associated with

Viral causes of abortion

578

disease processes that affect the fetus.

Bovine viral diarrhoea (BVDV)

578

It is also important to define what is normal or

Bovine herpes virus 1 (BHV1)

579

unavoidable loss. Spontaneous chromosomal abnor-

Other viruses

579

Bacterial causes of abortion

580

malities are commonly held to account for the bulk of

Brucellosis

580

unavoidable wastage and a loss of 20 per cent of

Leptospirosis

580

embryos by day 45 of gestation has been estimated

Bacillus licheniformis

581

(Roche et al. , 1981). After this period losses are far Listeria monocytogenes

581

lower: an abortion rate of 6.5 per cent was calculated

Salmonellosis

582

by Forar et al. (1995) who reviewed 26 published

Campylobacter

582

studies. Stillbirth losses of 4 to 6 per cent have been

Miscellaneous bacteria

584

quoted (Miller, 1988) and a study of more than 2000

Protozoal causes of abortion

584

beef cow calvings found a mortality rate of 4.5 per cent

Trichomoniasis

584

within 24 hours of birth (Nix et al. , 1998). In contrast, Neosporosis

584

the target used in beef production in Britain for live

Other infectious agents causing abortion

586

Chlamydia and rickettsia

586

calves born is 94 per cent (MLC, 1998) and the estimate

Mycotic abortion

586

of 1.9 to 2.0 per cent losses after day 42 of gestation

Non-infectious causes of abortion

586

(Sreenan & Diskin, 1986) may be a more realistic target

Investigation

587

for unavoidable fetal loss. This is further supported by

Conclusion

589

a survey of Irish dairy cows that found an abortion rate

of 1.7 per cent (Mee, 1992). From this it would seem

justifiable to use an interference level of 3 per cent

Introduction

for abortions or losses after confirmation of pregnancy

at around 6 weeks and one of 2 per cent for deaths at

The failure of the cow to carry a calf to full term or to

parturition and in the neonatal period.

produce a live calf causes financial loss through disrup-

tion of the normal pattern of milk production, the loss

of genetic material or an absolute loss in output for the

Pathophysiology

beef cow. These losses can have a profound effect at the

herd level. For example, neosporosis has been reported

The clinical manifestation of disease of the conceptus

to cause abortion rates of 26 per cent (Wouda et al. , is largely the consequence of the stage of develop-1999). Losses on this scale may be relatively unusual,

ment when exposure to the agent occurs (McGowan &

but annual losses of 10 per cent are not uncommon.

Kirkland, 1995), the difference in virulence between

Assessing the scale of these losses is made difficult by

strains of recognized fetal pathogens (Dubovi, 1992)

the definitions used and the differing clinical manifes-

and the degree and duration of exposure in the case of

tations. In Britain abortion is described as the birth of

non-infectious causes. The corpus luteum is necessary

a live or dead calf before 271 days' gestation and still-

for maintenance in the first half of gestation (Wendorf

birth as the production of a dead calf after 272 days of

et al. , 1983) and destruction of the corpus luteum during gestation (Noakes, 1986). However, agents that infect

that time will usually result in the termination of preg-

the conceptus, such as bovine viral diarrhoea virus

nancy and the expulsion of a fresh fetus and mem-

(McGowan & Kirkland, 1995) or leptospira organisms

branes. However, fetal death may either cause lysis of

577

578 • Chapter 37

*the corpus luteum and the expulsion of an autolysed
of the virus to cause abortion in the early stages of
fetus some days later or the corpus luteum may persist
gestation, at a time when both finding the fetus and
and the fetus and membranes then undergo mummifi-
achieving a diagnosis are more difficult, suggest that the
cation. If bacterial infection is present maceration
true rate of fetal loss due to BVDV may be higher.
of the fetus is the result. As a consequence, early
Infection is most often introduced into the herd by
abortions are rarely observed and when they are the
the purchase of a persistently infected (PI) breeding
advanced autolysis limits the examinations that can be
replacement and can be maintained in the herd by
carried out.*

the birth and survival to breeding age of further PI

*The fetus and placenta maintain the pregnancy in the
animals. In the beef herd infection can be maintained*

later stages. Fetal death removes this support and abortion follows after a period of a few days. Autolytic change is a feature in these cases too, characterized by the presence of bloody fluid in the body cavities (Dillman & Dennis, 1976) and a loss of microscopic cell detail. However, fetal death is not the only consequence animals are also infectious to others for at least 15 days of disease processes affecting the fetus and placenta. (Duffell & Harkness, 1985). Both persistently infected Fetal stress primarily due to anoxia will trigger the bulls (Meyling & Mikel-Jensen, 1988) and bulls under-steroid response necessary for normal parturition going acute infection (Paton et al. , 1989) can pass the (Knickerbocker et al. , 1986) and a relatively fresh fetus virus in semen. can be expelled in these circumstances; indeed, calves Acute infection during the breeding season and in

may be born alive and survive from day 260. Signs the first trimester of pregnancy can result in a range of prolonged fetal stress include staining of the perineum by meconium and inhalation or ingestion of meconium.

(Kirkland et al. , 1997). Infection in both the first and the second trimester can cause abortion or developmental

abnormalities, particularly of the nervous system

Placentitis

(Baker, 1987). The virus appears to be less pathogenic to the fetus in the third trimester and from 100 to 120

Placentitis is the principal lesion in several infectious days of gestation the fetus can mount an immune disease processes associated with fetal loss, and in cattle response to the virus, a response that becomes more the placenta is often retained after abortion in the effective as the pregnancy progresses.

second half of gestation. The precise reasons for pla-

Following acute infection, the virus multiplies in the

central retention are not known, but this is a further placentome, but does not cause placentitis, before factor limiting the investigation of many cases.

moving into the fetus. In the first trimester lesions in the fetus include meningitis with cerebellar cortical destruction and necrotizing bronchiolitis (Casaro et al. , **Causes of abortion** 1971), but these are rarely observed due to autolysis.

Sublethal damage in the first trimester and beyond can A wide range of infectious and non-infectious causes of result in the birth of calves with lesions such as retinal abortion have been reported, many of which are associated with only occasional or sporadic fetal loss. The difference in virulence between strains described for both type 1 and following discussion concentrates on the more prevalent type 2 viruses (Dubovi, 1992), along with stage of gestation conditions in British herds, with only occasional reference to exotic causes.

and the variation in the level of herd immunity, explain the wide differences seen in the outcome of BVDV

infection in breeding herds.

Viral causes of abortion

The virus can be isolated from an aborted fetus although the success of this is limited by autolysis. It can also be demonstrated in fixed tissues using immunohis-

Bovine viral diarrhoea (BVDV) (see p. 853)

tochemistry. This allows the agent to be associated with

Laboratory diagnostic rates of 5.4 per cent (Kirkbride,

lesions observed on histological examination (Ellis

1992a) and 6.5 per cent (Caldow et al. , 1996) have been et al. , 1995). Nested reverse chain transcriptase poly-reported, but the widespread evidence of infection in

merase chain reaction (PCR) techniques have been

English dairy herds (Paton et al. , 1998) and the ability used to detect viral RNA in tissues and may, in future,

Fetal Loss • 579

become the diagnostic technique of choice (Nettleton

attributed to natural infection with BHV1, fetuses show

& Entrican, 1995). As maternal antibody cannot cross

no typical gross lesion, but histological examination

the bovine placenta, antibody detected in fetal fluids

consistently reveals multifocal coagulative necrosis in

indicates fetal infection. However, when antibody is the liver and less frequently in the lung, lymph nodes, detected in late abortions without the presence of specific pathology BVDV may not have been responsible and the absence of fetal antibody suggest an acute for fetal death (Brown et al. , 1979).

infection and appear to conflict with the prolonged Maternal immunity to infection is not life-long. Infection period between experimental infection and abortion. tion with heterologous strains of the virus can also Virus can be isolated from up to 50 per cent of fetuses overcome natural immunity. Control should therefore that show histological lesions typical of BHV1 infection be based on a programme that includes best practice with the placenta being the most useful tissue for biosecurity, identifying and removing persistently infected breeding animals from the herd and screening that have been described include fluorescent antibody purchased replacements for the presence of PI animals.

staining on cryostat sections of fetal tissues, immunos-
Vaccines (p. 1011) now available in Britain have been
taining of lesions observed in fixed tissues and PCR
shown to prevent fetal infection (Brownlie et al. , 1995), techniques (Rocha et al.
, 1999).

but vaccination should not be relied upon as the sole
It seems likely that active immunity ensures that re-
preventive strategy. The success of eradication and
infection does not result in significant clinical disease
biosecurity as a means to control BVD can be meas-
and the use of a live modified vaccine has been shown
ured by the progress made by the countries that have
to prevent abortion following experimental challenge
embarked on national or regional control programmes
(Cravens et al. , 1996). However, it remains unclear how (Hult & Lindberg,
1998)

long after infection cows are protected from the abor-
tifacient effects of the virus.

As BHV1 is an insignificant cause of fetal loss

Bovine herpes virus 1 (BHV1) (see also p. 289)

in Britain there is little financial incentive to control

In Western Europe there is little to suggest that BHV1

the disease in breeding herds for this reason alone.

is a significant cause of abortion. In contrast, in North America BHV1 has been shown to be the most frequently diagnosed viral cause of abortion (Kirkbride, 1992a; Anderson et al. , 1997) and experimental challenge models to test vaccine efficacy have resulted in a high rate of fetal loss (Miller et al. , 1995). This difference may be the consequence of a variation in virulence between the strains of the virus found on the two continents.

However, the virus may be found in the semen of antibody-positive bulls to third countries within the European Union (EU). Several continental European countries have successfully eradicated the disease or have national control programmes in place and individual herds within these countries are unable to import seropositive animals. There is therefore a sound argument for pedigree and high genetic merit herds to eradicate the disease by a test and cull programme

two clinical syndromes: infectious bovine rhinotracheitis (IBR) and infectious pustular vaginitis (IPV). Vaccines offer an alternative to a test and cull programme and have been used in the Netherlands with a particular genotype of the virus and abortions (Kaashoek et al. , 1995). Even where breeding herds have been observed with both subgroups (Barr & BonDurant, 1997). As with all herpes viruses the agent difference in virulence between European and North American strains of the virus makes it prudent to can be re-activated at times of stress (Kaashoek et al. , exclude exotic strains of BHV1 by implementing biosecurity measures. In young animals epidemic IBR often follows the

introduction of market purchased animals to the herd.

Similar outbreaks of clinical disease may be seen in

Other viruses

adults, but subclinical disease is more common in this

age group. Infection in breeding herds tends to become
While a range of other viruses have been isolated from
endemic, with young cows infected as they enter the
aborted fetuses there is little to support their role as
breeding herd (Pritchard, 1992).

important causes of fetal death. The exceptions are
Experimental infection with the virus at 25 to 28
bovine herpes virus 4 (BHV4) and Akabane virus.
weeks of pregnancy resulted in abortions between 17

There is epidemiological evidence of an association
and 85 days later (Miller et al. , 1991). In abortions between BHV4
seropositivity and abortion in cows in

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Belgium (Czaplicki & Thiry, 1998) and in the USA the
are pursued. Vaccination has been used in many
virus was the third most frequently isolated from fetal
national or regional eradication programmes, often in
submissions to a diagnostic laboratory over a 10-year
the early stages of control (see p. 1013).
period (Kirkbride, 1992b). There is no published record
of the agent's involvement in British cattle and rele-

Leptospirosis (see also p. 735)

vant diagnostic tests are rarely used. It may be of value to consider BHV4 when the usual investigative

The disease in cattle is caused by the organisms col-procedures in problem herds have been unsuccessful.

*lectively referred to as *Leptospira hardjo* (*L. borg-Akabane* virus is an arthropod borne virus that has*

petersenii serovar *hardjo* and *L. interrogans* serovar been shown to cause a range of congenital abnormali-*hardjo*, previously named *Leptospira interrogans* seroties that include arthrogryposis and hydranencephaly as

vars hardjobovis and hardjoprajitno, respectively).

well as abortion. The virus occurs in Australia, Japan,

These are host-adapted serovars responsible for disease

South East Asia, Africa, Israel and Turkey and it would

in cattle. In New Zealand there is little evidence that

appear to be maintained in a range of mosquito and

these are significant pathogens of cattle and their

culicoides species (Charles, 1994). The significance of

importance lies in their zoonotic impact (Bolin & Alt,

Akabane virus to British herds is limited, but the trade

1998). Conversely, disease surveillance figures from

in live animals that would follow inclusion of countries

New Zealand have found *Leptospira pomona*, a serovar such as Turkey to the European Union could render this that is not adapted to cattle, in 1.3 per cent of abortion virus something other than an exotic curiosity.

cases examined (Thornton, 1991). In Victoria, Australia, experimental infection of pregnant heifers with a

Bacterial causes of abortion

locally isolated strain of *L. interrogans* serovar hardjo failed to cause abortion or to establish fetal infection

(Chappel et al. , 1989). In Britain and Ireland lepto-

Brucellosis (see also p. 461)

spirosis is associated with infertility, abortion and

According to EU criteria Britain is officially free of milk drop. As it is difficult to detect evidence of infec-

Brucella abortus infection, but animals from areas with tion in the fetus it is also difficult to determine the true

a lower status for this disease are imported and as a con-

significance of leptospirosis as cause of fetal death,

sequence the government continues to carry out post

however diagnostic rates of 1.7 per cent have been

import checks and routine serological monitoring of the

observed in Scotland (Caldow et al. , 1996) while in

herds in Britain.

Northern Ireland 42 per cent of aborted fetuses showed

The organism is an intracellular pathogen that is

evidence of infection (Ellis et al. , 1982). The disease is spread in infected uterine discharges. After infection

widespread in the national herd and bulk milk antibody

has occurred, usually by ingestion, an animal remains

testing showed 75 per cent of samples from dairy herds

infected for life and the principal sign of this infection

in England and Wales to have antibody present

is abortion due to placentitis. This is characterized as a

(Pritchard, 1999).

purulent intercotyledonary placentitis with necrosis

Infection is through exposure to contaminated urine

of the cotyledons. Live calves are produced from sub-

and leptospires penetrate intact mucosal surfaces to

sequent pregnancies although the uterine discharges

establish a bacteraemia. The agent localizes in the

remain a source of infection. Apart from the severe

urinary and reproductive tracts and is passed in large

effects on cattle production the disease is also an impor-

numbers in the urine in the early stages of the disease
tant zoonosis.

and may be passed intermittently for prolonged periods

In Britain there is a legal obligation for farmers to
thereafter. Spread of infection within a herd is most
notify the government's animal health service of any
rapid during the grazing season, an observation that has
bovine abortion and it is then at their discretion to
been explained by the longer survival times for the
require the collection of diagnostic material by the
organism that occur in the moist neutral pH conditions
farmer's veterinary surgeon. Microscopic examination
found at pasture in contrast to the more acid environ-
of smears made from vaginal swabs, bacteriological
ment that prevails when animals are housed, particu-
culture of milk and vaginal swab and a range of sero-
larly where ensiled forage is fed.

logical techniques can then be employed to screen for
Infection is generally considered to be introduced to
evidence of the disease. It is important for both farmers
a herd through the addition of infected cattle, but both

and their veterinary surgeons to recognize that these sheep and horses can maintain the infection and one procedures are not used to test for other abortion study has suggested that sheep, the use of natural agents.

service and the presence of water courses that have Control of the disease is the preserve of the State run through other farms may all be risk factors for Veterinary Service in Britain and test and cull policies leptospirosis infection (Pritchard et al. , 1989).

Fetal Loss • 581

As with several other infections, abortion occurs as a the sale of breeding replacements accredited free of chronic sequel, following up to 12 weeks after what is infection.

often a subclinical acute infection of the dam. Aborted fetuses and placentas do not show any characteristic

Bacillus licheniformis

pathology that is of diagnostic value, but the time Bacillus species are saprophytes that are widely dis-course of infection is such that the fetus may have been

tributed in the environment and with the exception of
able to mount an immune response and produce anti-
Bacillus anthracis are generally considered to have little body. This antibody
can be detected in fetal fluid by the
pathogenic potential. Species differentiation is not easy,
microscopic agglutination test (MAT). Isolation of lep-
but the principal isolate from bovine abortions is *Bacil-*
tospores from fetal tissues is a preserve of the specialist
lus licheniformis. *Bacillus* organisms have been isolated laboratory and is not
routinely used as a diagnostic test.
from 9.8% of fetal specimens (Caldow et al. , 1996) and The fluorescent
antibody test performed on cryostat
outbreaks of abortion associated with this agent are
sections or impression smears of fetal tissues found
commonly observed (David, 1993). Just how an organ-
favour for some time but inherent difficulties of such
ism that is considered to have little pathogenic poten-
techniques in autolytic tissues mean that these tests too
tial can appear to be such an important fetal pathogen
are now less frequently used. Once again the detection
is not clear. It has been suggested that the risk of abor-
of specific DNA may well become the technique of

tion may be related to the weight of infection in the choice and several PCR methods have been described environment, either in silage or in other forage or in (Smith et al. , 1994).

contaminated water. However, other factors may be

The literature on maternal serological response is important and in 45 per cent of fetuses where a diagnostic extensive and serves to underline the diagnostic limi-

nosis of *Bacillus licheniformis* abortion was made evitations of serological techniques. The MAT has long

dence of fetal infection with *Neospora caninum* was

been the standard test and principally detects antibody

also found (Caldow et al. , 1996). Despite this the ex-of the IgM class. These antibodies are produced as

perimental infection by the intravenous route alone

an early response to infection and may have fallen to

has resulted in placentitis and abortion with lesions undetectable levels by the time the animal aborts.

similar to those observed in naturally occurring

The enzyme linked immunosorbant assay (ELISA) can incidents (Agerholm et al. , 1999).

be designed to detect a specific subclass of antibody

Usually abortion occurs relatively late in pregnancy or a mixture, but the commercially available ELISAs and lesions of a necrotizing placentitis are observed usually detect predominantly IgG. After infection an with thickening of the intercotyledonary areas and animal produces detectable levels of IgG by 21 to adventitious placentation. Suppurative bronchopneumonia and occasionally pericarditis can be found in the 28 days and these persist for prolonged periods. Serology does not allow the differentiation between fetus. The organism is readily isolated from the placenta, fetal lung and fetal stomach contents, but on vaccinal titres and those that follow natural infection. For these reasons maternal serology is of little value occasion the organisms can no longer be cultured from in the individual animal that aborts and is best used as the fetus despite isolation from the placenta and the a herd diagnostic technique on the understanding presence of typical pathology. that it may have limited applications in vaccinated There is no indication that the bacteraemia that pre-

herds.

cedes the abortion is associated with any clinical disease

In dairy herds a principal reason for controlling the

in the dam and no serological test has been developed.

infection by vaccination is to reduce the risk of zoonotic

In herds experiencing outbreaks group treatment

disease in those milking the cows, but one study has

with antibiotic is sometimes advocated, but there is no

claimed an improvement in fertility indices after vacci-

evidence of efficacy. It is prudent to screen cows for the

nation (Dhaliwal et al. , 1996). In beef herds the risk of presence of other agents that may be acting as cofac-zoonotic infection is low and there is no published study

tors as well as to carry out bacterial counts on silage. A

to demonstrate the value of vaccination, although this is

threshold of 10⁴ colony forming units/g has been sug-

a commonly used control strategy. Similarly the use of

gested above which forage should not be fed to preg-

quarantine, serological screening and antibiotic treat-

nant cows (David, 1993). Other potential sources of the

ment of animals that are seropositive are measures that

organism should also be investigated, including drink-

have been recommended (BCVA, 1992). A successful
ing water.

national herd control programme based on the appli-
cation of biosecurity and test and cull exists in the

Listeria monocytogenes (see also p. 904)

Netherlands. In Britain a programme of accreditation
of freedom of disease has operated since 1989 (MAFF,

There are similarities between listeria and bacillus as
1989). Both approaches offer the benefit of allowing
causes of abortion, although *Listeria monocytogenes* is

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generally considered to be a more significant pathogen.

wildlife. The carrier state is particularly important in the

It may be associated with clinical disease in the dam,

spread of the disease and *S. dublin* shows a greater ten-but is usually only a
sporadic cause of cattle abortion.

dency towards the carrier state than *S. typhimurium* in *Listeria monocytogenes*
is a small Gram-positive rod

cattle. Studies on the epidemiology of *S. typhimurium*

that grows rapidly in aerobic or microaerophilic condi-

determinant type 104 have also demonstrated the

tions at temperatures of between 3 and 45°C. It can tol-

important role of newly introduced animals in the
erate pH values as high as 9.6, but is inhibited by pH
spread of this disease (Evans & Davies, 1996). While it
values lower than 5.6 and exists as a plant saprophyte
remains unclear how important wildlife are in initiating
or as an animal pathogen (Low & Donachie, 1997). It
new outbreaks, infection was found to be maintained in
is these characteristics that are crucial to the epidemi-
rodent and wild bird populations after an outbreak.

ology of the disease in ruminants, allowing growth of
Infection of the placenta follows bacteraemia and
the organism in poorly fermented silage and infection
extension to the fetus can follow from this. However,
by ingestion.

neither infection of the fetus nor indeed placentitis is
The clinical syndromes most commonly observed in
required to cause fetal death or abortion. Where fetal
cattle are iritis and meningoencephalitis, with abortion
infection has occurred gross lesions are non-specific;
an uncommon sequel of either of these. Silage feeding
histological lesions of fetal septicaemia may be found

is associated with a risk of listeriosis, however disease and the organism may be isolated from fetal tissues and can occur when animals are grazing pasture. In addition the placenta. If the cow is showing signs of septicaemia to *L. monocytogenes*, the closely related *Listeria* at the time of abortion it is advisable to attempt to isolate the organism from faeces. Serological tests for (Alexander et al. , 1992).

salmonellosis are available, but are not used routinely In listeric abortion the aborted fetus is often for diagnostic purposes in the investigation of bovine autolytic, but non-specific lesions of placentitis and fetal abortion.

septicaemia may be seen (Barr & Anderson, 1993) and Once a diagnosis of salmonellosis has been made the organism can be readily isolated from fetal viscera, control rests on efforts to minimize further spread of stomach contents and the placenta. Serological tests for infection within the herd through improved hygiene exposure to listeria exist, but are not used in the diagnosis and thorough cleaning and disinfection. There are

nosis of bovine abortion.

conflicting reports of the effectiveness of vaccination

The same restraints on treatment and control of

against S. typhimurium in the face of an outbreak

bacillus apply to listeria. High-risk forages, which in this

(Davies & Renton, 1992; Mee & Malone, 1995), but a

case are poorly preserved silages with a pH greater than

S. dublin vaccine is available and has been used suc-

5, should not be fed to pregnant cattle. In addition to

*cessfully in endemically infected herds (Wray et al. , ensuring conditions that
promote good anaerobic fer-1989). The removal of S. dublin carriers after an
outmentation during the ensilage process, measures should*

break appears to be an attractive course of action, but

be taken to reduce soil and faecal contamination of the

this approach is impractical as excretion is periodic.

grass prior to and during harvesting.

Faeces must be cultured with negative results on three

occasions at 14-day intervals before an animal is con-

sidered to be free of infection. In addition, animals must

Salmonellosis (see also Chapter 15; p. 850)

be individually housed or stall-tied with this method as

According to British diagnostic data, salmonella organ-

there is the possibility of passive excretion if animals
isms were isolated from 159 to 300 cases of bovine abor-
are loose housed or at pasture (Radostits et al. , 1994).
tion in each of the years of 1994 to 1998 (MAFF, 1998a).
Farmers should be aware of the risks that purchased
During this period *Salmonella dublin* made up 82 per
breeding replacements pose and added animals should
cent of these diagnoses. In contrast, *S. dublin* accounted be placed in quarantine
and screened by culture as
for 31 per cent of the non-abortion salmonella cases
above.

in adult cattle whereas the figure for *Salmonella*
typhimurium was 54 per cent (MAFF, 1998b). These

Campylobacter (see also p. 568)

data suggest that *S. dublin* is more likely to manifest as a primary abortion
problem than is *S. typhimurium*.

Venereal campylobacteriosis is considered to be an

There is a variation in the number of *S. dublin* cases important cause of bovine
infertility wherever natural

diagnosed on a regional basis, indicating an endemic
service is used and abortions are a feature of this
problem in some areas.

disease. Recent observations have suggested that the *Salmonella* organisms are members of the Enterobacteriaceae and as such can be found as part of the intestinal flora in many species of domestic animals and Britain than has been widely presumed (Caldow & Taylor, 1997).

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Campylobacter fetus is a microaerophilic, motile

Diagnosis of *C. fetus* as a cause of abortion rests on bacterium that has two recognized subspecies: *C. fetus*

the isolation of the organism from the fetus. As faecal

subsp. *venerealis* and *C. fetus* subsp. *fetus*. Differentiation-contamination of the placenta may occur after the abor-

tion has been based on the variation of antigenic and

tion, isolation from the placenta should not be consid-

biochemical characteristics of the subspecies, notably

ered diagnostic unless reinforced by histopathology.

that *C. fetus* subsp. *venerealis* will not grow in 1 per cent After venereal infection in the cow, antibody is

glycine whereas *C. fetus* subsp. *fetus* will. This difference-secreted in the uterine and vaginal fluid. Of this IgG is

tiation is important, as *C. fetus* subsp. *venerealis* is con-the most important

component and can be detected

sidered to be adapted to the bovine reproductive tract

eight weeks post infection, but is largely undetectable

and to be the cause of venereal campylobacteriosis,

by six months. IgA is produced by three to five weeks

whereas *C. fetus* subsp. *fetus* is a gut commensal caus-and is still detectable for up to a year after infection

ing sporadic abortion in cattle. However, strains of

(Corbeil et al. , 1974). Despite recovery, the developthe latter vary in their biochemical characteristics

ment of immunity and the establishment of normal

and strains identified as *C. fetus* subsp. *venerealis* have pregnancies, *C. fetus* has been isolated from the vaginal developed tolerance to glycine (Chang & Ogg, 1971).

mucus more than a year after infection (MacLaren &

In addition, strains identified as *C. fetus* subsp. *fetus*, Agumbah, 1988), creating the potential for cows as well

when used in experimental studies, have resulted in

as bulls to maintain the infection in the herd from one

disease indistinguishable from that caused by *C. fetus*

year to the next.

subsp. *venerealis* (Schurig et al. , 1973) and field The sampling technique most frequently used in bulls

outbreaks of venereal campylobacter infertility asso-

in Britain is the collection of sheath washings (BCVA, 1995) and phosphate buffered saline (PBS) is adequate (MacLaren & Agumbah, 1988). It would therefore appear that strains of both subspecies are capable of surviving from gross contamination and transported to the laboratory causing venereal infertility and whenever *C. fetus* is isolated within four to six hours. However, where immediate delivery to the laboratory cannot be achieved the venereal infertility should be investigated. The use of transport enrichment medium (TEM) has proved to be effective in maintaining the viability of the organism. In the case of sporadic abortion not associated with venereal infertility, invasion from the intestinal tract is probable (Hum et al. , 1994). A fluorescent antibody test is the probable route of infection. This test is available, but does not allow differentiation between the two subspecies and it is of limited value in the diagnosis of the disease. The venereal form of the disease is usually introduced to the herd via an infected mature bull and while

nosis of the disease. Vaginal mucus should also be collected from younger bulls or breeding females are less likely to be collected without gross faecal contamination and this can be a source, any animal that has previously been mated constitutes a potential source of infection. The use of hired off and perforations made in the last few centimetres of the bulls is considered to be a particularly high-risk practice. Twenty ml of PBS are injected through a 60 ml syringe. Infection in the bull follows natural mating with an infected cow, but is not associated with clinical signs (Lander, 1983). As with sheath washings, TEM can be used if there is to be a delay of more than six hours between sample collection and the inoculation of crypts of the prepuce, while bulls younger than 6 years of age are more resistant to infection (Clark, 1971). In the vaginal mucus agglutination test (VMAT) anti-

natural service the spread of infection is from the penis
gels prepared from *C. fetus* are used to detect agglu-
to the anterior vagina, where the organism becomes
tinins in the vaginal mucus. Immunoglobulin A is the
established before entering the uterus to cause a mild
antibody present in the mucus, but where serum or
endometritis and salpingitis. Pathological changes are
blood contaminates vaginal secretions false positives
most pronounced at 8 to 13 weeks after infection and
can arise as serum antibody to *C. fetus* is common in
have resolved by four to five months (Dekeyser, 1986).
cattle (Wilkie & Winter, 1971). In addition, where cattle
Where conception has occurred embryonic death and a
are in oestrus, large amounts of mucus is produced, IgA
delayed return to oestrus, abortion or the birth of a live
is diluted and false negatives can occur. Problems have
full term calf can follow. Of the abortions that are
been reported with the test and it has been suggested
detected the gestational length is usually around four
that only 50 per cent of infected animals will be posi-
to five months. A marked placentitis and pericarditis

tive by VMAT and of those 50 per cent will be negative may be seen on gross examination of the tissues and six months after infection. An enzyme-linked histological findings are of fetal septicaemia and immunoassay has been developed to overcome some of pneumonia.

the problems experienced in using the VMAT (Hewson

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et al. , 1985; Hum et al. , 1991), but the test is not currently The second category includes *Yersinia pseudotuberculosis* used in Britain.

culosis, *Mannheimia haemolytica*, *Erysipelothrix rhusiopathiae* As a consequence of the delay between infection and

siopathiae and *Staphylococcus aureus* where infection of investigation in most herds and the cows' ability to clear

the fetus follows bacteraemia in the dam. *Arcanobacterium*

the infection as well as the limitations of the vaginal

terium pyogenes may also gain entry to the conceptus

mucus agglutination test it is necessary to sample a

in this way and experimental infection via the intra-

minimum of 12 cows. Where endemic infection is sus-

venous route with this organism has resulted in abor-

pted animals in their first year in the breeding herd

tion (Semambo et al. , 1991).

should be sampled to offer the best chance of achieving

These infections are unlikely to represent a herd

a diagnosis. Bulls should be sheath washed in the course

problem and as such do not merit further discussion.

of a herd investigation, but a negative result is of little

value as the screening is a relatively insensitive test.

Artificial insemination using semen from

Protozoal causes of abortion

campylobacter-free studs offers a simple and successful

method of control, but for some herds, particularly

Trichomoniasis

where mating occurs at grass, this may not be practical.

Vaccination is used in many countries where the disease

Bovine trichomoniasis is caused by the protozoan par-

occurs in range cattle and a vaccination programme

asite *Tritrichomonas fetus* and is a venereal disease

to eradicate infection from within a herd has been

responsible for early embryonic death, infertility,

described (Hum & MacInnes, 1987). Antibiotic treat-

pyometra and abortion. It is a particular problem in

ment of the bulls is suggested as an additional measure range cattle, where as many as 50 per cent of the herds (Hum et al. , 1993). However, there is no licensed may be infected (Dennet et al. , 1974). There is no recent vaccine in Britain and an emergency vaccine must report of the disease from within Britain, but there is be produced under the authority of the Veterinary no biological barrier that would prevent the disease Medicines Directorate (see Chapter 59). from occurring here and diagnostic laboratories in Prevention can be achieved by maintaining a strict Britain do not routinely use techniques that would closed herd policy, but in practical terms the purchase identify the presence of this agent. Therefore tri- of virgin heifers and virgin bulls does not constitute chomoniasis should be considered in the differential a risk. Difficulties arise where bulls have to be re- diagnosis in herds using natural service where infertil- placed at short notice. In these situations a syn- ity, abortions and pyometra are observed. chronization and insemination programme would offer

Although most pregnancies are lost at around 17 the safest course of action with a young unproven days after conception, one review reported that death bull used to sweep up. If mature bulls are used these can occur at any time up to the fifth month of gestation should be screened and treated with antibiotics (Yule et al. , 1989), and fetal infection has also been (BCVA, 1995). demonstrated in late abortions and in full term calves (Rhyan et al. , 1988).

No specific gross changes are seen in the abortion

Miscellaneous bacteria

material. Histological examination reveals lesions of Species of bacteria associated only with sporadic abor-placentitis and pyogranulomatous bronchopneumonia tions can be split into those that can be readily isolated with evidence of tissue invasion with the parasite from the posterior reproductive tract of the normal (Rhyan et al. , 1995). The organism can be cultured animal and those that cannot.

from vaginal discharges, preputial smegma and fetal

The former category includes organisms such

tissues by using a specific medium in a pouch system as *Haemophilus somnus*, *Arcanobacterium pyogenes*, (Kvasnicka et al. , 1996). *Streptococcus dysgalactiae*, *Mycoplasma bovis* and *Ure-Trichomoniasis* can be controlled through the use of *aplasma diversum*. These may gain entry to the uterus artificial insemination and commercial vaccines are through the cervix as a sequel to some other disease available in the countries where the disease is endemic. process affecting the fetus. It is possible that strain differences in virulence exist, but at the present time **Neosporosis** (see also p. 283) these agents should be considered to be of minor importance. *Ureaplasmas* have been suggested as important, Protozoal abortion has been recognized as a problem but isolation methods for these organisms are rarely in the dairy herds of California since the mid 1980s employed in diagnostic laboratories (Sanderson & (Anderson et al. , 1991) and around the same time a Chenoweth, 1999). protozoan parasite of dogs was identified in Norway

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(Bjerkås et al. , 1984). This was subsequently named the lesions is considered significantly characteristic *Neospora caninum* (Dubey et al. , 1988) and the link was to warrant a diagnosis and immunohistochemistry made to the infection of cattle (Thilsted & Dubey, 1989). Since then *N. caninum* has been identified as a examination although tissue cysts do survive and may cause of abortion in many parts of the world and a be recognized. The fetus is capable of mounting an major volume of research publications has been gener-immunological response to neospora infection and ated (Dubey, 1999). Serological studies have found evi-detection of specific antibody in fetal or precolostral dence of exposure to the organism in 16 per cent of sera is a reliable indicator of fetal infection, but not of aborted fetuses in Scotland (Buxton et al. , 1997) and in infection causing fetal death (Otter et al. , 1997). At 13 per cent of cows that aborted in Northern Ireland present, despite the availability of molecular biological (McNamee et al. , 1996). Outbreaks described range techniques for the detection of neospora DNA they are from 11 per cent of cows that aborted in one herd in

not routinely used for diagnostic purposes.

England (Dannatt et al. , 1995), to as many as 57 per cent Maternal infection is followed by the production of

of a herd in The Netherlands (Wouda et al. , 1999). While antibodies within 14 days that peak by 46 days and in

neosporosis is a recently recognized disease there is

most cases the time lag between maternal infection and

nothing to suggest that the parasite or the disease is new.

abortion is such that antibody production will have

Neospora caninum is an apicomplexan protozoan

peaked before the abortion and may occasionally have

closely related to *Toxoplasma gondii* and although the fallen to undetectable levels by two months after

complete life cycle of the parasite is still to be deter-

the abortion (Wouda et al. , 1999). In the congenitally mined dogs have been shown to be a definitive host and

infected individual there is fluctuation in antibody level

to pass small numbers of oocysts in faeces from 8 to 23

and at times antibody is undetected. The observations

days after experimental infection (McAllister et al. , that congenitally infected animals have been found to

1998). Tissue cysts and tachyzoites have been found in

have a higher risk of abortion at their first gestation

cattle, sheep, goats and horses and it is possible that the compared to the second gestation and that abortions intestinal stage of the life cycle is not limited to the dog. occurred at an earlier stage in the first gestation than Vertical transmission occurs in addition to the point subsequently suggest the possibility of a protective source outbreaks, where cows are presumed to be immunity developing in infected animals (Thurmond & infected for the first time and abort. As post natal infection rates are probably as low as 1 per cent (Hietala & neospora, toxoplasma and sarcocystis species there Thurmond, 1999) most cases are due to vertical transmission where an infection rate of 95 per cent has been occur, however it has been shown that such cross-found (Davison et al. , 1999). Infection of the fetus may reactivity is negligible and of no importance often be a relatively benign event and care should be diagnostically (Dubey et al. , 1996). taken in attributing significance to evidence of infection

The lack of a complete understanding of the life cycle in an aborted fetus (Thurmond et al. , 1997). Co- and knowledge of the range of definitive hosts has led infection with other agents is often found and potential to a cautious approach to control, but there seems a clear argument emerging in favour of culling seropositive animals from the herd. These have been shown to some of the cases that appear as epidemics (Bartels et al. , 1999).

of abortion than seronegative cows within the herd. By Experimental infection of pregnant cattle has re- using information from the literature in a mathematical model it has been shown that annual culling of from those seen in naturally occurring outbreaks (Barr et al. , 1994). Abortion most frequently occurs in the method of control. Effective control at a slower rate

second half of pregnancy and the commonly observed could be achieved if the action was limited to excluding lesions are limited to placentitis, focal non-suppurative the offspring from seropositive cows from the breeding encephalitis, non-suppurative myocarditis and myositis herd (French et al. , 1999). At the present time the ques-and widespread non-suppurative infiltrates in other tion of the cost-benefits of these strategies has not been organs (Anderson et al. , 1991). The tissue cysts are well addressed, but given the large outbreaks that can occur, tolerated and the host response is limited to the tachy-action to prevent a sudden catastrophic, albeit low-risk, zoites. Histological examination of the fetus is impor-occurrence might be justifiable. Irrespective of culling tant in order to determine the severity and therefore practices, all fetuses, placentas and cattle carcasses the significance of the lesions in terms of fetal death should be disposed of in a way that prevents recycling (Thurmond et al. , 1999). The histological appearance of of *N. caninum* infection through definitive hosts.

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Other infectious agents

*immunity after infection or whether the cow is likely to
causing abortion*

return to normal fertility.

Prevention of mycotic placentitis is by reducing the

Chlamydia and rickettsia

*degree of fungal contamination pregnant cows are
exposed to, particularly in preserved forage. Fungal*

Q fever, Chlamydia abortus and Cytoecetes

growth occurs in other feedstuffs and bedding ma-

*phagocytophilia have all been identified as occasional terials and after a
diagnosis of mycotic abortion the*

causes of bovine abortion. There is serological evidence

management system should be reviewed in order to

of exposure to the agent of Q fever in Britain (Paiba et

identify possible sources of these organisms. A visual

*al. , 1999), but no recent report of abortion in cattle due examination of suspect
materials may be sufficient, but*

to this agent. Occasionally a purulent placentitis and

where doubt over the source remains it is also possible

the presence of elementary bodies in modified Ziehl

to use quantitative culture methods to assess the degree

Neelsen stained smears of placenta and stomach con-

*of fungal contamination and to identify the organisms
tents are observed. Establishing the precise aetiology in
present.*

*these cases can be difficult, but Chlamydophila abortus
has been isolated from such material and in one report
the strain was found to be identical to that responsible
for chlamydial abortion in sheep (Griffiths et al. , 1995).*

Non-infectious causes of abortion

*The outbreak may have been the result of close contact
between cattle and an infected sheep flock on the same
The failure to identify an infectious cause in many cases
premises.*

*has led to the suggestion that non-infectious causes are
Cytoetes phagocytophilia, the cause of tick borne
of importance (Norton & Campbell, 1990). Nutritional
fever (TBF), will cause abortion in cattle when first
factors that may be involved include excess dietary
exposure to ticks occurs during pregnancy. Diagnosis
protein or non-protein nitrogen and selenium or iodine
can be difficult to confirm, as there is no longer para-
deficiency. Twin pregnancies are associated with shorter*

sitaemia by the time the abortion occurs. A presumptive diagnosis may be based on the herd history and the (Echternkamp & Gregory, 1999). Various poisonous presence of ticks and antibody to the TBF agent. plants have been implicated, but none is indigenous to Britain. Ponderosa pine (*Pinus ponderosa*) and locoweeds (*Astragalus* sp.) cause significant problems in

Mycotic abortion

some areas of North America. Deaths due to aflatoxin. Mycotic abortion with placentitis and fetal infection is common and to nitrate toxicity may be preceded by abortion reported throughout the world. In one detailed study 7 per cent were mycotic abortions and *Aspergillus fumigatus* infection accounted for 62 per cent of these death is the consequence of the clinical disease in the dam. (Knudtson & Kirkbride, 1992). Other species of fungi

have been reported and it would appear that most are

However, the literature fails to support non-infectious causes as a numerically significant cause of abortion in cattle (Norton & Campbell, 1990) and in the UK often poorly preserved hay or ensiled feed that has undergone aerobic fermentation.

only iodine deficiency, manifest as hyperplastic goitre in Placentitis follows a septicaemia that has resulted full-term stillborn calves, is of importance. The condition is mainly reported in beef herds or in dairy heifers environment. The placenta is frequently thickened and and is thought unlikely to affect dairy cows due to adequate dietary iodine intake. A threshold of 15 g for the skin of the fetus are an occasional feature of this weight of the intact gland, above which goitre is suspected, has been in common use. A better indicator is observed. At the microscopic level hepatitis, pneumonia the ratio of thyroid weight to fetal body weight where

nia and placentitis are evident and Grocott's stain can be used to reveal fungal hyphae in fixed sections. The threshold value is 1 : 3000. However, oedema of the head and neck in stillborn calves can render this unreliable (Gee et al. , 1989) and histological examination of the placenta and the stomach contents, the latter being the gland to confirm the presence of a colloid goitre is diagnostic. These organisms are readily cultured from recommended (Capen, 1993). There does not appear to be any suggestion that goitrogenic factors should be considered and the inclusion of iodine in an oral mineral supplement should prevent the condition (see pp. 257, 301).

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Investigation

Examination of the fetus and placenta

In the ideal situation, the entire aborted calf and fetal membranes with the appropriate samples from the dam should be delivered directly to a veterinary diagnostic laboratory. Where transport difficulties or other delays preclude this, the field veterinarian should collect appropriate samples while carrying out a necropsy of the fetus. Therefore there is justification for investigating every

abortion. However, there are concerns over unnecessary costs and the diagnostic rate has been found to be adopted and the appropriate information recorded. The higher in cases where there has been more than one measurement of the crown–rump length can give an abortion in the herd previously (Caldow et al. , 1996). A formula to

Since there is a statutory requirement to report all abortions in Britain there is the opportunity to discuss the crown-rump measurement is $x = 2.5(y + 21)$, where y is the crown-rump length in centimetres and x is the gestational age in days. Measurement of the weight of the carcass is particularly important in stillbirths to explore

Investigations should follow a standard pattern:

(1)

History

the possibility of dystokia due to fetal maternal disproportion

(2)

Clinical examination of the cow

portion and for comparison with thyroid weight, for the

(3)

Necropsy of fetus

diagnosis of hyperthyroidism.

(4)

Collection of samples for the laboratory

Other important features to be aware of include the

(5)

Interpretation of results

presence of white plaques of fungal hyphae on the skin of fetuses where there is fungal involvement. Meconium staining of the perineum or the presence of meconium in

History

the airways or fetal stomach indicate fetal anoxia con-

The use of a standard form allows the systematic col-

sistent with placentitis or the prolonged parturition of

lection of the important details of the individual cow as

dystokia. Haemarthrosis of the shoulder or hip joints

well as production system, cow numbers, age structure,

may be seen in dystokia cases. Traumatic lesions in

biosecurity measures, replacement policy and source of

fetuses where dystokia has not been the problem are

replacements (Fig. 37.1). Details on the feeding system

occasionally observed and while trauma may be the

and how feeds are stored are also of relevance. This can

cause of the abortion other causes should not be

usefully be expanded to include details on herd fertil-

excluded too early in the investigation. Lesions of perity, retained membranes and any background clinical carditis, pleurisy, pneumonia or hepatitis can all be found problem that may be of relevance. Careful evaluation on the gross examination, but none of these changes is of the information is essential to guide the investigation specific. In calves that have lived for a short time after and to indicate the diagnostic tests to be employed.

birth it is important to record whether the calf has fed as this prevents useful bacteriological examination of the stomach contents, but also means that maternal antibody

Clinical examination

may have been absorbed, interfering with fetal serological tests.

In most cases examination does not yield useful infor-

When the placenta is available as much of the surface mation, but it is important to exclude conditions such area of the allantochorion as possible should be examined. Lesions can be focal and may be overlooked if fever and to allow treatment if the cow is ill. At this time

insufficient care is taken. Commonly observed lesions a blood sample for serology should be collected and if are intercotyledonary thickening, necrosis of cotyledons and adventitious placentation. The last lesion is salmonellosis is suspected a faeces sample as well.

Where stillbirth is the problem the body condition of considered to be a chronic response to insufficient the cow should be assessed and other cows in late gestational area and is seen following placentitis.

tation can also be examined in this way. Where the majority of animals in the group have scores of 3.5 and above there is cause for concern over excessive body Collection of samples for laboratory investigation condition.

Even when it has been decided to limit diagnostic If samples are collected in the course of a necropsy on tests to those required for brucellosis, a serum sample the farm, the collection and transport of samples to the can be stored frozen for possible retrospective laboratory should have been discussed with the laboratory examination.

tory personnel. The samples required and the means of

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Reference Number: C2.....

CASE HISTORY

Date of Abortion/Stillbirth.....

Ear no. of dam.....

Age of dam.....

Due on.....

Source of dam.....Home bred

Purchased

Breed..... Dam.....

Sire.....

Vaccination: BVD

L. hardjo

IBR

Other.....

No. of abortions.....

No. of stillbirths.....

No. of weak calves.....

Calving starts.....

Group size.....

Total cows.....

Replacements:

Home bred

Bought in

Mixture

Method of service

AI only

Natural only

Both

Have hired bulls been used? Yes No

Production: Beef Dairy

Body condition score of cows/heifers:

HOUSING/MANAGEMENT

Roughage

Concentrate

Mineral/trace element supplements

HERD FERTILITY (Dairy)

Average days to conception

All serves pregnancy rate

% barren after p.d. positive

HERD FERTILITY (Beef)

No. of barren cows

Length of breeding/calving season

DETAILS OF PARTURITION

Degree of assistance required/use of calving aid:

.....

Calf alive at onset of assistance: YES NO

Placenta expelled with calf:

YES NO

Vet involved with calving:

YES NO

Other information:

.....

.....

Fig. 37.1

A standard form to collect systematically important information when investigating fetal losses.

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preservation are determined by the causes of abortion

precedes abortion so that paired serology is valueless.

that occur in an area and on the diagnostic facilities

As many of the infections of importance tend to be

available to the laboratory. Nevertheless, basic rules do

endemic and circulating antibody may persist for long apply.

periods the presence of maternal antibody at the time

The minimum sample required for bacteriology is

of the abortion can only be taken to show that the cow

fetal stomach contents. These can be collected using a

has been exposed to the agent. The converse applies

sterile vacutainer and needle through the unopened

when the MAT is used for leptospirosis as the IgM anti-

abomasal wall. Where the placenta is available a cotyle-

body response can be so transient that the dam may be

don representative of the lesions observed should

seronegative at the time of abortion.

also be submitted fresh. Samples may also include

In essence, serology tests in the dam are of more

fetal liver and lung, allowing slightly higher diagnos-

value when used to exclude infection with negative

tic success for some bacterial causes of abortion . It results than to attempt to confirm a diagnosis with pos-has been suggested that in cases where the fetal mem-

itive results. This should not discourage the use of these

branes have been retained a placentome is removed

techniques to establish whether or not there is evidence

manually per vaginum (Johnson et al. , 1994) for of these diseases in the herd, as control strategies may

*bacteriological and histological examination. How-
still be required.*

ever, this is not required when the fetus is available for

*Evidence of the involvement of several infectious
examination.*

agents is often found in the course of an outbreak,

Fetal blood or fluid can be collected from any body

underlining the importance of proper evaluation of the

cavity, again by using a vacutainer. This sample is used

results, but also raising the question of a potentiation

for serological tests for BVD, neospora and leptospira.

effect amongst agents.

Fetal thyroid, spleen, kidney and brain are all tissues

that can be used for virus isolation for BVD, while the

Prognosis

placenta is the sample of choice for BHV1.

Histopathological examination is particularly useful

For the individual cow the prognosis is usually

to determine the significance of fetal infection with *N. caninum* or BVD, to confirm the significance of apparent thyroid enlargement and for further investigations oestrous cycles start quickly and, in general, subsequent pregnancy rates are satisfactory. Prognosis for the rest of the pregnant animals in the herd is a more serious consideration and varies with the diagnosed cause of abortion. The tissues of most value to collect are brain, lung, liver, thyroid (in stillbirths) and a cotyledon. It is important to be guided by the pathologist as to how

these samples should be fixed. Tissues for histology must never be frozen.

The minimum samples are fetal stomach contents for

Conclusion

bacteriology and fetal blood or serum for serological tests. These may be the samples of choice when investigations are often the most obvious manifestation of

tigating what appears to be a single abortion. If subsequent abortions occur then the range of tests can be of financial importance to the owner of the cattle herd. extended.

Failure on the part of the veterinary surgeon to pursue or advise correctly could have a severe penalty for the producer.

Interpretation of results

When evaluating results it is important to consider

References

them in relation to the diagnostic criteria for each

disease. Hence, while the presence of antibody to BVD

Agerholm, J.S., Jensen, N.E., Jensen, H.E. & Aarestrup,

or to *N. caninum* is considered to indicate fetal infection. F.M. (1999) Experimental infection of pregnant cows with

tion, it must be remembered that infection of the fetus

Bacillus licheniformis bacteria. *Veterinary Pathology*, **36**, 191–201.

need not necessarily be lethal, especially as gestational

Alexander, A.V., Walker, R.L., Johnson, B.J., Charlton, B.R. & age increases. Similarly, the isolation of foetopathic

Woods, L.W. (1992) Bovine abortions attributable to *Liste-*

bacteria or fungal organisms from the placenta in the

ria ivanovii – 4 cases. *Journal of the American Veterinary absence of histopathology is of no value as they may*

Medical Association, **200**, 711–14.

arise from environmental contamination after the pla-

Anderson, M.L., Blanchard, P.C., Barr, B.C., Dubey, J.P.,

centa has been passed.

Hoffman, R.L. & Conrad, P.A. (1991) *Neospora-like proto-In the majority of infections maternal infection*

zoan infection as a major cause of abortion in California

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dairy cattle. *Journal of the American Veterinary Association*, mucosal disease virus. *American Journal of Veterinary*

198, 241–4.

Research, **32**, 1543–62.

Anderson, M.L., Reynolds, J.P., Rowe, J.D., Sverlow K.W.,

Chang, W. & Ogg, J.E. (1971) *Transduction and mutation*

Packham, A.E., Barr, B.C. & Conrad, P.A. (1997) *Evidence*

to glycine tolerance in *Vibrio fetus*. *American Journal of of vertical transmission of Neospora sp. infection in dairy Veterinary Research*, **32**, 649–53.

cattle. *Journal of the American Veterinary Association*, **210**, Chappel, R.J., Millar, B.D., Adler, B., Hill, J., Jeffers, M.J., Jones, 1169–72.

R.T., McCaughan, C.J., Mead, L.J. & Skilbeck, N.W. (1989)

Baker, J.C. (1987) Bovine viral diarrhoea virus: a review.

Leptospira interrogans serovar hardjo is not a major cause Journal of the American Veterinary Association, 190, 1449–

of abortion in Victoria. Australian Veterinary Journal, 66, 58.

330–3.

Barr, B.C. & Anderson, M.L. (1993) Infectious diseases

Charles, J.A. (1994) Akabane virus. Veterinary Clinics of North causing bovine abortion and fetal loss. Veterinary Clinics of America Food Animal Practice, 10, 525–46.

North America: Food Animal Practice, 9, 343–68.

Clark, B.L. (1971) Review of bovine vibriosis. Australian

Barr, B.C. & BonDurant, R.H. (1997) Current Therapy in Veterinary Journal, 47, 103–7.

Large Animal Theriogenology, pp. 373–82. W.B. Saunders, Corbeil, L.B., Duncan, J.R., Schurig, G.G.D., Hall, C.E. & London.

Winter, A.J. (1974) Bovine venereal vibriosis: variations in

Barr, B.C., Rowe, J.D., Sverlow, K.W., BonDurant, R.H.,

immunoglobulin class of antibodies in genital secretions and

Ardans A.A., Oliver, M.N. & Conrad, P.A. (1994) Experi-

serum. Infection and Immunity, 10, 1084–90.

mental reproduction of bovine fetal Neospora infection and Cravens, R.L., Ellsworth, M.A., Sorenson, C.D. & White, A.K.

death with a bovine Neospora isolate. Journal of Veterinary (1996) Efficacy of a temperature-sensitive modified-live

Diagnostic Investigation, **6**, 207–15.

bovine herpes virus type-1 vaccine against abortion and

Bartels, C.J.M., Wouda, W. & Schukken, Y.H. (1999) Risk

stillbirth in pregnant heifers. Journal of the American

factors for Neospora caninum-associated abortion storms in Veterinary Medicine Association, **208**, 2031–4.

dairy herds in the Netherlands (1995–1997). Theriogenol-

Czaplicki, G. & Thiry, E. (1998) An association exists between ogy, **52**, 247–57.

bovine herpes virus-4 seropositivity and abortion in cows.

BCVA (1992) Guidelines for the diagnosis and control of Lep-Preventive Veterinary Medicine, **33**, 235–40.

tospira hardjo infection in cattle. British Cattle Veterinary Dannatt, L., Guy, F. & Trees, A.J. (1995) Abortion due to

Association, Gloucester.

Neospora species in a dairy herd. Veterinary Record, **137**, BCVA (1995) *Bull Hiring. British Cattle Veterinary Associa-*566–7 .

tion, Gloucester.

David, G.P. (1993) Abortion in cattle associated with Bacillus Bjerås, I., Mohn, S.F. & Presthus, J. (1984) Unidentified cyst-licheniformis infection – an update. Cattle Practice, **1**, 33–7.

forming sporozoon causing encephalomyelitis and myositis

Davies, T.G. & Renton, C.P. (1992) Some aspects of the epi-in dogs. Zeitschrift für Parasitenkunde, **70**, 271–4.

demiology and control of Salmonella typhimurium infection Bolin, C.A. & Alt, D.P. (1998) Clinical signs, diagnosis and pre-in outwintered suckler cows.

Veterinary Record, **131**, 528–31.

vention of bovine leptospirosis. In *Proceedings of the 20th Davison, H.C., Otter, A. & Trees, A.J. (1999) Estimation of ver-World Buiatrics Congress, Sydney, Australia, Vol. 2, pp.*

tical and horizontal transmission parameters of Neospora
899–904.

caninum infections in dairy cattle. International Journal for Brown, T.T., Schultz, A.D., Duncan, J.R. & Bistner, S.I. (1979) Parasitology, 29, 1683–9.

Serological response of the bovine fetus to bovine viral diar-
Dekeyser, P.J. (1986) Bovine genital campylobacteriosis. In
rhoea virus. Infection and Immunity, 25, 93–7.

Current Therapy in Theriogenology, Vol. 2 (ed. by D.A.

Brownlie, J., Clarke, M.C., Hooper, L.B. & Bell, G.D. (1995) Morrow), pp. 263–6. W.B. Saunders, Philadelphia.

Protection of the bovine fetus from bovine viral diarrhoea

Dennet, D.P., Reece, R.L., Barasa, J.O. & Johnson, R.H. (1974) virus by means of a new inactivated vaccine. Veterinary

Observations on the incidence and distribution of serotypes
Record, 137, 58–62.

of Tritrichomonas fetus in beef cattle in North-Eastern Aus-Buxton, D., Caldow, G.L., Maley, S.W., Marks, J. & Innes, E.A.

tralia. Australian Veterinary Journal, 50, 427–31.

(1997) Neosporosis and bovine abortion in Scotland. Vet-

Dhaliwal, G.S., Dobson, H., Montgomery, J. & Ellis, W.A.

erinary Record, **141**, 649–51.

(1996) *Reduced conception rates in dairy cattle associated*

Caldow, G.L., Buxton, D., Spence, J.A. & Holisz, J. (1996) *with serological evidence of Leptospira interrogans serovar Diagnoses of bovine abortion in Scotland. In Proceedings*

hardjo infection. Veterinary Record, **139**, 110–14.

of the British Cattle Veterinary Association, XIX World

Dillman, R.C. & Dennis, S.M. (1976) *Sequential sterile autol-Buiatrics Congress, Vol 1, pp. 191–4.*

ysis in the ovine fetus: macroscopic changes. American

Caldow, G.L. & Taylor, D.W. (1997) *Experiences with vene-*

Journal of Veterinary Research, **37**, 403–7.

real campylobacter infection in suckler herds. Cattle Prac-Dubey, J.P. (1999) Neosporosis – the first decade of research.

tice, **5**, 327–34 .

International Journal for Parasitology, **29**, 1485–8.

Capen, C.C. (1993) *Hyperplasia of the thyroid gland. In*

Dubey, J.P., Carpenter, J.L., Speer, C.A., Topper, M.J. & Uggla, *Pathology of Domestic Animals* (ed. by K.V.F. Jubb, P.C.

A. (1988) *Newly recognized fatal protozoan disease of dogs.*

Kennedy & N. Palmer), pp. 315–18. Academic Press,

Journal of the American Veterinary Medicine Association, London.

192, 1269–85.

Casaro, A.P.E., Kendrick, J.W. & Kennedy, P.C. (1971)

Dubey, J.P., Lindsay, D.S., Adams, D.V.M., Gay, J.M., Baszler, Response of the bovine fetus to bovine viral diarrhoea-T.V., Blagburn, B.L. & Thullies, P. (1996) Serological

Fetal Loss • 591

responses of cattle and other animals infected with

culture and Resource Management, East Melbourne, No.

Neospora caninum.

American Journal of Veterinary

22.

Research, 57, 329–36.

Hum, S., Stephens, L.R. & Quinn, C. (1991) Diagnosis by

Dubovi, E.J. (1992) Genetic diversity and BVD virus. Com-

ELISA of bovine abortion due to C. fetus. Australian parative Immunology and Microbiology of Infectious

Veterinary Journal, 68, 272–5.

Diseases, 15, 155–62.

Johnson, C.T., Lupson, G.R. & Lawrence, K.E. (1994) The

Duffell, S.J. & Harkness, J.W. (1985) Bovine virus diarrhoea-bovine placentome in bacterial and mycotic abortions.

mucosal disease infection in cattle. Veterinary Record, 117, Veterinary Record, 134, 263–6.

240–5.

Kaashoek, M.J., Moerman, A., Madic, J. Weerdmeester, K.,

Echternkamp, S.E. & Gregory, K.E. (1999) Effects of twinning Maris-Veldhuis, M.A., Rijsewijk, F.A.M. & Van Oirschot,

on postpartum reproductive performance in cattle selected

J.T. (1995) An inactivated vaccine based on a glycoprotein

for twin births. *Journal of Animal Science*, **77**, 48–60.

E-negative strain of bovine herpes virus 1 induces protec-

Ellis, J.A., Martin, K., Norman, G.R. & Haines, D.H. (1995) tive immunity and allows serological differentiation.

Comparison of detection methods for bovine virus diar-

Vaccine, **13**, 342–6.

rhoea. *Journal of Veterinary Diagnostic Investigation*, **7**, Kaashoek, M.J., Straver, P.H., Van Rooij, E.M.A., Quak, J. & 433–6.

Van Oirschot, J.T. (1996) Virulence, immunogenicity and

Ellis, W.A., O'Brien J.J., Neill, S.D., Ferguson, H.W. & Hanna, reactivation of seven bovine herpes virus 1.1 Strains: clini-J. (1982) Bovine leptospirosis: microbiological and serolog-

cal and virological aspects. *Veterinary Record*, **139**, 416–21.

ical findings in aborted fetuses. *Veterinary Record*, **110**, Kirkbride, C.A. (1992a) Viral agents and associated lesions

147–50.

detected in a 10-year study of bovine abortions and still-

Evans, S. & Davies, R.H. (1996) Case control study of

births. *Journal of Veterinary Diagnostic Investigation*, **4**, multiple-resistant

Salmonella typhimurium DT104 infection 374–9.

of cattle in Great Britain. *Veterinary Record*, **139**, 557–

Kirkbride, C.A. (1992b) Etiologic agents detected in a 10-year 8 .

study of bovine abortions and stillbirths. *Journal of Veteri-Forar*, A.L., Gay, J.M. & Hancock, D.D. (1995) The frequency nary Diagnostic Investigation, **4**, 175–80.

of endemic fetal loss in dairy cattle: a review. *Theriogenol-Kirkland*, P.D., McGowan, M.R., Mackintosh, S.G. & Moyle,

ogy, **43**, 989–1000.

A. (1997) Insemination of cattle with semen from a bull

French, N.P., Clancy, D., Davidson, H.C. & Trees, A.J. (1999) transiently infected with pestivirus. *Veterinary Record*, **140**, Mathematical models of *Neospora caninum* infection in

124–7.

dairy cattle: transmission and options for control. *Interna-Knickerbocker*, J.J., Drost, M. & Thatcher, W.W. (1986)

tional *Journal for Parasitology*, **29**, 1691–704.

Endocrine patterns during the initiation of puberty, the

Gee, C.D., Gaden, E.R. & Harper, P.A.W. (1989) An investi-

estrous cycle, pregnancy and parturition in cattle. In *Current gation of the causes of parturient calf mortality in Short-Therapy in Theriogenology* (ed. by D.A. Morrow), pp.

horn heifers. *Australian Veterinary Journal*, **66**, 293–8.

117–25. W.B. Saunders Philadelphia.

Griffiths, P.C., Plater, J.M., Martin, T.C., Hughes, S.L., Hughes, Knudtson, W.U.

& Kirkbride, C.A. (1992) Fungi associated

K.J., Hewinson, R.G. & Dawson, M. (1995) Epizootic

with bovine abortion in the northern plains states (USA).

bovine abortion in a dairy herd: characterization of a

Journal of Veterinary Diagnostic Investigation, **4**, 181–5 .

Chlamydia psittaci isolate and antibody response. British Kvasnicka, W.G., Hall, M., Hanks, D., Ebel, E. & Kearley, B.

Veterinary Journal, **6**, 683–93.

(1996) Current concepts in the control of bovine trichomo-

Hewson, P.I., Lander, K.P. & Gill, K.P.W. (1985) Enzyme-

niasis. *Compendium on Continuing Education, Food*

linked immunoabsorbent assay for antibodies to *C. fetus* in *Animal Medicine and Management (Supplement)* **18**, vaginal mucus. *Research in Veterinary Science*, **38**, 41–5.

5105–112.

Hietala, S.K. & Thurmond, M.C. (1999) Postnatal *Neospora* Lander, K.P. (1983) New technique for collection of vaginal

caninum transmission and transient serologic responses mucus from cattle. *Veterinary Record*, **112**, 570.

in two dairies. *International Journal for Parasitology*, **29**, Low, J.C. & Donachie, W. (1997) A review of *Listeria* mono-1669–76.

cytogenes and listeriosis. *The Veterinary Journal*, **153**, 9–29.

Hult, L. & Lindberg, A. (1998) The Swedish National Control McAllister, M.M., Dubey, J.P., Lindsay, D.S., Jolley, W.R., Wills, scheme on BVD – tips, tricks and traps. In *Proceedings of*

R.A. & McGuire, A.M. (1998) Dogs are definitive hosts of the Australian Association of Cattle Veterinarians, XX World Neospora caninum. International Journal for Parasitology, Buiatrics Congress, Sydney, Vol. 2, pp. 995–1001.

28, 1473–8.

Hum, S., Brunner, J. & Gardiner, B. (1993) Failure of thera-McGowan, M.R. & Kirkland, P.D. (1995) Early reproductive

peutic vaccination of a bull infected with *C. fetus*. Australian loss due to bovine pestivirus infection. British Veterinary Veterinary Journal, 70, 386–7.

Journal, 151, 263–70.

Hum, S., Brunner, J., McInnes, A., Mendoza, G. & Stephens J.

MacLaren, A.P.C. & Agumbah, G.J.O. (1988) Infertility in

(1994) Evaluation of cultural methods and selective media

cattle in South West Scotland caused by an intermediate

for the isolation of *C. fetus* subsp. *venerealis* from cattle.

strain of *C. fetus* subsp. *fetus*. British Veterinary Journal, 144, Australian Veterinary Journal, 71, 184–6.

27–44.

Hum, S. & MacInnes, A. (1987) Australian Standard Diagnos-McNamee, P.T., Trees, A.J., Guy, F., Moffett, D. & Kilpatrick, tic Techniques for Animal Diseases (ed. by L.A. Corner & D. (1996) Diagnosis and prevalence of neosporosis in cattle

T.J. Bagust), CSIRO for the Standing Committee on Agri-

in Northern Ireland. Veterinary Record, 138, 419–20.

MAFF (1989) *Cattle Health Scheme*. ADAS.

Pritchard, D.G., Allsup, T.N., Pennycott, T.W., Palmer, N.M.A., MAFF (1998a) *Veterinary Investigation Surveillance Report*

Woolley, J.C. & Richards, M.S. (1989) *Analysis of risk*

1998 and 1991–1998. *Veterinary Laboratories Agency*,

factors for infection of cattle herds with Leptospira interrogans Weybridge.

gans serovar hardjo. In *Proceedings of the Society for MAFF (1998b) Salmonella in Livestock Production 1998*.

Veterinary Epidemiology and Preventive Medicine (ed. by *Veterinary Laboratories Agency*, Weybridge.

G.J. Rowlands), pp. 130–8.

Mee, J.F. (1992) *Epidemiology of abortion in Irish dairy cattle* Pritchard, G.C. (1992) *Epidemiology of BHV1 infection in*

on six research farms. *Irish Journal of Agriculture and Food cattle breeding herds in Norfolk*. In *Denary Proceedings*,

Research, **31**, 13–21.

Society for Veterinary Epidemiology and Preventive Medi-

Mee, J.F. & Malone, A. (1995) *An outbreak of Salmonella cine*, pp. 168–85. *Edinburgh*.

dublin abortion in outwintered dairy cows. *Irish Veterinary* Pritchard, G.C. (1999) *Bulk milk antibody testing for Lep-Journal*, **48**, 314–15.

tospira hardjo infection. *Cattle Practice*, **7**, 59–61.

Meyling, A. & Mikel-Jensen, A. (1988) *Transmission of bovine Radostits*, O.M., Blood, D.C. & Gay, C.C. (1994) *Veterinary virus diarrhoea virus (BVDV) by artificial insemination*

Medicine, 8th edn, pp. 730–46. Baillière Tindall, London.

with semen from a persistently infected bull. Veterinary

Rhyan, J.C., Blanchard, P.C., Kvasnicka, W.G., Hall, M.R. & Microbiology, 17, 97–105.

Hanks, D. (1995) Tissue-invasive Tritrichomonas fetus in Miller, J.M., Whetstone, C.A., Bello, L., Lawrence, W.C. & four aborted bovine fetuses. Journal of Veterinary Diagnos-Whitbeck, J.C. (1995) Abortion in heifers inoculated with a

tic Investigation, 7, 409–12.

thymidine kinase-negative recombinant of bovine herpes

Rhyan, J.C., Stackhouse, L.L. & Quinn, W.J. (1988) Fetal and virus 1. American Journal of Veterinary Research, 56, 870–4.

placental lesions in bovine abortion due to Tritrichomonas Miller, J.M., Whetstone, C.A. & Van Der Maaten, M.J. (1991) fetus. Veterinary Pathology, 25, 350–5.

Abortifacient property of bovine herpes virus type 1 iso-

Rocha, M.A., Barbosa, E.F., Guedes, R.M., Lage, A.P., Leite,

lates that represent three subtypes determined by restric-

R.C. & Gouveia, A.M. (1999) Detection of BHV-1 in a

tion endonuclease analysis of viral DNA. Americal Journal

naturally infected bovine fetus by a nested PCR assay.

of Veterinary Research, 52, 458–61.

Veterinary Research Communications, 23, 133–41.

Miller, R.B. (1988) Reproductive failure in domestic animals:

Roche, J.F., Boland, M.P. & McGeady, T.A. (1981) *Reproductive wastage following artificial insemination in cattle. A discussion on the pathogenesis. In 11th International Conference on Animal Reproduction and Artificial Insemination, Veterinary Record, 109, 95–7.*

Dublin, pp. 284–92.

Sanderson, M.W. & Chenoweth, P.J. (1999) *The role of Ure-MLC (1998) Making Money Out Of Beef: A Way Forward For*

aplasma diversum in bovine reproduction. Compendium on Beef Producers. Meat and Livestock Commission, Winter

Continuing Education for the Practicing Veterinarian, 21, Hill, Milton Keynes. S98–102.

Nettleton, P.F. & Entrican, G. (1995) *Ruminant pestiviruses.*

Schurig, G.C., Hall, C.E., Burda, K., Corbeil, L.B., Duncan, J.R.

British Veterinary Journal, 151, 615–42.

& Winter, A.J. (1973) *Persistent genital tract infection with Nix, J.M., Spitzer, J.C., Grimes, L.W., Burns, G.L. & Plyler, B.B.*

Vibrio fetus fetus associated with serotype alteration of the (1998) A retrospective analysis of factors contributing to

infecting strain. American Journal of Veterinary Research, calf mortality and dystokia in beef cattle. Theriogenology, 34, 1399–403.

49, 1515–23.

Semambo, D.K.N., Ayliffe, T.R., Boyd, J.S. & Taylor, D.J. (1991) *Noakes, D.E. (1986) Fertility and Obstetrics in Cattle (ed. by Early abortion in cattle induced by experimental intrauterine infection. E.A. Chandler, P.G.C. Bedford & J.B. Sutton), pp. 65–9.*

ine infection with pure cultures of *Actinomyces pyogenes*.

Blackwell Scientific Publications, Oxford.

Veterinary Record, **129**, 12–16.

Norton, J.H. & Campbell, R.S.F. (1990) Non-infectious causes Smith, C.R., Kettereer, P.J., McGowan, M.R. & Corney, B.G.

of bovine abortion. *Veterinary Bulletin*, **60**, 1137–47.

(1994) A review of laboratory techniques and their use in

Otter, A., Jeffrey, M., Scholes, S.F.E., Helmick, B., Wilesmith, the diagnosis of *Leptospira interrogans* serovar hardjo infec-J.W. & Trees, A.J. (1997) Comparison of histology with

tion in cattle. *Australian Veterinary Journal*, **71**, 290–4.

maternal and fetal serology for the diagnosis of abortion

Sreenan, J.M. & Diskin, M.G. (1986) The extent and timing of due to bovine neosporosis. *Veterinary Record*, **141**, 487–9.

embryonic mortality in cattle. In *Embryonic Mortality in*

Paiba, G.A., Green, L.E., Lloyd, G., Patel, D. & Morgan, K.L.

Farm Animals (ed. by J.M. Sreenan & M.G. Diskin), pp.

(1999) Prevalence of antibodies to *Coxiella burnetii* (Q

142–58. Martinus, Nijhoff, CEC.

fever) in bulk tank milk in England and Wales. *Veterinary*

Thilsted, J.P. & Dubey, J.P. (1989) Neosporosis-like abortions *Record*, **144**, 519–22.

in a herd of dairy cattle. *Journal of Veterinary Diagnostic* Paton, D.J., Christiansen, K.H., Alenius, S., Cranwell, M.P.,

Investigation, **1**, 205–209.

Pritchard, G.C. & Drew, T.J. (1998) Prevalence of antibody

Thornton, R. (1991) Bovine abortions – laboratory diagnoses

ies to bovine virus diarrhoea virus and other viruses in bulk

1991. *Surveillance*, **19**, 24.

tank milk in England and Wales. *Veterinary Record*, **142**, Thurmond, M.C. &

Hietala, S.K. (1997) Effect of congenitally 385–91.

acquired *Neospora caninum* infection on risk of abortion Paton, D.J., Goodey, R., Brockman, S. & Wood, L. (1989) Evaluation and subsequent abortions in dairy cattle. *American Journal of Veterinary Research*, **50**, 1381–5.

of *Neospora caninum*-induced endemic and

bulls actively infected with BVD. *Veterinary Record*, **124**, Thurmond, M.C.,

Hietala, S.K. & Blanchard P.C. (1997) Herd-63–4.

based diagnosis of *Neospora caninum*-induced endemic and

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epidemic abortion in cows and evidence for congenital and

and infection and the immunofluorescent localization of the

postnatal transmission. *Journal of Veterinary Diagnostic*

organism in infected heifers. *Canadian Journal of Com-*

Investigation, **9**, 44–9.

Comparative Medicine, **35**, 301–12.

Thurmond, M.C., Hietala, S.K. & Blanchard, P.C. (1999) Pre-Wouda, W.,

Bartels, C.J.M. & Moen, A.R. (1999) Characteristic values of fetal

histopathology and immunopero-

tics of *Neospora caninum*-associated abortion storms in xidase staining in diagnosing bovine abortion caused by

dairy herds in the Netherlands (1995 to 1997). *Theri-*

Neospora caninum in a dairy herd. *Journal of Veterinary ogenology*, **52**, 233–45.

Diagnostic Investigation, **11**, 90–4.

Wray, C., Wadsworth, Q.C., Richards, D.W. & Morgan,

Wendorf, G.L., Lawyer, M.S. & First, N.L. (1983) Role of the J.H. (1989) A three-year study of *Salmonella dublin*

adrenals in the maintenance of pregnancy in cows. *Journal*

infection in a closed dairy herd. *Veterinary Record*, **124**, of Reproduction and Fertility, **68**, 281–7.

532–5.

Wilkie, B.N. & Winter, A.J. (1971) Bovine vibriosis: the distri-Yule, A., Skirrow, S.Z. & BonDurant, R.H. (1989) Bovine

bution and specificity of antibodies induced by vaccination

trichomoniasis. *Parasitology Today*, **5**, 373–7.

Chapter 38

Bull Infertility

D.N. Logue and W.M. Crawshaw

Introduction

594

cial insemination (AI) has proved to be one of the most

Bull use in the UK

595

successful reproductive technologies and it became a

The role of the veterinarian in bull use

596

commercial reality in the UK just before the end of the

Preventing infectious infertility and other diseases

596

Second World War. AI has fulfilled two objectives:

Preputial washing

597

firstly, it set in motion more focused genetic improve-

Infectious disease control

598

ment coupled with the widespread use of good bulls and

Investigation of bull infertility

598

secondly, it has limited the spread of venereal diseases.

History

598

Examination of the bull for breeding soundness

599

Unfortunately, the expression of oestrus in the cow

Manifestations, aetiology, treatment and prognosis of bull

varies in time of occurrence, duration and intensity and

infertility

610

many farmers find accurate and, more particularly, effi-

Failure to mount

610

cient oestrous detection difficult and time consuming.

Failure to achieve intromission

612

Furthermore, particularly in large beef suckler herds

Failure to thrust and ejaculate after intromission

616

under essentially range conditions, oestrous detection

Normal service with a poor pregnancy rate

617

and the handling of cattle for AI are less practical.

Conclusion

624

These factors ensure that many farmers continue to use natural service entirely or partially even though, due to the genetic improvement from AI, dairy farmers in

Introduction

particular could afford to have a longer calving interval and yet still expect to have the same financial returns Since the domestication of cattle the bull has been a (Hillers et al. , 1982; Stott et al., 1999).

talisman and the desire to own a special bull a parti- The problem is illustrated by a study of UK national cular urge in all stock rearing countries. The bull is a cattle population statistics where only approximately symbol of power in the East, where Indra the sky deity half the cows are bred by AI (Logue & Isbister, 1992), is represented in myth as a mighty bull, while in the this figure being considerably higher for the dairy herd. West as an example there is the Celtic legend of the Although confidentiality has stopped many of these sta- Brown Bull of Quelgny stolen by Queen Mae of tistics being easily available in the UK we have no Connacht from Dara in Ulster only to be retrieved by

reason to believe the proportion has changed.

the hero Cuchulain. Time has not diverted man a great

*However, this figure may increase following the 2001
deal from this interest in bull lineage and the perform-
foot and mouth disease outbreak. In the USA the pro-
ance of his offspring! The development of methodolo-
portion bred by AI is probably a little higher (70 per
gies to assess the transmission of superior economic
cent). In addition, there have been a number of factors
traits from bulls to their progeny over the last 50 years
that have counteracted the undoubted improvements in
has been one of the success stories of the cattle indus-
the efficiency of AI.*

*try. However, like the Brown Bull the truly outstanding
sire is still very rare and the purchase of a young*

(1)

*The economics of dairy and beef production have
unproven bull is a gamble. Despite this the repeated
resulted in a general trend to larger herds with fewer
need for young bulls (no bull is immortal) means that
staff and so less time available for oestrous detection.*

*many are bought with this vision in mind, despite the
Furthermore, the continuing financial pressure to cut
difficulties in achieving it.*

costs has also made many dairy farmers economize by

When all breeding was by natural service the

changing from paying for a technician AI service to

potential for genetic improvement by a good bull was

inseminating their own cows (DIY AI). Many of these

very limited. Furthermore, selling, sharing and hiring

people keep a bull as a precaution against poor fertil-

animals resulted in the spread of venereal disease with

ity arising from a doubtful insemination technique.

some disastrous results for those affected farms. Artifi-

Unfortunately, no figures are produced for the number

594

Bull Infertility • 595

Table 38.1

Number of cows in DIY AI herds (Anon. 1988).

England and

Northern

Scotland

Total

Wales

Ireland

DIY licences

3 300

1 000

700

5 000

Average herd size

69

37

89

—

Number of cows

227 700

37 000

54 300

319 000

Cows inseminated (assuming

170 775

27 750

40 725

239 250

75%) by DIY AI

Table 38.2

UK dairy cattle statistics (Anon. 1979, 1988, 1998).

increasing improvement in the health status of the

national herd in many countries there is less pressure

Year

Cow numbers ¥ 106

Herd size

for AI use for this reason alone. However, this confi-

dence is likely to be misplaced unless the farm takes

1960

4.01

20

adequate precautions when acquiring bulls.

1970

4.55

31

1980

4.69

55

1990

4.43

67

1997

4.41

72

Bull use in the UK

It is worthwhile defining the various types of herd breeding policy since this will influence the management of the bull and may give a guide to the needs of of cows inseminated in the DIY AI sector in the UK. the unit with regard to disease control.

However, census data, the number of DIY AI licences

Four basic types of policy can be recognized:

issued by area and the assumption that 75 per cent of the cows in each herd are inseminated does allow an

(1)

Beef suckler herd. The principal demand is to estimate (Table 38.1). It can be seen that DIY AI prob- produce calves for finishing. The cows are mated while

ably accounts for less than one in five of all dairy cow suckling. For many years and in most geographical localities it has been axiomatic that this system operates at

(2)

The gradual increase in herd size has meant that highest efficiency when there is a short (but efficient) breeding period (Kilkenny, 1978). Ideally this should be the added costs of keeping one or even several bulls are relatively lower per unit of production. For example, in less than 10 weeks and demands very high levels of fertility in both bull and cow.

the last 30 years (see Table 38.2), and this has been mirrored in Europe and in virtually all countries with an

(2)

Dairy heifers. A considerable proportion of dairy advanced dairy industry.

herds use natural service when breeding heifers. This is mainly to avoid oestrous detection and the need to

(3)

*A further effect of the larger number of cows per
gather the individual heifer for AI when often these are
herd has led to a more predictable production of heifer
run on a more remote area of the farm.*

*replacements because the male : female ratio remains
nearer to the expected 50 : 50 as the numbers increase.*

(3)

Sweeper bull. This term usually refers to the

*This has meant that a definite block of heifers of similar
running of a bull after a fixed period of 'AI-only' breed-
age can be released for natural service.*

*ing. Generally, in dairy cattle, the bull used is a beef
breed and thus any calves born are easily distinguished*

(4)

*In many beef and dairy herds, calving, and thus
from purebred dairy calves. The use of synchronization
breeding periods, are concentrated into relatively short
and fixed-time AI with batches of maiden heifers using
periods of time for production and management
an easy-calving sire has to some degree replaced
reasons. This leads to a requirement for high oestrous*

natural service in both dairy and beef heifers, but in detection rates and high fertility, both of which are best in many herds those heifers that return are served by a bull. This is achieved by using a combination of AI and natural service with a sweeper bull. The timing of the introduction of the sweeper is paramount since mating pressure will

(5)

Finally, in the past an important reason for using AI was to reduce the introduction of disease. With the obvious drop in the pregnancy rates achieved with AI can result in a significant increase in mating pressure. Clearly a

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drop in the pregnancy rates achieved with AI can result in a significant increase in mating pressure. Clearly a

cantly on efficiency.

The most important source of infectious disease for
(4)

Modified sweeper. The sweeper bull is utilized on cattle is other livestock, particularly cattle. Uncon-

for mating selected cows throughout the breeding
trolled movement of a bull into a herd carries a signifi-
season, for example cows of low genetic potential,
cant risk of introducing disease. This can be minimized
repeat breeders, cows not exhibiting firm signs
by the following measures but presupposes that other
of oestrus and cows with a history of being difficult to
biosecurity precautions are in place:
get in calf. Subsequently, the bull is used as a 'true'
sweeper.

(1)

Bulls (and any other cattle) should be purchased
Finally, a minority of dairy herds still primarily use
only from herds certified free from specific diseases of
natural service.

concern by reputable cattle health schemes such as
those registered with the Cattle Health Certification
Standards (CheCS) body in the UK. While individual

The role of the veterinarian in

farm programmes can be undertaken or declarations

bull use

made by the farmer and his veterinarian to the effect that a particular disease has not been seen, clearly these will have less certainty. The latter point is illustrated by

The role of the veterinarian in the control and moni-

Kallis et al. (1999) who revealed by faecal culture that toring of bull fertility is three-fold (see Box 38.1).

40 per cent of 100 Dutch dairy herds considered to be

Veterinarians should be authoritative in these roles

free of Johne's disease on the basis of owner and vet-

for their clients or be able and willing to refer matters

erinary declaration were actually infected.

to another veterinarian with the appropriate resources

if the occasion demands.

(2)

Quarantine incoming cattle before entry into the

main herd to allow testing for diseases that may be

carried and development of any incubating diseases. In

Preventing infectious infertility and

most cases four weeks should suffice, but this depends

other diseases

on circumstances and the diseases under consideration.

Quarantine could also be applied to other species of
Before considering the reproductive performance of
livestock entering the farm that potentially carry dis-
the bull on the farm the first priority must be to keep
eases infectious to cattle.

disease out! No veterinarian or farmer should be
without some appreciation of the risks that are being
(3)

During the quarantine period an adequate testing
taken by the purchase of a bull (or even semen or
programme and/or prophylactic treatment against dis-
embryos). In addition to the notifiable diseases (such as
eases of concern should be employed. The latter may
foot-and-mouth disease) there are a whole plethora of
be necessary where testing is unreliable or gives an
others that can cause economic loss. Unfortunately in
equivocal answer (e.g. for venereal campylobacteriosis
the past, farmers in the UK, and for that matter else-
and Leptospira hardjo).

where, have been pretty cavalier in their attitude to
introducing disease. Veterinarians, by discussing these

The diseases of importance vary with country and issues with their clients, can do much to reduce the risks area and are determined by the economic or practical of introducing disease and so protect the viability of importance of the disease, an assessment of the risk of their clients' farms. The loss of output arising from the introducing the disease, the availability of low cost and reliable tests and whether or not the disease is already present in the herd. In the UK the following non-

Box 38.1

Role of the veterinarian in bull fertility.

statutory diseases are suggested as being worthy of

(1)

Preventing the introduction of infectious infertility and

consideration based on their prevalence and the

other diseases to the farm

economic impact that they may have on the herd.

(2)

Assessing fertility and the level of investigation that is

needed:

Bovine virus diarrhoea virus (BVDV)

(a)

before purchase

Infectious bovine rhinotracheitis virus (IBRV) (bovine

(b)

after purchase

herpes virus 1 [BHV-1])

(c)

before use each breeding season

Leptospira hardjo

(d)

after poor fertility results

Johne's disease (paratuberculosis)

(3)

Monitoring herd fertility (including the bull) by the

Venereal campylobacteriosis

assessment of breeding records

Salmonellosis (especially S. dublin)

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Whilst neosporosis is a significant cause of bovine

enrichment medium. Negative laboratory results in the

reproductive failure any epidemiological role of the

face of a strong history of infertility and natural service bull is undetermined and the reliability of current laboratory tests for detecting carriers remains question-suspicion.

able. However, the precautionary principle suggests it is unwise to acquire bulls bred in herds infected with

Preputial washing

Neospora caninum at least until more is known about this infection. Outside and unfortunately even within

The method of preputial washing used routinely by the the UK, diseases considered notifiable in the UK

Scottish Agricultural College is summarized below.

such as brucellosis and tuberculosis also need to be considered.

Protocol for sampling bulls for Campylobacter

The two most important true venereal diseases are

fetus sp. screen

trichomoniasis and *campylobacteriosis*, although there is evidence for the venereal transmission of some

Materials required:

Ureaplasma sp., *Mycoplasma bovis*, *BHV-1*,

- Recovery fluid: 60ml phosphate buffered saline

brucellosis and even *Haemophilus somnus*. Bovine (PBS) (sterile).

trichomoniasis is a venereal disease characterized by

- Sampling catheter: 10cm length of 3.5 ¥ 6.5mm

early to mid-gestation pregnancy losses, reduced portex tubing (sterile).

calving rates and pyometra; it is caused by the flagel-

- 60ml syringe: attached to catheter (sterile).

lated protozoan *Tritrichomonas fetus*. As far as is

- 3 ¥ 20ml sterile sample bottles.

known, it is transmitted by venereal contact only.

- Individual set of sampling materials for each animal
- Campylobacteriosis*, which also presents as a large in-sampled.

crease in returns to service with early to mid-gestation pregnancy losses, is classically caused by *C. fetus*

Procedure:

venerealis. However, there is evidence that this organ-

- Clip preputial hairs.

ism does not always follow the classic laboratory iden-

- Wash preputial orifice with warm water. Do not
tification procedures and this has led to the proposition
disinfect.

of *C. fetus intermedius*, which also appears to cause

- Load syringe with 60ml warmed PBS.

the same clinical problems in cattle. This organism has

- Insert catheter as far into prepuce as possible.

many of the properties of *C. fetus fetus*. In these two Clamp one hand over
preputial orifice to seal

venereal infections the bull is the long-term carrier

around catheter and hold it in place (use hand

of the organism, apparently without ill effects (i.e. no

nearest bull's head to facilitate massage with the

visible lesions are evident in recently or chronically

other hand).

infected bulls) and a distinct correlation exists between

- Pass 60ml warmed PBS through catheter. Leave
age and infection, with older bulls being far more likely
syringe in place.

to sustain an infection than younger bulls. Infection

- Retain fluid and catheter in prepuce by continuing of the female occurs at the time of coitus. Diagnosis of to clamp one hand over preputial orifice and mas-campylobacteriosis in the female is by examination of sage thoroughly along full length of prepuce to scro-vaginal mucus, collected by the method of Lander tum for one minute.

(1983), for antibody by the vaginal mucus agglutination

- Collect fluid into the original syringe by withdrawal test (VMAT) and for the bacterium by culture. Since using the plunger as the catheter is slowly antibody takes some time to appear after an infected withdrawn.

service and false negative VMAT results are common,

- Dispense sample into sterile containers and label.

at least 12 cows should be sampled at least 50 days after

- If the bull urinates during collection the sample a potentially infected service. Cows with blood in the should be discarded and the process repeated.

mucus can give false positive results and cows in oestrus

- All samples should be submitted to the laboratory

are more likely to give false negative results and such within 6 hours of collection.

animals should not be sampled. In the bull, demonstra-

tion of the *Campylobacter fetus* bacterium in preputial With preputial washings false negative results are

washings by fluorescent antibody test and culture is

again to be expected on a single screening, indeed four

used in the UK for diagnosis. Where samples of vaginal screenings have been recommended (Dufty, 1967)

mucus or preputial washings are collected with a view

before a bull can be confidently declared free of infec-

to culture they should reach the laboratory within six

tion. A more practical approach to reducing the risk of hours of collection due to the fragility of the bacterium.

introducing this infection may therefore be prophylac-

Alternatively, samples can be submitted in transport

tic treatment of purchased, previously bred bulls by

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preputial lavage with penicillin and streptomycin

(BVDV, IBRV and Johne's disease in the UK). How-

antibiotic (Melrose et al. , 1957) during the quarantine ever, as some may remember, lack of appreciation of

period. This could be refined by employing prophylactic the organism and its epidemiology meant that a pilot tic treatment after a single negative preputial wash attempt to control *L. hardjo* by MAFF was not successful. Nevertheless, the most reliable way to prevent introduction of this infection is to acquire only applied to these new schemes.

unbred males and females. Control in infected herds Another consideration is whether the bull might is by disposal of infected bulls, suspension of natural itself be at risk of being infected by cattle in the herd it service and use of AI with uninfected semen for 2 years is being introduced to and so having a period of infertility (Clark, 1971) for all exposed cows, thus allowing the tility as a result of illness. Completion of a vaccination cows to develop immunity and eradicate the organism. programme during the quarantine period for the bull Where this is not practicable, use of autogenous vaccine might be considered in light of knowledge of the disease (there is no licensed vaccine in the UK), only using

status of the herd.

young bulls (<4 years) or operation of 'clean' and 'dirty'

herds should be considered. The problems of diagnosis

and control of venereal campylobacteriosis should not

Investigation of bull infertility

be underestimated and the reader is referred to Caldow

and Taylor (1997) for a more detailed discussion of the

Bull infertility is presented as one, or a combination of

subject in suckler herds.

the four manifestations, namely:

(1)

Failure to mount

Infectious disease control

(2)

Failure to achieve intromission

(3)

Failure to thrust and ejaculate

Control of infectious disease is very dependent on an

(4)

Poor pregnancy rate with normal service

assessment of the risk of introducing the disease and

behaviour

on the quality of diagnosis, either of the disease or the carrier state, using laboratory tests. For example, in the UK bulls are not usually tested for trichomoniasis as it only are the aetiological factors varied and numerous has not been diagnosed in home bred animals for many years. However, other diseases such as BVDV are very common and campylobacteriosis is not usual. In history of the bull (including previous fertility) and the first instance the background of the bull must be his present circumstances can one gain an informed appraisal of the most appropriate way in which to exposed and, as a consequence, be a potential carrier conduct the assessment of fertility.

of any disease. Furthermore, when subsequent testing Diagnosis is based upon a combination of bull and is contemplated the limitations of laboratory testing

herd history, examination of the bull and the results regimes specific for each disease must be appreciated of the various tests that are undertaken including an before exclusion is considered. For example, testing in-examination of a representative ejaculate of semen. individual healthy animals for antibody and/or by faecal Even if the diagnosis is tentative and the aetiology culture for the presence of infection with the bacterium uncertain the findings will probably allow a prognosis of Johne's disease leads to a high proportion of false and may give a rationale for treatment.

negative results. Therefore, exclusion of this disease is best achieved by purchase of bulls from herds moni-

History

tored free of Johne's disease under a reputable cattle health scheme rather than by individual animal testing All investigations of bull fertility are built upon the and quarantine. Knowledge of the effects of disease, usual prerequisite of obtaining a good history of the methods of control and the laboratory technology to problem. This clearly involves both records of any bull

detect disease is continually advancing. Understanding in question but should also entail assessment of the and awareness of BVDV in particular have improved herd as a whole. Usually the client will have given some greatly over the last decade. The infection is widespread information about the bull by telephone and, ideally, in the UK and recent estimates of associated losses, will have presented some farm records for perusal prior which primarily arise from reduced fertility and abortion to the examination. At the outset, ask and record the tion, suggest that these can be over £45 per cow answers to the questions in Box 38.2. One is looking for annually in an infected suckler herd (Gunn et al. , 1998). an estimate of the extent of the infertility by comparison to other bulls, when it was first manifest, if it has have become a realistic option for certain diseases been continuous or varied and so on.

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Box 38.2

Information required to define the problem.

less are more likely to be infertile or have a lower than 'normal' fertility.

Type and breed of bull?

(3)

If the cause of the problem is not clear from

Age?

(1) and (2) above and/or the bull is under a sale

Demeanour?

Ringed?

agreement or insured for infertility then it is

Type of herd?

necessary to consider a full breeding sound-

Role of bull in herd?

ness examination. However this is not to be

When and where acquired and time with herd?

undertaken lightly and firstly we recommend

Calves previously born to the bull?

consideration of:

Extent and duration of problem?

(a)

Sale agreement: if the time period for a claim

More than one bull with a problem?

has not elapsed ensure the farmer has

Can the problem be related to introduction of a new male or

contacted the market or vendor, informed

female?

them of the complaint and checked that a

Are AI figures available for the same period?

full breeding soundness examination will

Is there an insurance/sale agreement status?

support the claim.

(b)

Insurance case: ensure that the farmer has

contacted the insurance company, informed

In cases where points (1) to (3) above apply it may

them of the complaint and checked that the

be possible to give a firm diagnosis and an accurate

policy covers infertility and that a full breed-

prognosis based on the history and visual examination

ing soundness examination will support the

of the bull when presented with a cow in season, i.e.

claim.

without even collecting and examining a semen sample.

However, depending on circumstances it may still be

The goal is to obtain a cost-effective resolution to

advisable at least to attempt to collect a semen sample,

the problem. It may be very attractive to examine

even if only to confirm the history.

the semen of the bull but is it really necessary? For

example, if there is a clear history of infertility and

Examination of the bull for

severe testicular abnormalities are found on physical

breeding soundness

examination of a commercial beef bull, then clearly one

is not going to recommend that the bull is used as a reli-

The objectives of the visit to the farm are:

able sire. Collecting and examining semen will be of

- To see the conditions under which the bull is ex-*

little additional value to the client unless demanded by

pected to work.

the insurance company. Once it is clear that a full

- To confirm that the history is reliable and, if possi-*

breeding soundness examination is required a decision

ble, to examine records.

as to who will carry this out should be made. Referral

- To examine the bull. In order to study semen for within the practice or elsewhere may be needed to abnormal characteristics the operator must use a access the necessary expertise and equipment.*

technique to obtain and prepare semen for exami-

The order of events for the full breeding soundness nation that will be representative of the ejaculate of examination should be:

the bull in natural service.

- To ascertain the status of the bull as a possible*

(1)

Define the problem.

vector of reproductive disease and possibly other

(2)

Prepare for the visit.

diseases of a less specific nature but just as impor-

(3)

Set up the on-farm laboratory.

tant from an economic point of view.

(4)

Assess the collection area and ability and reliability of the farmer and other animal handlers.

Where an infertile bull is suspected the following

(5)

Introductory examination of the bull.

simple preliminary breeding soundness examination

(6)

Semen collection and assessment.

steps are worth considering before conducting a full

(7)

Observation of natural service.

breeding soundness examination which includes semen

(8)

Full physical examination of the bull.

collection.

(9)

Collection of other diagnostic samples.

(1)

Ascertain by personal observation or a reliable

(10)

*Preliminary diagnosis, prognosis and advice (e.g. report from the farmer whether or not the bull is treatment or sexual rest).
serving normally.*

(11)

Final diagnosis, prognosis and advice after all test

(2)

Physically examine the bull for any obvious results are known.

genital or other abnormalities. Bulls aged 2 years

(12)

*Further action for the herd and its future breed-
or more with a scrotal circumference of 30 cm or
ing management.*

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Define the problem

restraint and facilities. For experienced beef

bulls it is definitely a case of no cow in oestrus, no

*This is often less easy than might be assumed and is
collection!*

based on the answers to the questions in Box 38.2 and

(4)

There should be suitable facilities for restraint and on the information derived from the preliminary breeding examination of the bull, e.g. crush with removable sides to allow access to the penis and prepuce.

On occasion the history is vague and the farmer is uneasy

(5)

A suitable roomy fenced area with a non-slip surface and with an escape route for the collector should be available to allow collection of semen.

Careful and sensitive questioning may be needed to avoid missing some obvious point. In most cases

This can be inside or outside.

where a full breeding soundness examination is being

(6)

Instruct the bull handler or those helping in the carried out the manifestation will be poor pregnancy sequence of events, especially in relation to the rate with normal service behaviour. However, some

bull mounting for collection.

remain undefined beyond infertility seen as 'too many

(7)

Any cows that have been running solely with this

returns' until semen collection is attempted.

bull should be nearby for pregnancy diagnosis

or sampling (vaginal mucus or blood) should the

Prepare for the visit

occasion demand it. Unfortunately, often another

bull will have remated these. However, even then

A full breeding soundness examination can go

examination may allow confirmation that the cows

smoothly and efficiently and be completed in a couple

have conceived to the second bull.

of hours or alternatively take most of the day and not

(8)

If possible, another bull should be available near-

achieve a collection. Furthermore, it is potentially

by. This can be useful to confirm the teaser

dangerous. Success, efficiency and safety all depend

on good organization and attention to detail. Generally

speaking a convenient date will be set for the examination and all parties involved (man and beast) need to

Box 38.3

Check list for breeding soundness kit.

be organized accordingly. The following factors, if they

- *Artificial vaginas, liners, cones, cord and adhesive tape can be arranged, should help to make the visit go more*

- *Semen collection tubes (preferably graduated) smoothly and efficiently:*

- *Lubricant for artificial vaginas (e.g. KY jelly)*

(1)

For a full clinical examination and for semen col-

- *Kettle and jug to fill artificial vaginas with warm water*

lection it is desirable that the bull be restrained,

- *Thermometers to measure artificial vagina temperature*

that he can be led on a halter and, ideally, that he

- *Electro-ejaculator(s)*

- *Two funnels and wire funnel holder for collection of*

should be used to serving while being restrained

electro-ejaculated semen

by a halter or ring-rope. The reasons for this are

- *Electric plug adapters and circuit breakers*

obvious but in our experience with fewer staff on

- *Electric extension cable*

farms, bulls are much less used to being handled

- *Microscope and light source*

and therefore restraint is often counterproductive.

- *Warm microscope stage*

This leads to difficulties in reconciling the safety

- *Heated cabinet*

of the collector and handlers with the desire to

- *Transformer packs*

obtain a good semen sample.

- *Stains: nigrosin–eosin, Giemsa and methylene blue*

(2)

The bull should be sexually rested for at least

- *Small test tubes and holder for use in staining and*

one but preferably three weeks prior to the visit.

diluting semen for examination

This will enhance his libido and avoid depletion

- *2.9% sodium citrate, 0.9% NaCl and phosphate buffered*

saline

of semen reserves, ensuring the best chance of

- *Glass pipettes and rubber bulbs*

obtaining a representative sample of semen.

- *Slides, coverslips and slide holder box plus slide labels*

(3)

A quiet cow in oestrus, preferably halter-trained

- *Small sterile bottles for fresh semen samples*

and that has not been served at that oestrus,

- *Immersion oil*

should be available as a teaser. Synchronization

- *Paper wipes, cotton wool and swabs*

with a progesterone releasing intravaginal device

- *50ml syringe with tubing, phosphate buffered saline and*

of at least six females, in our experience, maxi-

sterile containers for preputial wash

mizes the chances of one or preferably more being

- *Tape measure (for scrotal circumference)*

available on the allotted date. While keen young

- *Vacutainers, needle holder, needles and spirit*

dairy bulls will usually mount a quiet non-oestrus

- *Virus and Mycoplasma transport media*

cow, our experience is that collection can still

- *Record sheets, slide markers and pens*
- *Spare light bulb for microscope*

prove difficult, particularly with inadequate



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Fig. 38.1

The on-farm laboratory.

cow is in oestrus when the bull under test has

preferable to have a small portable cabinet specifically designed for this type of activity.

cally designed for this type of activity.

(9)

Use a checklist of equipment and materials (see

- *Stains. It is well worthwhile using more than one*

Box 38.3).

stain routinely. The most commonly used are nigrosin–eosin and Indian ink for sperm morphology, methylene blue for examining the semen for

Set up the on-farm laboratory

extraneous cells and Giemsa for a combination of the two. Nigrosin–eosin and Indian ink are used

This should be set up on arrival so that there is time for directly, i.e. after preheating to 37°C they are mixed

the heated cabinet, solutions, glassware and warm stage with the semen immediately after its collection and

to reach the required temperature. Select a small area

a smear is then made. The second stains are used on

of bench space, which is covered, has an electricity

a direct smear of the semen, which is air-dried and

supply and is not exposed to adverse weather (Fig.

then fixed before staining.

38.1). It should be as near the area of semen collection

- Solutions to dilute semen for examination of pro-

as possible. Given the standard of some farm electrics

gressive motility. A normal saline solution may suf-

a circuit breaker is a wise investment.

fice, although solutions commonly used for diluting

The on-farm laboratory should contain the following:

semen are phosphate-buffered saline or 2.9 per cent

- *Microscope with facilities to examine semen by ¥50*

trisodium citrate. These solutions should also be

to ¥100 (for initial motility) by ¥200 to ¥400 (for

preheated to as near 37°C as possible.

progressive motility) and ¥1000 (for morphology).

- *Artificial vagina (AV). In general, inexperienced*

- *Warm stage (or equivalent). This is a prerequisite*

bulls prefer rough liners and younger bulls a shorter

for the reliable estimation of motility. Ideally, it

artificial vagina, i.e. about 25 cm long. At least two

should be kept within 1°C of 37°C because either

should be assembled (Fig. 38.2) and be filled with

a high or low temperature can have considerable

warm water (prepared in an electric kettle for

artefactual effects upon sperm motility. Unfortu-

example) to a lumen temperature of about 60°C

nately, this equipment is quite expensive and there-

(they will often have cooled to around 45–48°C before many veterinarians use a small prewarmed the time they will be used). The lumen temperature flat bottle or brass plate as an alternative. If this should be between 42 and 50°C immediately prior approach is used then it is best to have two so that to semen collection and this must be checked with they can be alternated and so kept at as steady a a thermometer. In practice it is best to enter the col- temperature as possible.

lection area with the AV at 50°C since there will

- *Heated cabinet. While it is possible to use the farm inevitably be some cooling before a collection is kitchen oven to keep slides, coverslips, pipettes and made. A sterile lubricant should be applied to the stains, regulation of a reasonable temperature range opening of the AV immediately before going to the (35–40°C) is often difficult. For this reason it is collection area.*



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Fig. 38.3

On-farm semen collection in a straw yard using an artificial vagina (AV). The bull is searching for the vulva of the teaser cow. The collector is diverting the penis towards the AV.

surface such as grass or a firm straw bed rather than on

concrete (Fig. 38.3). However, in most circumstances the latter is the preferred surface since it generally gives a sound secure footing. The bull falling causes most injuries to collectors. It is for this reason that most AI centres use service stocks and obviously if these are available they should be used. However, whatever

Fig. 38.2

Assembly of the AV. A 30 cm long rubber AV cylinder with latex liner and cone favoured by the authors for collection adaptation or compromise is used, the collector must from adult bulls. A valve on the filling aperture conveniently remember that his safety and that of the others involved permits air to be blown in to finely adjust the internal pressure.

is paramount. The main prerequisites are common

The graduated collecting tube is anchored to the cone by string sense, plenty of room and a safe footing. Listen care-and is protected thermally and physically by enclosure in a clear fully to all remarks about the demeanour of the bull.

plastic container secured with adhesive tape. A small (1–2 cm) Be aware that sometimes farmers may be carried away

slit can be made in the cone near the AV to act as a safety valve.

by the novelty of this examination. Try to discourage

This reduces the risk of disconnection of the cone or collecting bystanders!

tube that may occur during the thrust if there is too high a pressure in the AV. The AV cylinder is disinfected between bulls and the latex can be sterilized by

boiling.

Introductory examination of the bull

If practical it is worthwhile giving the bull an initial cursory examination prior to collection, gently running one's hands over the bull, touching the prepuce and genitalia. This allows the bull to get used to the personnel involved, particularly the collector of semen, and of the farmer and other animal handlers also allows the collector to judge what size and pressure of artificial vagina might be best and how the bull is likely to react to collection. Remember frightened bulls the collection to take place. Remember that while he or she may not understand the needs of collection well they do know the bull much better than you do! Some bulls can be totally inhibited in the wrong surroundings.

Semen collection and assessment of ejaculate

Ideally, one should work in the place where the bull normally serves the cows. A good surface will encour-

The libido of the bull and the likelihood of a successful age a bull to mount, for example a bull with laminitis semen collection can be enhanced by allowing him to or an old bull will often work better on a relatively soft have sight and sound of one or more oestrous cows

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without physical contact for a short while before col-prepuce guide the erect penis towards the opening of lection is attempted. It is important to interfere as little the artificial vagina held in the other hand. Touching the as possible with the normal service behaviour of the penis directly will generally cause the bull to dismount. bull to obtain a representative ejaculate. The best tech-The bull will vigorously seek the vulva by rapid back-nique for the collection of semen is therefore the use of ward and forward movement of the penis. The tempta-an artificial vagina. The teaser cow should be restrained tion to force the AV over the penis at this point should in the collection area. The bull is held separately, but be avoided as this can reduce the quality of the service. nearby, from where he can be introduced for the col-

The bull should mount, clasp the cow, erect fully and lection and returned immediately afterwards to avoid search for the vulva with the penis, and, on finding the repeated service of the teaser between collections. The artificial vagina in its place, thrust and ejaculate in one teaser cow is an important factor in obtaining a representative semen sample. A teaser cow in oestrus will seconds and a good thrust takes both hind legs off the stand to be mounted. This is very important, particularly in a forward direction. The sound of an explosive exhalation indicates ejaculation. It is important refuse to mount if the cow does not stand steadily when that the bull makes a firm thrust as this reflects stimulation of the full complement of ejaculatory fluids and the bull 'tests' her. Furthermore, since many bulls are not used to being handled and therefore have to be so ensures that the sample is as representative of the allowed to mount free of control it is wise to have some-natural ejaculate as possible. A poor quality ejaculate

thing that they are more interested in than the collection associated with a poor thrust may not be representative, tor! The cow must be physically capable of bearing the often showing relatively poor motility, and semen collection weight of the bull and ideally she should be restrained in the collection area by at least a halter. Unfortunately, place at the end of the AV, ideally within the cone itself, many cows have never been haltered so they tend to and thus the semen should be unaffected by the temperature object to this and thus, by struggling, put the bull off temperature of the AV. However, in some cases this is not working. Furthermore, in other cases their stubborn what occurs and the sample may have to be allowed to lack of initial movement is itself off-putting to the bull run down from the AV, which is held vertically after collection and if nothing is happening it is worthwhile trying to collection. In such a case it may not only be affected by lead the cow in front of the bull. In some cases, where the AV temperature but also more contaminated than it is deemed safe, collection can only be achieved with

usual with preputial detritus.

both the cow and bull running loose and a roomy col-

Because of the rapidity of service in experienced

lection area is essential for this to avoid the collector

bulls and the necessity of examining the semen sample

being knocked over or crushed.

as soon as possible after collection it is often difficult to

examine the penis fully and collect semen at the same

Introduction of the bull to the teaser cow: When the bull time. However, an appreciation of its firmness is possi-is brought into the collection area for service the

ble, as is a quick visual assessment of both the penis and

collector should watch only the bull and make the

the prepuce. Should a more thorough examination be

following mental notes:

indicated, mounting can be repeated later without a col-

lection being made, the penis (that part within the

- Demeanour – is he interested in the cow?*

prepuce) being directed toward the examiner, thereby

- Gait – is he lame, if so where?*

exposing the protruded portion for a more prolonged

- Libido – is he keen to work? Watch for the pumping*

examination. This posture can often be held for a few of the ischiocavernosus muscle (this will be apparent by a rippling tail-head movement) and the dripping of clear pre-ejaculatory fluid from the prepuce. Wherever possible, more than one collection should be made, especially if the first is of poor quality. The penis may also be seen protruding a little from the prepuce: does this appear normal? Usually more than one collection may be required to satisfy the collector that the bull has worked well and that the series of samples obtained is as representative as possible. Only experience allows this judgement.

she will move, even if restrained, and this may be quite sufficient to stop the bull mounting.

Experience of collector: There are considerable variations between how bulls cope with the use of an artifi-

*Penile erection and ejaculation: As soon as the bull
cial vagina and the experience of the collector can help
mounts the cow the collector should move forward
considerably in obtaining a good ejaculate as opposed
quickly, but without panic, and with one hand on the
to a poor one. It is difficult to rationalize just what the
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*collector does to ensure a good collection. Firstly, there
tractions with each series of pulses and these increase
is the judgement as to whether to try and collect when
in strength with voltage. The bull may raise his tail, rock
the bull first mounts. Quite often the first mount will be
gently to and fro and, if the probe is incorrectly posi-
'premature' with a poor erection of the penis and in
tioned, lift one hind leg and, if excessive stimulation is
most cases it is best just to deflect the penis by using
applied, the bull will go down. It has been suggested
pressure on the prepuce and let the bull dismount.
that the bull be sedated in some circumstances.*

*While this carries the risk of putting the bull off mount-
However, our experience is that the danger of the bull*

ing again, this is unlikely in bulls with normal libido.

going down means that the technique is best used

Time and effort spent in teasing (even to the point of

without sedation. Sexual rest before electro-ejaculation

removing the bull or the cow from the scene temporar-

seems to improve the likelihood of obtaining a repre-

ily) are usually rewarding and eliminate any doubts that

sentative sample.

can arise when a poor sample is obtained at the very

In our experience the latter techniques are only of

first mount. Secondly, there is the process of actually

value if one obtains an ejaculate with motile spermato-

allowing the penis to enter the artificial vagina. In some

zoa and can study their morphology. Failure to obtain

cases, where the bull will not immediately thrust, it may

an ejaculate does not mean the bull is infertile! Even

be induced to do so by a short, quick, back and forward

then concentration is so variable that at least in our

movement of the artificial vagina. This should be firm

experience it cannot totally be relied upon as repre-

but gentle. Related to this is the decision as to the

sentative of naturally ejaculated semen.

length of the artificial vagina, the pressure of the water

in the liner, the type of liner and temperature. For

Assessment of ejaculate: While there has been, and still example, few bulls will thrust well with an artificial

is, considerable research into the prediction of fertility

vagina temperature less than 42°C, but equally there is

on the basis of tests upon semen (Linford et al. , 1976; no need for it to be more than 55°C. The use of a very

Miller & Hunter, 1987), the general form of on-farm

warm artificial vagina with a rough liner can leave the

semen examination is still largely based on the pio-

penis looking very red! It is vital to remember that the

neering work of the early AI industry. In particular, the

tip of the penis is highly sensitive, and that an ejacula-

reader is referred to the study by Bishop et al. (1954).

tory thrust is usually induced immediately it enters a

A full ejaculate examination generally entails

properly prepared AV.

observing and recording the following:

- Volume

Other techniques of semen collection: Occasionally, the

- *Initial motility of semen*

collection of an ejaculate by artificial vagina proves

- *Progressive motility of spermatozoa*

impossible and it is deemed necessary to examine a

- *Morphology of spermatozoa*

semen sample. In such circumstances, if the bull will

- *Presence of other cells*

mount and serve an oestrous cow then the removal of

- *Concentration of spermatozoa*

semen and vaginal mucus from the vagina of the cow

- *Final overall assessment*

and examination of the spermatozoa in the mucus is, in

our opinion, more sympathetic to the bull's welfare

The first three measurements above are usually

than electro-ejaculation. It is for this reason that the

carried out on the farm, followed by an initial assess-

cow should not be served prior to the visit. Semen can

ment of the results. Smears of semen are also prepared

be collected by massage of the ampullae per rectum

on farm, but not necessarily examined; this is essential

(McGowan et al., 2002) and can be done without a

for the nigrosin–eosin stained smears. Samples and teaser cow in oestrus.

smears are then returned to the laboratory for comple-

In the last resort, and in our opinion only if it is felt

tion of the other tests followed by the final overall

absolutely necessary, one should collect semen by

assessment. The ejaculate should be returned to the on-

electro-ejaculation. This technique depends on electri-

farm laboratory as quickly as possible after collection.

cal stimulation of the sacral segments of the spinal cord,

It must not be allowed to become chilled until after the

which are mainly parasympathetic nerves emerging

first three steps and preparation of the nigrosin–eosin

from the pelvic plexus. This stimulation will often cause

smears are completed.

a form of erection (possibly incomplete) and ejacula-

Volume: *Use a graduated collecting tube.*

tion. The ejaculate often differs from a ‘normal’ ejacu-

Initial motility of semen: *A small drop of semen is*

late by having a considerably greater volume and a

placed on to a warm slide, which is put on a warm

lower sperm concentration (even in the ‘sperm-rich’ microscope stage (35–37°C) and examined (¥100) as portion). This is due to the excessive contribution of the soon as possible after collection. It is scored on a scale accessory glands, in particular the seminal vesicles. of 0–5, ranging from 0 being completely static to 5 being Another disadvantage of this technique is that, at vigorously active. In the bull the major criterion of least with some machines, the animal shows tetanic con- scoring initial motility is wave motion, which is a func-

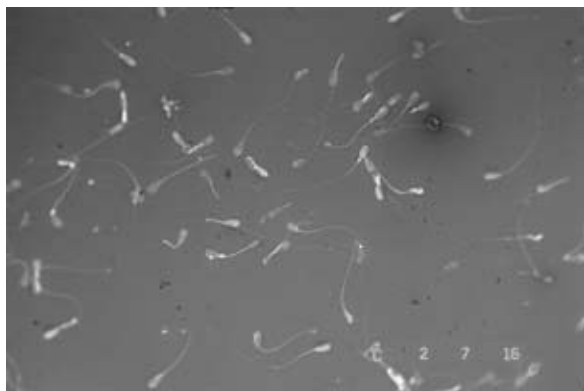
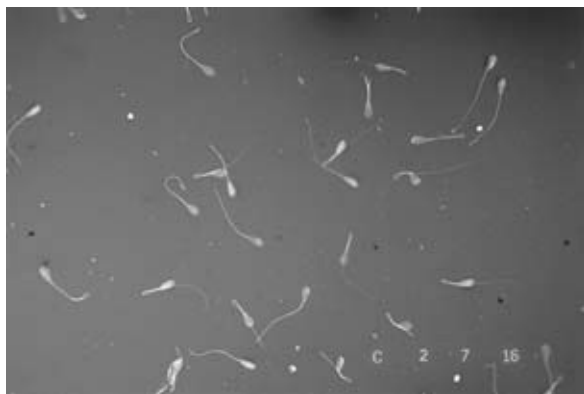


Table 38.3

Guide to assessment of initial motility.

Score

Motility

Approximate %

live, where density

and morphology

are normal

5

Rapid vigorous waves

90+

4

Good wave motion

80

3

Sluggish wave motion

60

2

Active sample, but no wave

40

1

Some motile sperms

20

0

No movement

0

Fig. 38.4

Spermatozoa morphology (nigrosin–eosin ¥ 400).The

reflected tails are artefactual, possibly due to chilling during tion of concentration, proportion of motile sperm and

staining and smear preparation. There is no evidence of cyto-

their activity (see Table 38.3). This simple test, if applied

plasm in the bend of the tail and Giemsa-stained air-dried

correctly, correlates quite well with fertility. Any bull

smears did not show the defect.

that consistently produces semen with a score of less

than 2 should be suspect.

Progressive motility of spermatozoa: *Progressive*

motility, the percentage of motile spermatozoa moving

in a forward direction, is best estimated by diluting the

semen (roughly 1 : 50 depending on concentration) in a

prewarmed (37°C) physiological solution (see p. 601)

and examining this at ¥400 magnification under a warm cover slip. A figure in excess of 50 per cent is certainly desirable. High percentage progressive motility generally also means rapid progressive movement. Examination at this power (¥400) will also allow some initial observation of the morphology of the spermatozoa and the presence of unusual cells in the semen.

Morphology of spermatozoa: The morphology of the spermatozoa is closely related to testicular function and

Fig. 38.5

Spermatozoa morphology (nigrosin–eosin ¥ 400). A the appearance of head, midpiece and tail should all be range of defects including proximal cytoplasmic droplet, dag studied. A general morphological examination entails defect, reflected tail, detached head and microhead. the observation of spermatozoa (and other cells) under high power (¥1000), noting any abnormalities and expressing these as the number of abnormal spermato- in the preparation of a blood film) and allowed to dry zoa/100 spermatozoa counted. This is best done quietly in the warm cabinet or on the warm stage. Chilling

away from the farm although a cursory on-farm look at

¥

during preparation can lead to artefactual effects on

400 will allow tentative prognosis for the owner and

sperm morphology. This stain will also allow the com-

also satisfies the collector that the smear is acceptable.

putation of the live : dead ratio since eosin is a vital dye,

The use of at least two different staining techniques is

staining so-called dead (or immotile) spermatozoa

recommended: one entailing dilution of the semen prior

pink, but leaving live spermatozoa unstained and pale,

to making the slide and the other using a direct smear,

almost white. The percentage live figure correlates

which is stained after fixation. These are described more

well with percentage progressively motile and not

fully below.

surprisingly gives similar correlation coefficients with

(1)

Nigrosin–eosin stain (Figs 38.4, 38.5). Freshly col-

fertility.

lected semen should be mixed with prewarmed stain in

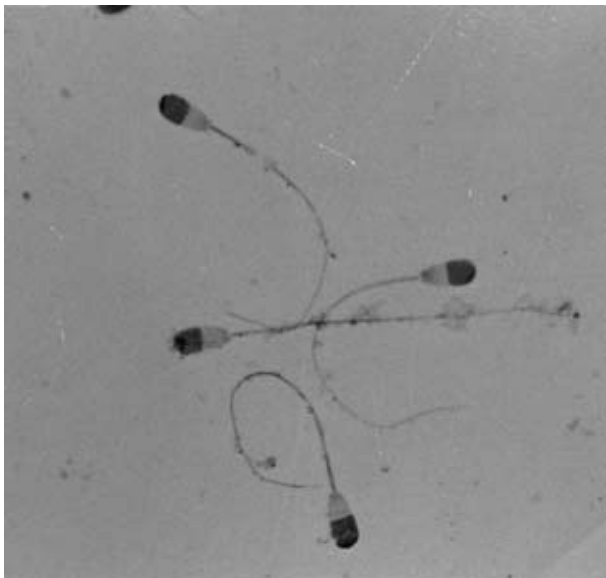
(2)

Indian ink smear. This can be prepared in the same a small test tube roughly according to spermatozoa concentration (average 1[semen] : 10 [stain]) and allowed to incubate at 35–37°C for one minute. One small drop is

then taken and smeared thinly across a prewarmed slide (using the same technique as would be employed made on warmed slides and allowed to air dry and this

(3)

Giemsa (Fig. 38.6) and methylene blue stains. In this case at least three direct smears of the semen are made on warmed slides and allowed to air dry and this



Box 38.4.

Spermatozoa abnormalities.

Head abnormalities

Microhead

Macrohead

Pyriform

Acrosome defect

Neck abnormalities

Detached head

Detached tail

Fractured neck

Other

Midpiece

Double

Swollen

Abaxial

Curved

Distal mid piece reflex

Other

Tail

Coiled (dag defect)

Tail reflected

Fig. 38.6

‘Knobbed sperm’, an inherited defect of the Friesian

Proximal cytoplasmic droplet

breed (Giemsa) (¥700).

Distal cytoplasmic droplet

would usually be done on the farm. On return to the

Table 38.4

Visual criteria for estimating semen concentration

laboratory one is fixed using 10 per cent formal saline

(¥106 spermatozoa/ml) (from Logue & Greig, 1987).

(buffered), one using methanol and the third by gentle

heat. After the first two have been washed in water they

Appearance

Estimated number of

are stained in a simple buffered 10 per cent Giemsa

spermatozoa (¥106)

solution for approximately three hours. Depending on

its source, ordinary tap water will do. This stain should

Thick cream

3000

highlight the acrosome and allow further identification

Cream

2000

of any abnormalities of this region in particular. If one

Milk/cream

1000

brings the slides back to the laboratory before fixation

Milk

500

Water/milk

250

the use of the two different fixes can be of particular

Cloudy

100

*value, since often one gives a better resolution of the
acrosome than the other, presumably due to differences
in the relationship of thickness of the smear, time of air
drying and the fix. The heat-fixed slide is stained by
inherited, for example the knobbed sperm. Results of
methylene blue (see p. 601). Comparison of direct
morphological analysis should be presented in tabular*

smears with those prepared following mixing of semen form, showing the proportions of the various abnormalities and stain (e.g. nigrosin–eosin) allows detection of abnormalities seen (Box 38.4).

factual morphological effects that can arise from faulty

Presence of other cells: A variety of cells are normally found in semen and these cells are best observed using a special technique in preparing the latter (Fig. 38.4).

Normally found in semen and these cells are best observed

Considerable experience is required before reliable

either in the direct smear stained by Giemsa and/or a

counts of morphological abnormalities can be obtained

direct smear heat fixed and stained by methylene blue.

and the reader is referred to Barth and Oko (1989) for

The latter demonstrates leukocytes particularly well.

authoritative guidance on the procedures required.

Most of the cells are of preputial or urethral epithelial

Overall one is aiming for at least 70 per cent normal

origin and are of little consequence. However, the presence

spermatozoa. However, this needs to be qualified as the

presence of large numbers of leukocytes, small darkly stained

effect upon the fertility of the semen depends upon the

ing cells that appear to be degenerating spermatids, type and extent of abnormality (Barth & Oko, 1989); multinucleate giant cells and so-called 'round' cells is thus head and midpiece abnormalities (including cyto-indicative of spermatogenic disruption.

plasmic droplets) are more important than simple

Concentration: This can be estimated using the cri-reflected tails. Some abnormalities are known to be teria in Table 38.4. When a more accurate measurement

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is needed then resource to a calibrated turbidity meter analysis semen samples tend to fall into one of three or a simple haemocytometer is needed.

categories. Firstly all parameters are well above the

Final overall assessment: There has been little study minimum reference figures in Table 38.5 (the semen of the correlation of these commonly measured semen should be of normal fertility). Secondly the important parameters with the fertility of bulls used under natural parameters fall well below (the semen is likely to be service conditions. For semen collected by AV and used

subfertile or sterile). Thirdly the important parameters for artificial insemination there is evidence for correlations are mostly at or just below the minimum reference figures. It is the third category that causes difficulties. If fertility and that is why we have described these tests

there is a history of infertility and there are no other (Bishop et al. , 1954). In North America, the New Society significant findings, the bull could be re-examined after

for Theriogenology Breeding Soundness Evaluation three months to see if there is improvement in the System (Hopkins & Spitzer 1997) is based on examination of semen parameters. If the semen picture is normal at this time of semen collected by electro-ejaculation and may time this suggests the bull was recovering from some not be applicable to AV collected samples. Our experience of testicular degeneration at the first examination. ence with an electro-ejaculator is that in some cases If the semen quality has deteriorated further the prog- perfectly normal bulls fail to give a good sample. nosis is very poor. However, if the quality is similar then However, the Society suggests that to be classified as

test mating to at least ten fertile females followed by

satisfactory the 'gross motility' must be at least 'fair', the pregnancy diagnosis is the only recourse.

'individual progressive motility' at least 30 per cent and

Ancillary tests: *Research into other attributes of*

morphology analysis should show at least 70 per cent

semen, and particularly spermatozoa, that correlate

normal sperm cells. The Society's 'gross motility' is

well with fertility has been consistently directed to the

equivalent to the initial motility assessment described

development of simple cheap methods to improve the

here except that a cover slip is placed on the drop

accuracy of our predictions. Unfortunately, to date, such

of semen; 'fair' ('generalized oscillation') probably

an addition suitable for on-farm use by a practising

equates with a score of 2 and 'individual progressive

veterinarian has not been found. The most widespread

motility' is equivalent to progressive motility. Therefore

tests have been the use of sperm motion analysis using

the measurements that correlate best with fertility are

a variety of computer based packages, cervical mucus

generally believed to be initial and progressive motility, penetration tests and zona pellucida (ZP) penetration percentage dead spermatozoa and percentage morphotests (the simplest of these being the number of accessorily sperm 'trapped' within the ZP). All these measure density are usually also recorded. Table 38.5 shows the ability of the spermatozoa to gain access to the egg minimum reference figures used by the authors when for fertilization more objectively. However, only a more accurate evaluation of the acrosome using phase contrast microscopy seems applicable to the practical must be appreciated that correlation of findings in examinations that we are discussing here and even this semen and the fertility of natural service bulls is an seems more relevant to studying the effects of processing damage upon spermatozoa stored for either AI or information available, which reflects the difficulties of small

other reproductive technologies. Finally there are a scale on-farm investigations of fertility. In the final number of laboratory investigations showing the value of a variety of fluorescent stains which can allow evaluation of the chromosome content (X or Y), DNA variability, membrane integrity, mitochondrial function and vital function (for review see Garner, 1997). In the long term some of these tests may allow an improvement in

Table 38.5

Minimum reference figures (with range) for assessment of ejaculate.

the accuracy of assessment of semen, but until that time it is still an inexact science!

Volume (ml)

4 (2–12)

Appearance

White (some

Observation of natural service

samples have a

yellow tinge)

Finally, after semen collection is completed it is very

Density (¥109/ml)

1.0 (0.3–2.5)

worthwhile standing back and observing the bull

Initial motility (0–5)

≥2

serving the cow naturally. Obviously, this is only possi-

Progressive motility (%)

>50

ble with a cow in season and the consent of the owner,

Total dead spermatozoa (%)

<50

who may not wish to have the cow served. This proce-

Total morphological abnormalities (%)

<30

dure avoids the risk of declaring a bull fertile on the

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basis of normal semen findings when the bull is actually

- *Spermatic cords*

unable to complete natural service.

- *The scrotal sac itself.*

The testes should exhibit freedom of movement

Full physical examination of the bull

within the scrotum and all structures should be of

We have already suggested that the first opinion vet-

normal size, shape, and free from inconsistency in

erinarian may undertake a full examination of the bull

outline, i.e. bumps and nodules. A simple objective

as a preliminary to any further investigations. We rec-

measurement of testicular size is scrotal circumference.

ommend that even if this was done previously it is

This is determined by measuring the circumference of

always worthwhile going through the examinations

the scrotum at its largest point with a tape measure

again. Hard experience has taught that no one is infal-when the testes are in (or manipulated into) a normal

libile and defects can either be missed or could have

fully descended position. In the normal adult bull this

become more apparent between examinations! In any

should be between 33 and 45 cm, depending on breed

event a full examination of the reproductive tract is

and mature size (see Table 38.6). More recently, the use

essential, as is the examination of several other aspects

of real-time ultrasound has been employed as a further of the clinical appearance of the bull. Obviously, where diagnostic aid (Figs 38.7, 38.8). It remains to be seen lameness or some other defect is apparent during precisely how valuable it will be. However, it is now collection the relevant area should now be examined being used by a number of veterinarians, including more thoroughly.

the authors, and has highlighted a variety of defects including orchitis and calcinosis.

Mouth: Assess the age of the bull by examining the incisor teeth. This is often very helpful in impressing

Pelvis: A rectal examination is needed to examine the upon owners how immature their young bulls are.

accessory sex glands. Some of these are found just

Many bulls of continental breeds have still no adult under the hand after it has been fully inserted through incisors at two years of age.

the anus and the ampullae of vas deferens and seminal vesicles can be identified. The prostate and bul-

Eyes: It is also worthwhile examining the eyes, at the bourethral glands cannot

normally be distinguished.

very least to rule out congenital cataracts. Severe

The main part of the former is found at the beginning

cataracts can lead to unpredictable behaviour of the

of the root of the penis, while the latter is found just

bull and are found in all breeds. A good ophthalmo-

anterior to the ventral bend of the root at the bul-

scope is needed to diagnose less obvious abnormalities

bospongiosus muscle. Again ultrasound has an obvious

such as colobomata of the retina.

application, in particular for the examination of seminal

vesicles.

Penis and prepuce: Palpate around the orifice of the

prepuce, the sheath itself and the penis as it runs back

Confirmation of identity: The breed and physical

to the scrotum. It is also worthwhile examining the

description of the bull should be recorded along with

umbilicus for evidence of a hernia.

ear tag numbers and any tattoo numbers where legible.

*Scrotum: Palpation of the scrotum allows definition of Collection of other
diagnostic samples*

the following structures:

The composition of the collection is influenced in part

- *Testes*

by the health status of the national herd and also by that

- *Epididymes (head, body and tail)*

of the herd the bull is in or has been introduced to. The

Table 38.6

Suggested minimum scrotal circumference measurement (cm) to exert selection pressure for increased testicular size for bulls by breed and age in North America (Reproduced from Coulter et al., 1987 with permission from Elsevier Science).

Age of bull

Simmental

Angus and

Hereford and

Limousin

(months)

Charolais

Shorthorn

12–14

33

32

31

30

15–20

35

34

33

32

21–30

36

35

34

33

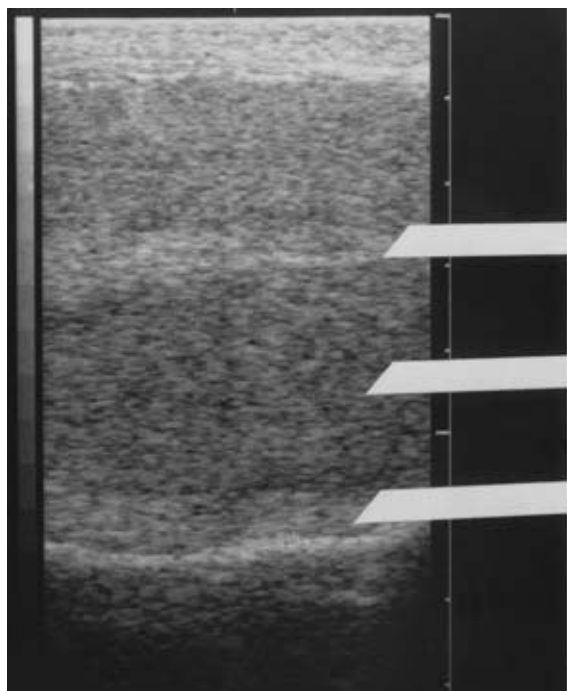
Over 30

37

36

35

34



rt
t
ta



ep:t
t



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Fig. 38.9

Samples for further laboratory investigation routinely

returned from the farm by the authors. From three ejaculates in this case: nigrosin–eosin stained smears, air-dried smears and raw semen; also heparinized blood and preputial wash.

(2)

Blood for BVDV antigen and antibody, unless bull previously tested.

If indicated by history, semen findings and/or clinical

Fig. 38.7

Real-time ultrasound of the testis. rt, rete testis; t, signs collection of further samples, as detailed below, parenchyma of testis; ta, tunica albuginea.

should be considered:

- *Semen for culture of bacteria, virus or Mycoplasma sp.*

- *Blood for serology for IBRV, Leptospira hardjo or Johne's disease.*
- *Blood for biochemistry (e.g. albumin or liver enzymes) or routine haematology.*
- *Faeces for examination for helminth eggs, culture for Salmonella sp. or smear or culture for the bacterium of Johne's disease.*

In some cases sampling of the females involved may also be required, e.g. vaginal mucus for VMAT for C. fetus sp. antibody.

Preliminary diagnosis and prognosis

Prior to leaving the farm and before all the examinations of semen and further laboratory tests have been concluded the farmer will be keen to hear a verbal preliminary diagnosis and more importantly a prognosis

Fig. 38.8

Real-time ultrasound of the tail of the epididymis.

and some advice about what should now be done with ep : t, tail of epididymis; t, testis.

the bull. These can usually be given, with the proviso that they are pending the outstanding test results, since

any major abnormalities of the semen or physical minimum routine employed by the authors in the UK abnormalities in the bull should be apparent before is as follows (Fig. 38.9):

leaving the farm. Nevertheless, it is wise not to be too (1)

Preputial washing for culture and fluorescent dogmatic, particularly with reference to the presence or antibody test for Campylobacter fetus sp. absence of any form of infectious infertility.

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Final diagnosis, prognosis and advice after all test

- *Season*

results are known

- *Social factors*

Having obtained the history of the bull, examined it

- *Overwork*

fully for breeding soundness (and any cows if neces-

- *Nutrition*

sary) and with knowledge of all relevant laboratory test

- *Orthopaedic abnormalities and housing conditions.*

results, a written report should be prepared. This should summarize the history, findings and any laboratory test

Age

results. It should then reach a conclusion, which should

The age at which a bull is capable of mounting, pro-include a diagnosis, however tentative, and, if available truding the penis fully and ejaculating semen (puberty) an appropriate treatment and/or management regime varies with the bull and breed (see p. 479). However, to prevent or control the problem should be offered.

one would normally expect a bull of a high production

Finally a prognosis should be given. A discussion of dairy breed to be capable of serving a cow at 12 months the aetiology of the problem can also be included. In of age, although usually semen production would have general, the most important aspect of this conclusion is commenced some two months earlier. Indeed mount-the prognosis. Effectively there are three alternatives: ing behaviour would be normal in the prepubertal state.

(1)

No abnormality can be detected and the bull

However, one would not advise use for breeding before should be of normal fertility. If the history is of 15 months of age. Continental beef breeds tend to poor fertility recommend a 'test-mate' with at mature at a later age than most dairy breeds and so least 10 fertile females.

more care is needed with such animals when attempt-
(2)

An abnormality consistent with a history of poor ing to use them at a young age, for example less than fertility has been found and the bull is considered 18 months. From a behavioural point of view young long-term infertile or unlikely to return to normal bulls often display naivety and awkwardness in their fertility.

approach to an oestrous cow. There is evidence that this
(3)

The bull is infertile or of questionable fertility, but can be affected by how young males are reared, so that it may recover. In this case, depending on the young bulls raised with female calves are less likely to

diagnosis, the value of the bull and the manner in which the farm use him the bull can be kept, of their dams until weaning (Kilgour, 1984).

treated if necessary and the situation re-evaluated

In extreme cases young bulls may go through a pronounced phase of showing no sexual interest even when presented with a cow in oestrus. This 'sexual inhibition' may be heightened by the presence of another bull.

Further action for the herd and its future

While the condition often indicates an inherently poor breeding management

libido, the problem may correct itself with time. Similarly, despite being clinically normal, bulls can show a

Where any findings made in the bull are likely to impinge on the rest of the herd, now or in the future, this should be mentioned in the report. Examples would include the presence of significant infectious agents or

Low sexual drive may also be due to overwork or evidence of overworking a young bull.

pain, either from an orthopaedic problem or possibly emanating from the reproductive tract (see p. 611).

Manifestations, aetiology, treatment

Genetic factors

and prognosis of bull infertility

There is evidence from work with identical twins that many aspects of mating behaviour, in particular sexual drive and mode of approach to service, are inherited the bull are discussed in more detail below under each (Bane, 1954). Furthermore, despite the wide variation of the four manifestations.

between individuals there is plenty of evidence that some types and breeds of bulls display better libido

Failure to mount

than others. For example, dairy breeds tend to show a stronger sexual drive than beef breeds and there is

The following factors have an influence on the ability now evidence of a quite high heritability for libido

to mount.

(Chenoweth, 1983).

- Age

However, there is little evidence that variation in

- Genetic factors

libido is due to differences in circulating hormone

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concentrations (Bane, 1954). This was demonstrated

direct relationship between nutrition and fertility in

dramatically in an endocrinologically abnormal bull

working bulls. Nevertheless, it is widely believed by

with a 60, XXY chromosome complement, in which the

farmers that performance testing young working bulls

mating behaviour was normal (Logue et al. , 1979).

is detrimental to their longevity as working sires.

There is little firm evidence of any relationship

between poor libido in the bull and poor oestrous

Orthopaedic abnormalities and housing conditions

expression in female offspring, but there is some evi-

dence of a relationship in sheep. Thus libido is a factor

Often the bull will show intent to mount, moving to the

*that should not be ignored in any selection programme
cow and standing behind her, but does not mount
(Chenoweth, 1983).*

*because of painful or physically limiting orthopaedic
abnormalities. Obviously, the level of libido of the bull
mediates the extent of this effect and on some occasions*

Season

*the frustration becomes so great that the bull will strike
There is conflicting evidence about the effect of season
the cow.*

of the year, largely because of the environmental con-

*A large number of orthopaedic problems are liable
ditions in which the bulls were kept. A sluggish sexual
to interfere with the ability to mount and serve a cow.*

drive has been related to periods of extreme heat, cold

The most common problem areas are in the foot, the

*and light reduction (Vincent, 1972; Foote et al. , 1976; hock, the stifle and the
back.*

Gwazdauskas, 1985).

The treatment and prognosis of failure to mount

depends very much on the diagnosis of the clinician.

Many of these problems result from injuries to muscles,

Social factors

tendons and joints caused by the bull slipping when

Age, genetics and season all interact in the herd situa-

attempting to serve on poor underfoot conditions or

tion where the bull is running with a group of cows

foot lameness (see Chapter 31). Thus, adequate housing

either as the only male or along with several other bulls

and service management are prerequisites of good bull

as a mating team. Thus generally the oldest and largest

fertility. The specific requirements of the bull for service

bulls are dominant and spend most of their time in

should be borne in mind and thus any prognosis should

the presence of the sexually active oestrous females

be guarded. This is because many bulls are prepared to

(Chenoweth, 1983). Subordinate bulls spend consider-

amount before a healing process is complete, thus exac-

ably less time with that group and in some cases their

erbatating the condition, and also because frequently the

attempts to serve will be totally disrupted by the dom-

bull produces soft, dystrophic bone in and around the

inant bull.

site of injury resulting in added healing difficulties with subsequent arthritis (Bartels, 1975; Weaver, 1997).

Some injuries appear to be more specific bull problems, Overwork

such as spinal changes (Bane & Hansen, 1962), stiffl

Bulls do become satiated. This state varies with the individual and the herd structure and in temperate climates 38.10).

seems more likely to present during the winter than in

It should also be remembered that other less specific longer daylight periods. In simple terms, introduction of conditions can cause poor libido simply due to the bull a young, totally inexperienced, 18-month to two-year- feeling unwell or being in pain. This is particularly old bull to 20 cycling cows is considered to be risking relevant if the penis and/or prepuce has been damaged overwork, while introduction to as many as 40 is fool- (see below). Finally, other diseases such as bovine hardy! Finally, poor female fertility can compound the

spongiform, encephalopathy (BSE) (see p. 909) and problem because of returns to service.

progressive ataxia (see p. 179) of Charolais cattle (Palmer et al. , 1972) should be borne in mind.

As already mentioned, it is most important to intro-

Nutrition

duce the bull gradually to an adult workload and to

High planes of nutrition result in puberty and maturity

monitor his progress closely at every stage. In particu-

being reached at an earlier age (Salisbury & Van

lar, the bull should be introduced initially to quiet adult

Demark, 1961). There is also some evidence that high

cows which are clearly in season and virtually hand-

energy intakes in early development are detrimental to

mated until the bull and his owner are confident that he

subsequent libido. This effect has been described in

can work reliably and fertility is confirmed by early

both dairy and beef bulls, but without looking closely at

pregnancy diagnosis of those females using real time

whether the regime caused the physical defects such as

ultrasound after day 26. Table 38.7 gives some general

laminitis in the bulls. There does not appear to be a indications of workload.



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preputial defects and orthopaedic problems. It is important not to belittle the problems of diagnosis, for example an early manifestation of either corkscrew penis or a venous drainage defect could conceivably be masked by the overzealous application of an artificial vagina. Observation of the natural service behaviour of the bull should generally avoid this error although some young bulls can display penile abnormalities intermittently. As ever prognosis, treatment and prevention depend upon the exact diagnosis.

In order to understand penile problems fully a brief discussion of the anatomy and physiology of erection in the bull (Fig. 38.11) is necessary.

Intromission can only be achieved if the penis is fully erect. This is a stiffening process brought about by the filling of the corpus cavernosum penis with blood pumped in by the ischiocavernosus penis muscle. Since drainage of the corpus cavernosum penis is very slow in normal bulls, blood pressure in the corpus cavernosum penis builds up well in excess of that in a car tyre

Fig. 38.10

Delayed spastic paresis in the bull.

(over 200 psi) (Beckett et al. , 1974). This is sufficient to harden and straighten the penis and it is consequently

forced out of the sheath. The latter, owing to its elastic

Table 38.7

Recommended workload of bull with age (running nature, envelops the erecting penis up to the start of loose with females).

the free end of the penis. As the penis protrudes and reaches the point of ejaculation, it stiffens still fur-

Age

No. of cows

*ther and spirals in an anticlockwise direction. This
'corkscrew' is caused by the fibrous architecture of the
<2 years*

10 cows

glans, in particular the dorsal apical ligament and spiral

2–3 years

20 cows

distribution of collagen fibres along the tip of the penis,

3 years

30 to 50 cows depending on bull

*allied to the rise in blood pressure in the corpus caver-
nosum penis at ejaculation. Further distortion may be
caused by the very transitory erection of the erectile
tissue at the tip of the penis. It is believed that this may*

*The prognosis for poor libido of indefinable origin
be caused by pressure waves in the corpus spongiosum
must be extremely guarded, since, despite any mitigat-
penis (Ashdown, 1973).*

ing circumstances, it is likely that this problem is inher-

*Clinically, we suggest that there are two major pre-
ent in the make-up of the bull and will recur. In young
sentations associated with failure to achieve intromis-
bulls of around 18 months to two years of age the prog-
sion and each can be further subdivided as follows.*

*nosis is more hopeful, but a maximum time scale of six
months in which to show some evidence of overcoming*

(1)

The penis cannot be protruded sufficiently:

the condition is appropriate.

(a)

Balanoposthitis

Attempts to treat low libido by a variety of hor-

(b)

Short penis

mones, either alone or in combination, have rarely

(c)

Rupture of the corpus cavernosum penis

proved of value and there is a dearth of recommenda-

(d)

Persistent frenulum.

tions apart from sexual rest in any of the common texts.

(2)

Failure to locate vulva or enter vagina:

In fact, matters remain as described by Lagerlöf in

(a)

Psychogenic problems

1951!

(b)

Penile problems: fibropapillomata, drainage

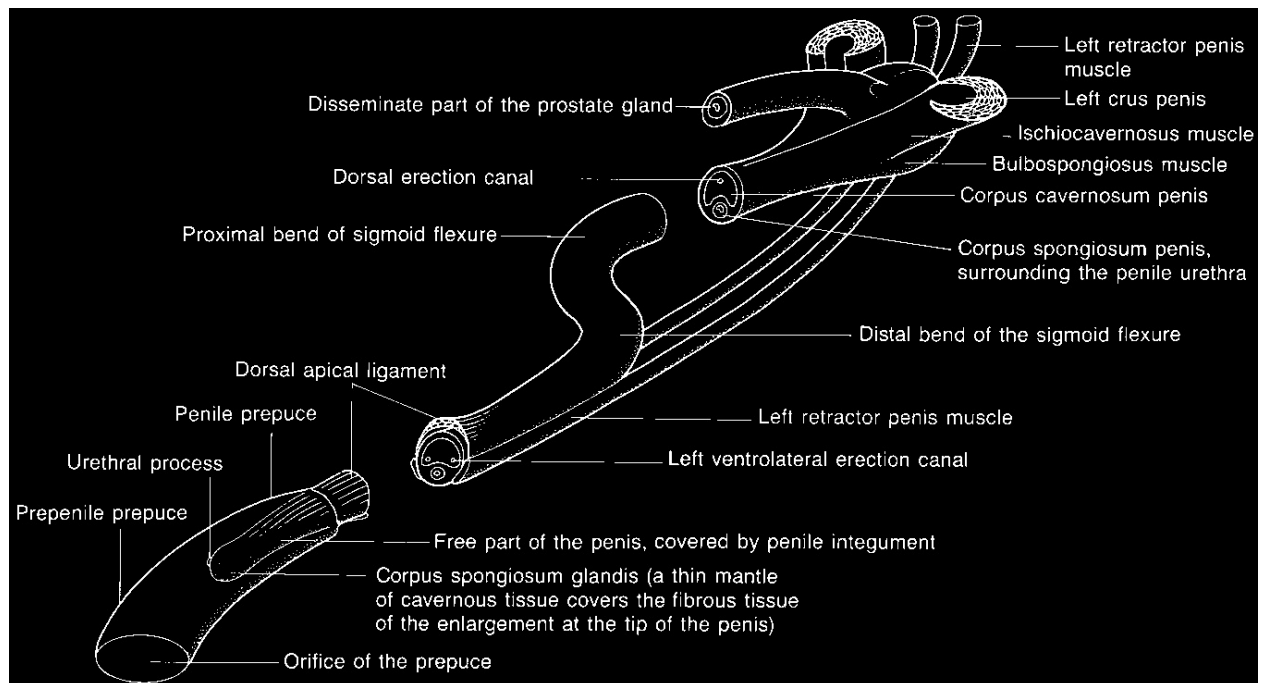
defects, corkscrew penis, deviations.

Failure to achieve intromission

The penis cannot be protruded sufficiently

*The majority of bulls that are presented as being able
to mount, but then fail to gain intromission, suffer from*

*Balanoposthitis: Some cases of inflammation of the
a variety of conditions, which include penile or
epithelium of the penis and prepuce can result in inabil-*



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Fig. 38.11

Diagram to show the anatomy of the penis and prepuce of the bull. (Courtesy of Dr R.R. Ashdown.) infection (Memon et al. , 1980–85). An example is where the bull everts his preputial epithelium, which is then

damaged and infected (Long & Dubra, 1972; Fig. 38.12).

The breeds most commonly affected are the Aberdeen Angus and polled Hereford, which have no retractor preputiae muscle, and the Bos indicus breeds and crosses (Larsen & Bellenger, 1971). Ubiquitous organisms such as Arcanobacterium (Actinomyces) pyogenes and Staphylococcus aureus can often be isolated from such lesions. The smegma produced by the bull is very tacky and the authors know of at least one case where the problem was caused by the firm matting of the hairs over the preputial orifice due to this substance, with

Fig. 38.12

Abcess formation of the preputial epithelium in the resultant failure to protrude the penis. Others have
bull.

described hair rings around the free end of the penis, although this usually produces an annular constriction ity to mate because of pain or physical interference. a few centimetres behind the tip of the penis.

There are three areas of damage:

The penile epithelium: *This can be damaged either traumatically, or by infection, in particular by*

- *The region around the preputial orifice.*

the virus of infectious pustular balanoposthitis

- *The penile epithelium.*

(BHV-1). Damage with subsequent scar tissue can

- *The reflected prepuce which, by virtue of its*

result in a failure to protrude or deviation.

extreme elasticity and mobility, stretches and

The reflected prepuce: *This area is extremely im-
reflects itself along the extended erect penis.*

portant because it must function properly to allow the

The region around the preputial orifice: *Problems in
penis to protrude (Ashdown & Pearson, 1973a). Any
this area may be the result of trauma with subsequent
traumatic damage with resulting scar tissue formation*



Fig. 38.13

Severe tear of
prepuce.

tends to be self-perpetuating since the scar tissue often usually in such cases some of the penis can be protruded. A severe drainage defect problem could also more, infection at such sites is common with subsequent possibly present in this manner (see later). Diagnosis is abscessation. An extreme example of such damage can be based on the increasing difficulty of the bull to protrude occasionally be seen after collection with an artificial the penis after mounting. The penis should be felt to vagina and in some of these cases the tear is very large confirm a firm erection because the condition should indeed (Fig. 38.13). Usually the bull in question is a be differentiated from either a drainage defect of the young inexperienced animal or one returning to work corpus cavernosum penis or a longstanding balanoposthitis after a prolonged layoff (Monke, 1980). Treatment anoposthitis. In our experience the prognosis is hope-

of this condition is largely symptomatic; however, in less.

cases with severe scar tissue formation, surgery is the only course of action other than salvage (Larsen & Rupture of the corpus cavernosum penis: As mentioned Bellenger, 1971; Walker, 1980). Although a guarded earlier, the pressures erecting and firming the penis are prognosis is necessary the chances of recovery will immense and in some unfortunate cases the tunica depend on the site, extent and aetiology of the condialbuginea ruptures (Fig. 38.14). It is possible that in tion. Even in less severe cases sexual rest for at least some cases the tunica albuginea is unable to sustain three months is recommended to ensure adequate integrity because of the pressure alone, but it is more healing. Prevention is best effected by good bull management, regular monitoring of the bull when he is likely that it ruptures under additional strains caused by a sudden penile deflection due to an unexpected move-serving a cow and recording of his performance. Should ment by the cow while service is taking place, or the bull

the bull be part of an AI stud then the importance of slipping while the penis is in the vagina. Rupture gen-collection technique must be re-emphasized.

erally occurs in the region of the sigmoid flexure. It is most common on the dorsal aspect of the distal bend of

Short penis: As the bull grows so the penis should also the sigmoid flexure but can also occur on the dorsal

develop. In some cases it would appear that this does surface of the penis between the root and proximal not occur and the bull becomes less and less able to protrude the penis sufficiently to achieve intromission. The haematoma, which subsequently organizes and prevents the full erection and protrusion of the penis. Diagnosis may even be due to several different conditions is based on a sudden onset of an inability to serve since the penis appears to firm up quite normally. above and anterior to the scrotum. Often the bull also

Fibrous metaplasia of the retractor penis muscle has presents with a prolapse of the prepuce and because the been described (Arthur, 1960) and this has been suc- condition can be painful he may walk stiffly. Where this cessfully treated by myectomy. However, since the con- condition is presented in the acute phase and the sur- dition appears to be heritable surgery is probably not gical approach is adopted, immediate sexual rest fol- advisable. At its simplest, a grossly enlarged pendulous lowed by surgical removal of the haematoma some five abdomen may be a cause of the problem, although to seven days later, before it can organize (Walker,



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Failure to locate vulva or enter vagina

Psychogenic problems: There would appear to be two distinct problems:

- Neck or side mounting;*
- Normal mounting, but without success, the bull being incapable of inserting the penis into the vagina despite a good erection and apparently normal seeking movements, even to the extent of touching the vulva.*

The aetiology for both these conditions, especially the former, is obscure though neck mounting is not uncommon in young inexperienced bulls. However, they usually quickly move round to the rear of the cow.

For the latter one could hypothesise either that this could be an early manifestation of a premature partial drainage defect of the corpus cavernosum penis or that there was an inadequate sensitivity of the penile tip (possibly acquired traumatically or by infection) resulting in a lack of appreciation of the position of the penis relative to the vulva.

A diagnosis of psychogenic depression of libido must be tentative because it is based on a failure to obtain

Fig. 38.14

Rupture of the corpus cavernosum penis.

any other. One should always bear in mind that this condition may be the result of penile damage or an early manifestation of a drainage defect of the corpus cavernosum penis, or may be of orthopaedic origin.

1980), is indicated. In more chronic cases and in those

There is no known treatment apart from initial sexual acute cases where surgery is not adopted gradual rest for at least three to four weeks followed by hand-teasing may eventually result in adhesions being broken mating after some considerable teasing. Often collection by a small artificial vagina using a rough liner may assisted by frequent gentle massage in the area of the obtain an ejaculate and start the bull working. The haematoma. Recurrence is quite common and, if possible, prognosis is very guarded.

ble, identification of the site of rupture is advisable

because the prognosis is more hopeful for a rupture at

Penile problems:

the distal bend of the sigmoid flexure rather than at the

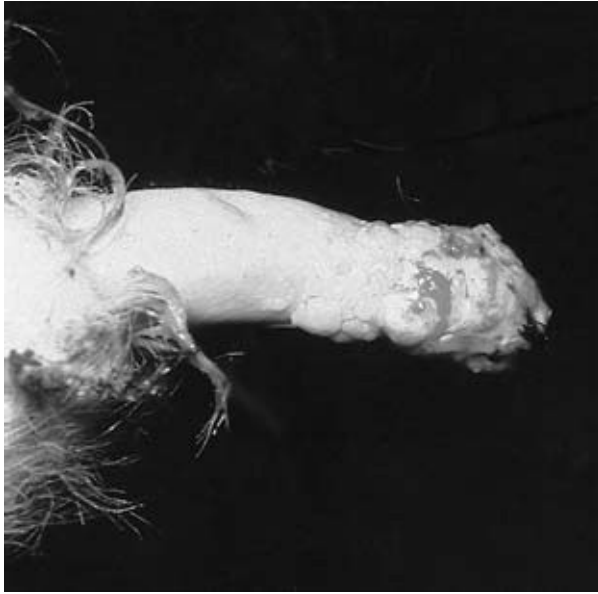
Fibropapillomata: *These are generally found on or proximal bend. This is because the latter is associated near the tip of the penis (Fig. 38.15). They are caused with drainage defects of the corpus cavernosum penis by a virus and are thus transmissible. They can occasionally also cause considerable problems in the female difficult but the introduction of young sexually inexperienced bulls to heifers, especially at grass, should be done penis of young animals less than four years of age and with care. Service in slippery surroundings should be generally regress after a period of two to six months. avoided if at all possible.*

In some cases, however, they persist much longer and some become eroded and infected (Walker, 1980).

Persistent frenulum: *The penile frenulum, which*

Treatment depends on the extent and severity of the normally attaches the free end of the penis to the tumour. A small single pedunculated tumour is clearly prepuce of the prepubertal bull, is normally 'lost' during much easier to deal with than a large, sessile, early puberty, possibly breaking during play and/or cauliflower-like lesion. Most authorities advise surgery masturbation. Occasionally, this does not occur and in severe cases, but it is essential to avoid weakening or its persistence prevents the tip of the penis straightening on erection. It is usually bent ventrally and can result in blood escaping at service and this setback caudally. Although this is not a common condition to surgery has been seen by one of the authors. Note diagnosis is easy, as is treatment, which involves simple that possible leakage from the corpus spongiosum penis surgery.

(CSP) into the terminal part of the urethral lumen has



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defects is largely academic at present since there is no treatment and the prognosis for all of these is hopeless.

Corkscrew penis: *This is one of the most dramatic defects and is often diagnosed by the client. Spiral deviation of the penis within the vagina at ejaculation is physiological but in the clinical condition the spiral deviation precedes intromission and so prevents service. This is most commonly seen in beef bulls of around four years of age (Blockey & Taylor, 1984). In all cases the spiral is anticlockwise and can almost comprise a complete circle. However, it does present with*

varying degrees of severity from intermittent spiralling (which can present problems with diagnosis) to being so severe that spiral deviation occurs while the penis is still in the prepuce and it may then prevent protrusion. Usually it does not occur until the penis makes contact with the vulva of the cow. It should be remembered that spiral deviation has been described as occurring after entry into the vagina immediately prior to ejaculation

Fig. 38.15

Fibropapilloma on the tip of the penis.

in bulls with apparently normal mating behaviour (Ashdown & Pearson, 1973b). The only known treatment is surgical and there are a variety of techniques, all of which depend on either fixing the dorsal apical also been described. This could also cause spurting of ligament to the dorsal aspect of the penis or addition-blood from the penis at service (Ashdown & Majeed, 1978). Since these lesions are transmissible, segregation of affected cattle, especially heifers, from a young bull

tunica albuginea (Mobini et al. , 1982). Postoperative is advisable. Contaminated bedding may infect young

recurrence is quite common after apparent recovery.

bulls and sexual play between young bulls may result in

The problem is said to be heritable but there is

superficial abrasion of the penis and thus susceptibility

little evidence other than anecdotal to confirm

to infection. The use of an older bull on infected

this.

females reduces the risk of infection of the bull. Prog-

Deviations of the penis: *These deviations are largely*

nosis should be guarded as regards both spontaneous

believed to be caused by scar formation such as that

recovery and return to service after surgery .

following balanoposthitis or surgery (Walker, 1980). As

Drainage defects: *Careful anatomical studies of the*

with corkscrew penis, deviations are usually obvious.

normal and abnormal penis have revealed a number of

However, ventral deviations can be confused with a

arteriovenous shunts, which allow the venous cavities of

drainage defect of the corpus cavernosum penis and

the penis to drain more quickly than usual. This results must be differentiated from these. They can usually be in an inability to erect fully or to sustain an erection distinguished by the flexibility of the erect penis that is (Ashdown et al., 1979). It is now considered possible to apparent in drainage defects. Some bulls can accom-distinguish four different venous drainage defects based

modate deviations surprisingly well and if they are not on history and clinical findings. However, in the last too pronounced, careful hand-mating may allow these analysis all are associated with inadequate erection in bulls to learn to serve again. Surgical treatment is again the bull and a diagnosis of venous drainage defect is a possibility (Walker, 1980) for more severe cases. A probably sufficient in most cases (Logue & Greig, guarded prognosis is advisable even for relatively dis-1985). Diagnosis is dependent on a careful assessment crete deviations.

of the history and a thorough clinical examination of the bull while serving. It is advisable not only to attempt

Failure to thrust and ejaculate

to collect semen by artificial vagina but also to palpate

after intromission

the erect penis as much as possible as the bull attempts to mate and finally, if at all possible, the bull should Occasionally, one encounters a bull that is capable of be allowed to attempt to mate naturally and his lack placing the penis in the anterior vagina but then fails to of ability to mate confirmed. Differentiation between thrust vigorously and ejaculate. In some cases the cause the various categories of corpus cavernosum penis may be orthopaedic, but in other cases the cause may

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be neural in origin, whether due to local receptor prob- parties involved in the future of the bull. One theory lems causing a lack of sensitivity of the penis, which may for the relatively lower fertility seen in young bulls be acquired or congenital, or due to a more general when compared with adults is related to the develop- psychosomatic defect. Another possibility is that this ment of both testes and epididymis in the young bull, problem might be due to either a slight defect in the which has roughly half the sperm output of an adult, but

drainage of the corpus cavernosus penis itself or a less than one-quarter of the storage available in the tail defect in the corpus spongiosum penis, or even of the of the epididymis with probably only enough for two to erectile tissue in the tip of the glans. The prognosis is three days' sperm production. Hence it is much easier poor. Finally, in some cases it may just be due to inability to deplete the young bull. Furthermore, it will be experience where, given time, the problem may resolve apparent that relative to the testes the epididymis, par-but the prognosis should be guarded.

ticularly the tail, is still enlarging and just how this interacts with sperm maturation in these young bulls is not

well understood (Amann & Almquist, 1976; Amann and

Normal service with a poor pregnancy rate

Schambacher, 1983).

In summary, while the bull may be capable of repro-

The vast majority of conditions causing this manifesting quite early in life it is not until after three years tation are related to problems associated with sperm of age, or possibly even a little later in the case of late-

production and transport. An understanding of the maturing continental bulls, that one can safely say that complexity of the mechanisms involved in governing the fertility of the bull is unlikely to improve further. and modifying these processes is essential. There are While detailed studies are not really available it does two basic components in the ejaculate: spermatozoa appear that fertility also diminishes with advancing age and seminal fluid. The former are produced in the sem- (Amann & Schambacher, 1983). In the older bull poor iniferous tubules of the testes by spermatogenesis and fertility is associated with degeneration of some of the the latter from the testes, epididymes and accessory sex seminiferous tubules and with the development of tes- glands.

ticular calcinosis in the seminiferous tubules resulting The clinician should consider the following items in non-productive areas of testes. Testicular calcinosis when presented with a bull that is serving normal cows can be quite marked in some older bulls; however, it can satisfactorily but achieving a low pregnancy rate.

also occur in young animals (Turnbull, 1977). Clearly

- *Age*

there is very little that the veterinarian or farmer can

- *Overwork*

do to influence this factor other than limit the mating

- *Testicular hypoplasia (and testicular atrophy)*

pressure to both young and old bulls as recommended

- *Testicular degeneration and atrophy*

earlier and this is discussed further below.

- *Interference with storage and transport of*

spermatozoa

- *Abnormalities of accessory sex glands*

Overwork

Much of the information about semen quality and work

rates come from AI centres. However, the demands

Age

upon bulls running and mating with a herd of cows are

In addition to the influence of age and experience upon

obviously very different from those at an AI centre

libido there is a gradual development towards max-

since young active bulls may serve a cow ten times or

imum fertility in the young bull and this is under more during oestrus. This sort of pressure (ten ejaculates in two hours) results in a diminution of total sperm effects can be seen in the increase in scrotal circumference with age (Coulter & Foote, 1978). This parameter (Salisbury & Van Demark, 1961), mainly due to a fall in is quite valuable as an objective estimate of normal concentration. Concentration in such circumstances development of the testes and epididymes. Figures from falls to as low as 100×10^6 sperm/ml, a level that, in a bull examinations suggest that while a scrotal circumference evaluation as described here, would certainly ference of 35–40 cm is desirable in a young bull (approximately 18 months old), measurements below 30 cm are result in the fertility of the bull being questioned. However, theoretically, a complete rest of around two weeks should allow a return to normal sperm concentration. However, we have collected perfectly normal semen from young

trations in the ejaculate. In practice this is generally the well-grown bulls with scrotal circumferences around case. In other words, the main effect of overuse is to this size. We therefore recommend collecting semen exhaust the epididymal reserves. However, at very high from 'borderline cases' if there are likely to be other and sustained mating pressures a slower return to



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normal number of spermatozoa per ejaculate may be found. Furthermore, it has also been noted that as the number of ejaculates increases, sperm motility tends to fall. In addition, the likelihood of an occasional azoospermic ejaculate increases (Salisbury & Van

Demark, 1961). Finally, there is some information indicating that young bulls given a diet below maintenance for energy take considerably longer to replenish their sperm reserves. This is particularly relevant to young post sale bulls, which are purchased in very good body condition and then put outside with cows in a hard environment, thereby losing condition rapidly. By inference such bulls take considerably longer to replenish their sperm reserves (Salisbury & Van Demark, 1961). The

Fig. 38.16

Unilateral testicular hypoplasia in the Ayrshire

history, number of cows, frequency of working and the

breed. Note the adhesions on the normal testis caused by needle age of the bull will generally indicate this diagnosis. At

biopsy.

worst the semen picture will be of low concentration

(200 \times 10⁶/ml or less), marginal initial motility (2/5),

but reasonable progressive motility (60 per cent +)

and morphology (<20 per cent abnormal). However,

is believed that chromosomal abnormalities may inter-

often the bull has been removed from the breeding

rupt the meiotic process to such a degree that infertile group and rested before examination so the sample appears normal. As already indicated, treatment is (Chandley, 1979). This does not appear to be as much sexual rest since it takes nine weeks for development of a problem in cattle as in man, though there is evidence of reduced fertility in cattle carrying a 1/29 mature spermatozoon. Hence ideally the bull should Robertsonian translocation (Gustavsson, 1974; Logue & Harvey, 1978) and bulls with 61 XXY chromosomes are, as might be expected, completely sterile (Logue et al. , 1979). However, the nature of the bovine karyotype the prognosis is good. However, one must always be aware that such a diagnosis may be complicated by has meant that identification of reciprocal translocations is not easy and it is likely that, with improved tech-

another underlying cause and so it is advisable to be
niques, some will come to light. These abnormalities are
'guarded but hopeful'.

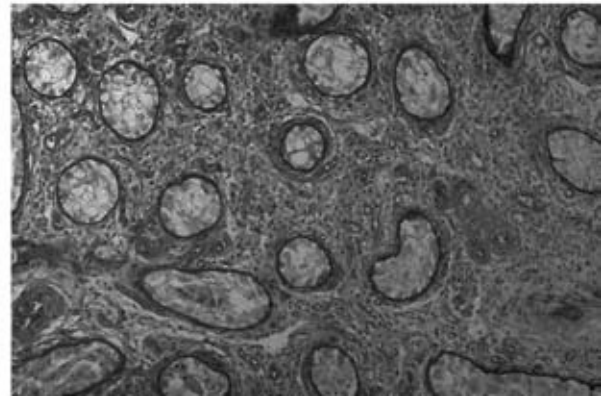
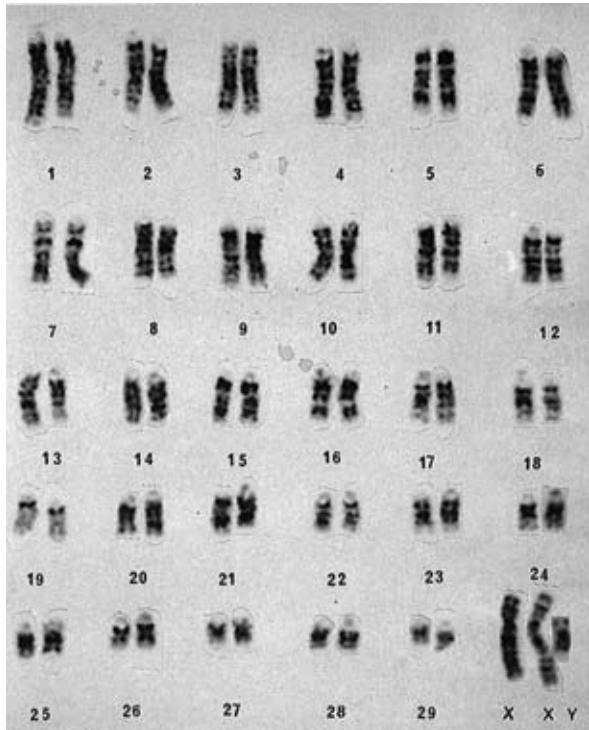
more likely to be associated with infertility than the
Robertsonian translocations, which are not uncommon
in the cattle family.

Although testicular atrophy should be dealt with
Testicular hypoplasia (and testicular atrophy)
under degeneration from a clinical point of view it is
Testicular hypoplasia is defined as the congenital pres-
also being considered here because unless one has a
ence of either one or two small testes (i.e. bilateral or
firm history of the previous size of the testes it can be
unilateral). It has been closely studied in the Swedish
extremely difficult to differentiate between testicular
Highland breed of cattle, where it has been demon-
hypoplasia and testicular atrophy, especially in the
strated to be a heritable defect caused by a recessive
bilateral state. Both present as the 'small testes syn-
gene with incomplete penetrance (Fig. 38.16). In both
drome'(Fig. 38.19). Testes that have been affected by

unilaterally and bilaterally affected cases sexual behaviour subsequent to a severe local infectious condition and secondary sexual characteristics are normal or trauma may feel small, firm and nodular on palpation (Lagerlöf, 1951). It is not known whether a similar inheritance pattern applies to other breeds. It is theoretically possible that toxic or infectious agents affect genuine doubt about the diagnosis.

ing the dam at a critical stage of organogenesis of the Scrotal circumference measurement can be helpful in testes could also cause hypoplasia, either bilateral or giving an objective guide to testis size, as it is a simple, unilateral. Mention should also be made of bilateral testicular hypoplasia that is associated with the abnormal highly repeatable, measurement. In other species it has been related to spermatozoa production and, intriguingly, ovulation rate in the female offspring (Land, chromosome (Figs 38.17 and 38.18). Other cytogenetic

1978). However, from a practical point of view it is sufficient to note that those males with a below normal





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(a)

(b)

Fig. 38.17

(a) G-band karyotype 61XXY. (b) XXY bull. Tubules lined by Sertoli cells only, Leydig cells moderately increased in proportion (Masson ¥195). (Courtesy of the Veterinary Record.) scrotal development have a greater likelihood of being

infertile. A further diagnostic aid that may prove of

value is the determination of the chromosomal consti-

tution of the animal.

As already mentioned, care must also be taken not

to jump to conclusions. Surprisingly good quality semen

has been collected from bulls with disappointingly small scrotal circumferences. However, such occasions are rare, so that in general the prognosis for the 'small testes syndrome' is poor. As far as the unilateral condition is concerned this is more problematical since the animal is often fertile. Here every attempt to obtain as reliable a history as possible should be made in order to differentiate between hypoplasia and atrophy because of the strong evidence of the heritable nature of hypoplasia.

If the diagnosis is hypoplasia the owner should be advised that such stock are only fit for the production of 'slaughter generation' animals.

Testicular degeneration and atrophy

In many infertile animals the main histological finding is the absence of dividing spermatogenic cells, the

*Sertoli cells and interstitial cells still being present. This **Fig. 38.18***

A 61XXY bull with bilateral testicular hypoplasia.

is often the result of testicular degeneration. Evidently,



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Fig. 38.19

'Small testes syndrome' (scrotal circumference 24

Fig. 38.20

Severe acquired testicular abnormalities in a Charo-

cm) in a Limousin bull aged 2 years with no previous history of lais bull aged 5 years. Chronic orchitis of the left testis mani-fertility. The bull also intermittently displayed spiral deviation of fested as atrophy, fibrosis and adhesions. The right testis whilst the penis. Semen density was very low (4.0 ¥ 106/ml).

of normal size was very soft in consistency. The bull showed a complete absence of libido.

however, histology does not show the defects of func-

*Insults to spermatogenesis causing temporary or
tion, in particular the delicate interaction between the
permanent testicular degeneration can be categorized
cells of the testes and their hormonal influences from,
as either local or systemic.*

*and feedback to, other hormonal control centres. Sper-
Local:*

*matogenesis is a very sensitive process and can be easily
upset. Nevertheless, it can also recover, particularly if*

Scrotal sac disruption: Obviously, severe trauma to
the insult has been transient. Studies in the rat have
the scrotum can have a direct effect upon the testes and
shown firstly that Sertoli cell function was disrupted by
epididymes, but one of the main functions of the scrotum
a whole variety of noxious stimuli and that one side
is temperature control and any disruption of this can
effect of such damage was an increase in serum follicle
interfere with spermatogenesis. The first area of sper-
stimulating hormone (FSH) concentrations due to a
matogenesis affected is meiosis; the spermatogonia tend
decreased output of inhibin. Secondly, the Leydig cells

not to be affected and, after removal of the insult, regeneration were also affected since serum luteinizing hormone (LH) concentrations were higher and testosterone concentrations lower. Finally, there was evidence of a reduced response to human chorionic gonadotrophin (hCG) (de Kretser, 1979). While weather conditions are capable of insult and cause variations in semen quality (Parkinson, 1987), it really to the spermatogenic epithelium interferes with andro- requires trauma, a severe skin infection, an allergic reaction with scrotal oedema, a scrotal hernia or a deformity to an increased FSH concentration, which in turn alters of the pampiniform plexus (so-called varicocele) to the balance of testosterone–oestradiol synthesis produce serious infertility. Diagnosis should be determined towards the latter with a subsequent alteration in LH mined by a clinical examination. The most obvious prob-

concentrations. Since there is evidence that LH can
lems are varicocele and inguinal herniation. Care needs
directly affect the Leydig cell, testosterone release and
to be taken when making the latter diagnosis as a pad of
so ABP release by the Sertoli cell, it can be hypothe-
fat can lie above the pampiniform plexus and feel very
sized that if events are prolonged or extreme these
like a hernia. The treatment and prognosis both depend
effects may accumulate and eventually result in a per-
on the cause. Thus for a simple skin infection one would
manent derangement. However, it is of considerable
be more hopeful of a return to normal fertility than for a
interest to veterinarians in the field that immunization
varicocele or a hernia.

of bulls to gonadotrophin-releasing hormone (GnRH)

Orchitis/epididymitis: Inflammation of the testes is

is generally reversible (Robertson et al. , 1982). This commonly a purely local
problem and frequently it only

then implies that in many clinical cases where such a
involves one testicle. However, often the infection so
regeneration of the seminiferous epithelium does not

interferes with the temperature gradient of the scrotum occur the underlying aetiology is more than just a that the remaining testicle is indirectly affected (Fig. simple hormonal dysfunction.

38.20). A similar effect is seen with epididymitis,

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although in this case there is the further problem of infected with BVDV or to have high antibody titres to obstruction of spermatozoa and testicular fluid on the Leptospira hardjo. However, in some cases the animals affected side. Often orchitis and epididymitis are have subsequently proved fertile; this includes BVDV concurrent. The causes of orchitis/epididymitis infection are not well documented. In many cases unpublished information). It should again be remembered that the bull might be presented some time after affected testes, but whether this is the original pathogen the incident when recovery is taking place. This can is uncertain. Other bacteria, such as those of the

take a considerable time – at least some two to three months. Finally one interesting condition, worthy of mention, is idiopathic angioneurotic oedema. This is occasionally seen in young bulls, particularly of mental breeds, and generally occurs soon after introduction to grazing. There is often tremendous oedema of all the dependent areas, especially the scrotum, with low concentration of spermatozoa, poor/marginal motility and poor spermatozoan morphology, especially consequent azoospermia. The latter is presumably due to a temperature effect. In our limited experience recovery is usually complete, however with other illnesses such as large multinucleate cells, so-called ‘giant cells’. These conditions should also be relatively easily diagnosed by a clinical examination. Confirmation can be obtained from the semen findings. Generally, there will be low concentration of spermatozoa, poor/marginal motility and poor spermatozoan morphology, especially consequent azoospermia. The latter is presumably due to a temperature effect. In our limited experience recovery is usually complete, however with other illnesses this is not always the case, especially in older

Culture of semen may allow the identification of a bulls. Treatment and prognosis are dependent on the causative micro-organism. However, the findings will to history, the origin of the insult and the age and type of some degree depend on the time scale. Thus a bull with the bull.

a spermatocele (see later) on the head of the epididymis

Drugs: *Although therapeutic products are subject to could have been affected several years previously and very stringent safety screening there are certain drugs the effects of the lesion subsided, allowing the semen that might affect male fertility. Anabolic steroids given quality of the bull to return to normal.*

to young growing bulls have been shown to have an Treatment by antibiotics is usually ineffective and the undoubted effect upon spermatogenesis, resulting in a main aim should be to prevent testicular degeneration reduced testicular size and delayed development of the of the sound testis and epididymis. The strategy adopted seminiferous tubules and interstitial cells (O'Lamhna & depends on the severity of infection. Thus assuming the

Roche, 1983; Deschamps et al. , 1987a,b). Although it remaining testis is not affected the removal of a grossly

was suggested that these effects were temporary, in fact

infected testis is the most straightforward remedy.

there was a delay in puberty and the effect was more

Unfortunately, even then the local, and possibly sys-

severe in younger animals (these studies have only

temic, damage to the other testis may be sufficient to

involved animals of up to 15 months and may not truly

cause a permanent derangement of semen production.

reflect the field situation). In addition there was evi-

Thus the prognosis, even in animals that have recovered

dence of interference in the hypothalamic–pituitary

either after surgery or after conservative treatment,

axis and the size and structure of the testes and epi-

should be guarded. The owner should be reminded that

didymis. Finally, the fact that the studies were relatively

the daily sperm output may be affected in apparently

short term makes one very wary of such a claim. The

recovered animals and that they should carefully

only problem that might arise is with clandestine and

monitor the cows that have been served in the bull's improper administration of a drug, such as an anabolic first series of matings to ensure that the proportion of steroid or b agonist, with the intent of improving the returns to service is acceptable.

appearance of the bull or even liveweight performance

test results. It is possible to test for residues. It is possible that a bull could suffer from an adverse reaction to

Systemic:

one of the common treatments, although such cases are

Systemic illness: Severe illness can disrupt spermatogenesis by one or all of three routes: firstly, by treatment. Given the effect of anabolic steroids, by interfering with the temperature gradient of the testes, inference treatment with corticosteroids is also likely to secondly as a consequence of toxæmic damage to the affect semen quality (Li & Wagner, 1983), and indeed spermatogenic epithelium and finally by interfering some slight changes have been described short term. with the complex hormonal secretions which control

However, administration to adult Holstein bulls did not spermatogenesis and their interactions. The roles of appear to cause any practical spermatogenic problems bacteria and viruses in the aetiology of this condition (O'Connor et al. , 1985). Nevertheless, there is more are not well understood. For example, a few apparently recent evidence that some non-steroidal anti-infertile bulls have been found to be persistently inflammatory drugs such as flunixin could have an

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effect (Archibald et al. , 1990). Treatment of such earlier have an effect solely upon the epididymes, since, adverse reactions is symptomatic while the prognosis as already mentioned, any disruption of testicular function depends on the exact form that the adverse reaction takes. Field experience has also failed to show any

The epididymis, unlike the testis, develops from the fertility effect associated with the administration of the mesonephric (Wolffian) ducts and is prone to several commonly used antibiotics. However, again the periods

congenital malformations (Blom & Christensen, 1960).

of administration were relatively short. Abbitt et al.

Aplasia of part of the epididymis, ductus deferens and (1984) reported that neither dihydrostreptomycin sulphate nor oxytetracycline hydrochloride had any real defect in the bull. The aplasia is usually unilateral and effect upon reproductive function, even when given in can be very specific. It is generally associated with greater than normal therapeutic doses.

spermatocele formation (so-called sperm granulomas)

The diagnosis of testicular degeneration is dependent proximal to the aplasia (Blom & Christensen 1960).

upon a history of fertility, followed by infertility with

These are swellings of the epididymis reflecting a the latter being confirmed by the collection of a semen rupture of the tubules and the consequent foreign body sample of poor spermatozoa motility, concentration and reaction to the escaped products of the testes and are morphology. In addition there will sometimes also be most commonly seen on the head of the epididymis.

small, darkly staining, degenerate spermatids, which in
While they have been described as being a result of seg-
some cases 'raft' together in clumps. In the extreme case
mental aplasia (see above), in some cases spermatoc-
the sample may be aspermic. In cases of testicular
les arise in this position apparently spontaneously.
degeneration the testes may be smaller than normal
Spermatocele formation has been diagnosed in the field
due to atrophy, but often they are within the norm.
following a sudden failure to mount and thus, by infer-
While collection of semen is the main diagnostic tool
ence, it is painful in the acute phase (Logue & Greig,
many workers have desired to examine the seminifer-
1986). Since this was the only behavioural demonstra-
ous tubules. A number of authors have attempted to use
tion of pain it could be that any discomfort is only
a testicular biopsy as a means of obtaining histological
appreciated during sexual arousal. The exact relation-
evidence of spermatogenic dysfunction (Galina, 1972).
ship of the spontaneous condition to segmental aplasia
This technique is hazardous even when one uses a

is uncertain but one cannot ignore the possibility of modern disposable biopsy needle due to haemorrhage an infectious condition causing an epididymitis and (the testis is very vascular), spermatogenic disruption subsequent spermatocele.

However,

epididymitis

and adhesions. However, with time, good spermato- resulting from infection by Brucella sp. and the genic recovery can occur (Galina, 1972; Logue, 1975).

Haemophilus/Histophilus group appears to occur most

Because testicular degeneration can be either temporary or permanent, it is impossible to be certain of infections have also been implicated in epididymitis (La Faunce & McEntee, 1982).

tion. However, some guidelines can be obtained by

Unilateral spermatoceles need not necessarily cause

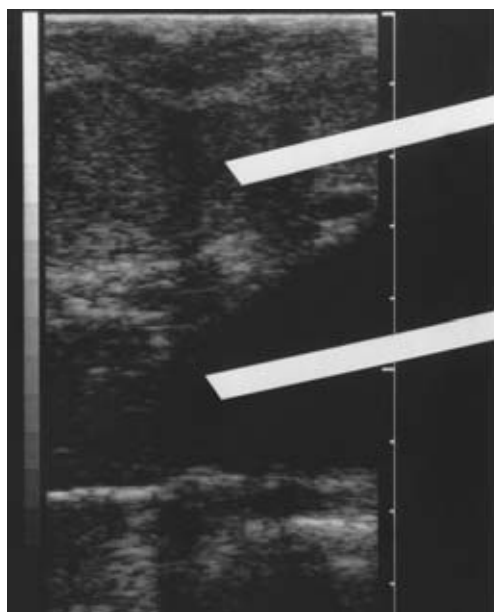
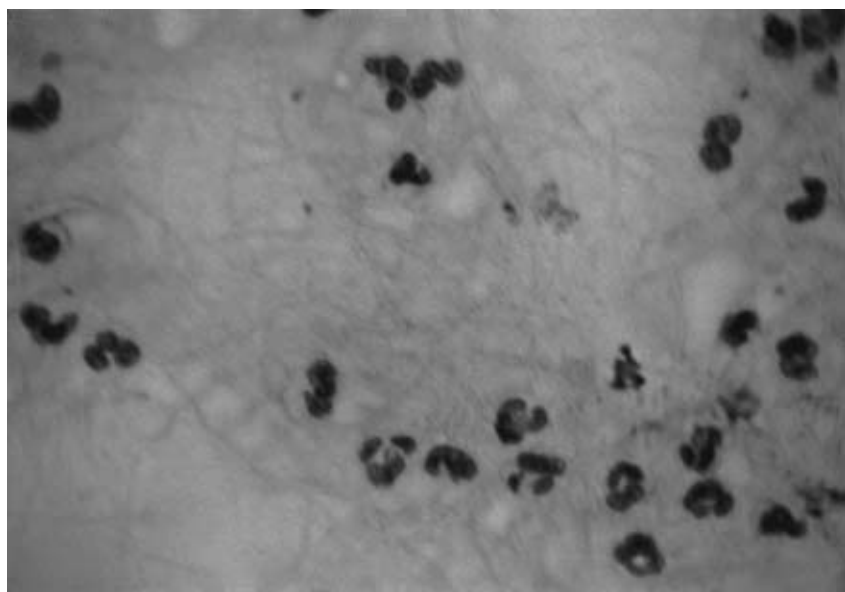
looking at the length of infertility and the semen

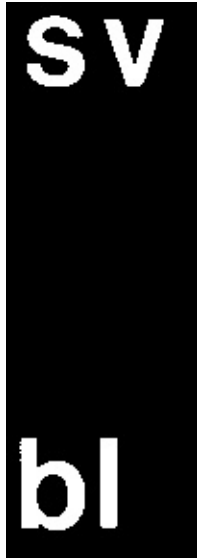
sterility since once the inflammatory reaction has

picture. Should the time scale be relatively short, say settled the remaining testicle can produce an three months, and there is evidence of normal motile apparently normal ejaculate. However, it is clearly not spermatozoa, albeit at a low concentration, then it is advisable to use such an animal with a large number of worth waiting another three months for a re-examination. Where the degeneration of the seminiferous bulls involved were at an AI centre and the concentration of the ejaculate never returned to the level seen gradual diminution in size of one or both testes, i.e. testes before the incident. In one of these bulls the epididymis testicular atrophy. Unfortunately, unless one had prior knowledge that the affected bull had previously had and the bull had to be destroyed. No significant micro-normal-sized testes in the bilateral condition this small organism could be found (Logue, D.N. & Hignett, P.G., size could quite easily be considered to be testicular

unpublished information). Ease of diagnosis depends on the site and the extent of the problem. Should there be a spermatocele then this is generally easily palpated provided one is careful and palpates the entire epididymis. However, some defects can be very small and Interference with transport and storage of spermatozoa ultrasound may well prove a very useful diagnostic aid.

Treatment is usually symptomatic and certainly in the early stages antibiotic administration is probably worthwhile. It is generally impossible to differentiate clinically which of the local and systemic effects mentioned





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Fig. 38.21

*White blood cells in
semen (methylene blue) (¥700).*

Abnormalities of the accessory sex glands

*The accessory sex glands consist of the seminal vesicles,
ampullae, prostate and bulbourethral glands.*

*As already mentioned, abnormalities of the seminal
vesicles are the most commonly diagnosed condition.*

*Segmental aplasia of the seminal vesicle occurs and
often appears to be related to a congenital defect (Blom,
1979a,b). Numerous infectious agents have been
isolated and implicated in the aetiology of seminal
vesiculitis in the bull, such as members of the Chlamydia*

group, *Mycoplasma bovis* and *Mycoplasma bovis* (La Faunce & McEntee, 1982). However, in severe clinically recognizable cases the most common organism isolated is *Arcanobacterium pyogenes* (Arthur, 1960; McCauley, 1980). Some authors believe that these infections of the seminal vesicles are secondary to congenital defects. Diagnosis of these abnormalities is firstly dependent upon rectal palpation (where enlargement, firmness, pain and adhesions may be detected in the case of seminal vesiculitis) and ultrasonic scanning; and secondly upon laboratory tests, particularly the examination of semen for white blood cells which indicate the presence of inflammation (Fig.

Fig. 38.22

Real-time ultrasound of the seminal vesicle. sv, 38.21). Hopefully, ultrasound scanning will prove of seminal vesicle; b, bladder.

value in the reliable diagnosis of severe seminal vesiculitis and also of segmental aplasia of the seminal vesicle (Fig. 38.22). However, as mentioned before, the application of the technique is still in its infancy (Little

In some cases small compact masses up to the size of & Woods, 1987). Treatment of an infectious condition a pea, so-called cysts, can be felt both on the epididymis obviously requires antibiotic, preferably one with a and on the spermatic cord itself. They are developmen- broad spectrum, and given that there is no evidence of tal in origin and of no consequence as long as they do severe effects upon the structure of the seminal vesicles, not interfere with sperm transport.

prognosis is good.

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Conditions affecting the other smaller accessory

of the corpus cavernosum penis. Veterinary Record, 104, sex glands of the pelvis in the bull have rarely been

423–8.

recognized clinically, but this may yet be possible using

Ashdown, R.R. & Glossop, C.E. (1983) Impotence in the bull: ultrasound.

(3) Rupture of the corpus cavernosum penis proximal to the sigmoid flexure. Veterinary Record, 113, 30–7.

Ashdown, R.R. & Majeed, Z.Z. (1978) Haemorrhage from the

Conclusion

bovine penis during erection and ejaculation: a possible explanation of some cases. Veterinary Record, 103, 12–13.

The investigation of infertility in the bull is one of the

Ashdown R.R. & Pearson, H. (1973a) Anatomical and experimental studies on eversion of the sheath and protrusion of most interesting examinations in farm animal practice.

the penis in the bull. Research in Veterinary Science, 15, The approach is standard and success is dependent 13–24.

upon attention to detail in all the facets mentioned in

Ashdown, R.R. & Pearson, H. (1973b) Studies on ‘corkscrew’ this chapter. One of the main advantages the general penis in the bull. Veterinary Record, 93, 30–5.

practitioner has compared with the specialist is an

Bane, A. (1954) Sexual function of bulls in relation to intimate knowledge of the farm, the ability of those heredity rearing intensity and somatic conditions. Acta involved as stockworkers and also a fair idea of the Agricultura Scandinavica, 4, 95–208.

reliability or otherwise of the history and records. On

Bane, A. & Hansen, H.-J. (1962) Spinal changes in the bull and the other hand, the major disadvantages are the small

their significance in serving ability. Cornell Veterinarian, 52, number of cases that the practitioner will see in a year,

363–84.

so limiting the experience that can be built up, and the

Bartels, L.E. (1975) Femoral – tibial osteoarthritis in the bull.

Clinical survey and radiologic interpretation. Journal of the standard of equipment,

particularly microscope,

American Veterinary Radiology Society, 16, 151–8.

required. Finally the amount of time and flexibility of

Barth, A.D. & Oko, R.J. (1989) Abnormal Morphology of

time that need to be devoted to such work must be

Bovine Spermatozoa. Iowa State University Press, Iowa.

balanced against other, possibly more remunerative,

Beckett, S.D., Reynold, T.M., Walker, D.F., Hudson, P.S. & demands of a busy practice.

Burphit, R.C. (1974) Experimentally induced rupture of

corpus cavernosum penis of bull. American Journal of

Veterinary Research, 35, 765–7.

References

Bishop, M.W.H., Campbell, R.C., Hancock, J.L. & Walton, A.

(1954) Semen characteristics and fertility in the bull. Journal Abbitt, B., Berndtson, W.E. & Seidel, G.E. (1984) Effect of of Agricultural Science, 44,

227–48.

dihydrostreptomycin or oxytetracycline on reproductive

Blockey, M.A. de B & Taylor, E.G. (1984) Observations on

capacity in bulls. American Journal of Veterinary Research, spiral deviation of the penis in beef bulls. Australian Veteri-45, 2243–6.

nary Journal, 61, 141–5.

Amann, R.P. & Almquist, J.O. (1976) Bull management to

Blom, E. (1979a) Studies on seminal vesiculitis in the bull 1.

maximise sperm output. In Proceedings of the 6th Technical Semen examination methods and post mortem findings.

Conference on Artificial Insemination and Reproduction, pp.

Nordisk Veterinaermedicin, 31, 241–50.

1–10. National Association of Animal Breeders.

Blom, E. (1979b) Studies on seminal vesiculitis in the bull 2.

Amann, R.P. & Schambacher, B.D. (1983) Physiology of male

Malformation of the genital organs as a possible predispos-

reproduction. Journal of Animal Science, 57 (Suppl. 2), ing factor in the pathogenesis of seminal vesiculitis. Nordisk 380–403.

Veterinaermedicin, 31, 293–305.

Anon. (1979) The Federation of UK Milk Marketing Boards

Blom, E. & Christensen, N.O. (1960) The etiology of

Dairy Facts and Figures 1979, Thames Ditton.

spermiostasis in the bull. *Nordisk Veterinaermedicin*, **12**, Anon. (1988) *The Federation of UK Milk Marketing Boards*

453–70.

Dairy Facts and Figures 1988, Thames Ditton.

Caldow, G.L. & Taylor, D.W. (1997) *Experiences with*

Anon. (1998) *The National Dairy Council Dairy Facts and*

venereal campylobacter infection in suckler herds. Cattle

Figures 1998, London.

Practice, **5**, 327–34.

Archibald, L., Gronwall, R.R., Pritchard, E.L. & Tran, T.

Chandley, A.C. (1979) *Chromosomal basis of human infertil-*

(1990) *Acrosome reaction and concentration of*

ity. British Medical Bulletin, **35**, 181–6.

prostaglandin E2 in semen of rams treated with Flunixin

Chenoweth, P.J. (1983) *Sexual behaviour of the bull: a review.*

meglumine (BANAMINE). Theriogenology, **33**, 373–83.

Journal of Dairy Science, **66**, 173–9.

Arthur, G.H. (1960) *Reproductive abnormalities of male*

Clark, B.L. (1971) *Review of bovine vibriosis. Australian Vet-animal. In Wright's Veterinary Obstetrics*, 3rd edn, pp. 507–8.

erinary Journal, **47**, 103–107.

Baillière Tindall and Cox, London.

Coulter, G.H. & Foote, R.H. (1978) Relationship of body-

Ashdown, R.R. (1973) Functional anatomy of the penis in

weight to testicular size and consistency in growing Holstein

ruminants. In *Veterinary Annual* (ed. by C.S.G. Grunsell & bulls. *Journal of Animal Science*, **44**, 1076–9.

F.W.G. Hill), Vol. 13, pp. 22–5. John Wright & Sons, Bristol.

Coulter, G.H., Mapletoft, R.J., Kozub, G.C. & Cates, W.F.

Ashdown, R.R., Gilanpour, H., David, J.S.E. & Gibbs, C.

(1987) Scrotal circumference of two-year-old bulls of

(1979) Impotence in the bull: (i) Abnormal venous drainage

several beef breeds. *Theriogenology*, **27**, 485–91.

Bull Infertility • 625

De Kretser, D.M. (1979) *Endocrinology of male infertility*.

Lagerlöf, N. (1951) Hereditary forms of sterility in Swedish

British Medical Bulletin, **35**, 187–92.

cattle breeds. *Fertility and Sterility*, **2**, 230–42.

Deschamps, J.C., Ott, R.S., McEntee, K., Heath, E.H.,

Land, R.B. (1978) Genetic improvement of mammalian fertil-

Heinricks, R.R., Shanks, R.D. & Hixon, J.E. (1987a) Effects of zeranol on reproduction in beef bulls. *Scrotal circumference*-109–35.

ence, serving ability, semen characteristics and pathologic

Lander, K.P. (1983) New technique for collection of vaginal changes of the reproductive organs. *American Journal of mucus from cattle. Veterinary Record*, **112**, 570.

Veterinary Research, **48**, 137–47.

Larsen, L.H. & Bellenger, C.R. (1971) surgery of the pro-

Deschamps, J.C., Ott, R.S., Weston, P.G., Shanks, R.D., Kesler, lapsed prepuce in the bull: its complications and dangers.

D.J., Bolt, D.J. & Hixon, J.E. (1987b) Effects of zeranol on Australian *Veterinary Journal*, **47**, 349–57.

reproduction in beef bulls. Luteinising hormone, follicle-

Li, P.S. & Wagner, W.C. (1983) In vivo and in vitro studies on stimulating hormone and testosterone secretion in response

the effect of adrenocorticotrophic hormone or cortisol on

to gonadotrophin-releasing hormone and human chorionic

the pituitary response to gonadotrophin releasing hormone.

gonadotrophin. *American Journal of Veterinary Research, Biology of Reproduction*, **29**, 11–24.

48, 31–6.

Linford, E., Glover, F.A., Bishop, C. & Stewart, D.L. (1976) Dufty, J.H. (1967) Diagnosis of vibriosis in the bull. *Australian The relationship between semen evaluation methods and*

Veterinary Journal, **43**, 443–7.

fertility in the bull. *Journal of Reproduction and Fertility*, **47**, Foote, R.L., Munkenbank, N. & Greene, W.A. (1976) Testos-283–91.

terone and libido in Holstein bulls of various ages. *Journal Little, T.V. & Woods, G.L. (1987) Ultrasonography of accessory of Dairy Science*, **59**, 2011–13.

sex glands in the stallion. *Journal of Reproduction and*

Galina, C.S. (1972) An evaluation of testicular biopsy in farm Fertility, **35**(Suppl.), 87–94.

animals. *Veterinary Record*, **88**, 628–31.

Logue, D.N. (1975) A study in bovine cytogenetics. PhD thesis, Garner D.L. (1997) Ancillary tests of bull semen quality. In

Glasgow.

Bull infertility (ed. by S.D. Van Camp). The Veterinary Logue, D.N. & Greig, A. (1985) Infertility in the bull. 1. Failure Clinics of North America, WB Saunder, Philadelphia.

to mate. In Practice, **7**, 185–91.

Gunn, G.J., Stott, A.W. & Scanlan, S.A. (1998) Estimating the Logue, D.N. & Greig, A. (1986) Infertility in the bull. 2. Infer-losses associated with bovine viral diarrhoea (BVD) within

tility associated with normal service behaviour. In Practice, the Scottish cow-calf herd. In Proceedings of the XXth

8, 118–22.

World Buiatrics Conference, Sydney, Australia, 1998.

Logue, D.N. & Greig, A. (1987) Infertility in the bull. 3.

Gustavsson, I. (1974) Appearance and persistence of the 1/29

Collection and examination in semen. In Practice, **9**, 161–70.

translocation in cattle. In Colloque les Accidents Chro-

Logue, D.N. & Harvey, M.J.A. (1978) Meiosis and spermato-

mosemiques de la Reproduction (ed. by J. Bové & A. Bové), genesis in bulls' heterozygotes for a presumptive V29 Robertsonian translocation. *Institut National de la Santé et de la Recherche*

sonian translocation. *Journal of Reproduction and Fertility, Médicale, Paris.* **54**, 159–65.

Gwazdauskas, F.C. (1985) *Effects of climate on reproduction*

Logue, D.N., Harvey, M.J.A. & Lennox, B. (1979) Hormonal in cattle. *Journal of Dairy Science*, **68**, 1568–78.

and histological studies in a 61 XXY bull. *Veterinary*

Hillers, J.K., Thonney, S.C. & Gaskins, C.T. (1982) *Economic Record*, **104**, 500–503.

comparison of breeding dairy cows artificially versus natu-

Logue, D. & Isbister, J. (1992) Bull infertility. *In Bovine fertility. Journal of Dairy Science*, **65**, 861–5.

Medicine (ed. by A.H. Andrews, R.W. Blocrey, H. Boyd & Hopkins, F.M. & Spitzer, J.C. (1997) *The New Society for The R.G. Eddy*), p. 483. Blackwell Scientific Publications,

riogenology Breeding Soundness Evaluation System. *The London.*

Veterinary Clinics of North America, Food Animal Practice, Long, S.E. & Dubra, C.R. (1972) Incidence and relative clinical significance of preputial eversion in bulls. *Veterinary Record*, **91**, 283–93.

cal significance of preputial eversion in bulls. *Veterinary Record*, **91**, 165–9.

fication of dairy herds as free of paratuberculosis using

McCauley, A.D. (1980) Seminal vesiculitis in bulls. *In Current culture of*

strategically pooled faecal samples. In Proceed-Therapy in Theriogenology: Diagnosis, Treatment and Pre-

ings of the Sixth International Colloquium on Paratubercu-

vention of Reproduction Diseases in Animals (ed. by D.A.

losis (ed. by E.J.B. Manning & M.T. Collins), pp. 55–8.

Morrow), pp. 401–405. W.B. Saunders, Philadelphia,

International Association of Paratuberculosis, Madison.

London, Toronto.

Kilgour, R. (1984) Sexual behaviour in male farm animals. In

McGowan, M., Galloway, D., Taylor, E., Entwistle, K. &

Male in Farm Animal Reproduction (ed. by M. Courot), pp.

Johnston, P. (2002) The veterinary examination of bulls.

108–32. Martinus Nijhoff, Dordrecht.

Cattle Practice, 10, 55.

Kilkenny, J.B. (1978) Reproductive performance of beef cows.

Meischke, H.R.C. (1979) A survey of bovine teat papillo-

World Review of Animal Production, 14, 65–74.

matosis. Veterinary Record, 104, 28–31.

*Kumi-Diaka, J. & Dennis, S.M. (1978) The Sertoli cell index as Melrose, D.R.,
Brinley Morgan, W.J., Stewart, D.L. &*

a measure of testicular degeneration in the bull. Veterinary Thomson, D.M.

(1957) The treatment of Vibrio fetus

Record, **103**, 112–14.

infected bulls. *Veterinary Record*, **69**, 691–3.

La Faunce, N.A. & McEntee, K. (1982) *Experimental*

Memon, M.A., Dawson, L.J., Vsenik, E.A. & Rice, L.E.

Mycoplasma bovis vesiculitis in the bull. Cornell Veterinar-

(1980–85) Preputial injuries in beef bulls: 172 cases. Journal **ian**, **72**, 150–67.

of the American Veterinary Medical Association, **193**, 481–5.

626 • Chapter 38

Miller, D.J. & Hunter, A.G. (1987) *Individual variation for in bulls: correlation with environmental temperature. Veteri-vitro fertilization success in dairy bulls. Journal of Dairy nary Record*, **120**, 479–82.

Science, **70**, 2150–3.

Robertson, I.S., Fraser, H.M., Innes, G.M. & Jones, A.S. (1982) *Mobini, S., Walker, D.F. & Crawley, R.R. (1982) An experi-Effect of immunological castration on sexual and produc-*

mental evaluation of the response of the bull penis to

tion characteristics in male cattle. Veterinary Record, **111**, carbon fibre implants. *Cornell Veterinary Journal*, **72**, 529–31.

350–60.

Salisbury, G.W. & Van Demark, N.L. (1961) *Physiology of Monke, D.R. (1980) Avulsion of the bovine penile epithelium*

Reproduction and Artificial Insemination. W.H. Freeman, at the fornix. In Proceedings of the 8th Technical Conference San Francisco, London.

on Artificial Insemination, pp. 48–65. National Association Stott, A.W., Veerkamp, R.F. & Wassell, T.R. (1999) The eco-of Animal Breeders.

nomics of fertility in the dairy herd. *Animal Science*, **68**, O'Connor, M.L., Gwazdauskas, F.C., McGilliard, M.L. &

1148–54.

Saake, R.G. (1985) *Effect of adrenocorticotrophic hormone*

Turnbull, P.A. (1977) *An abattoir survey of bull genitalia.*

and associated hormonal responses on semen quality

Australian Veterinary Journal, **53**, 274–9.

and sperm output of bulls. Journal of Dairy Science, **68**, Vincent, C.K. (1972) *Effects of season and high environmen-151–7.*

tal temperature on fertility in cattle. Journal of the

O'Lamhna, M. & Roche, J.F. (1983) *Effect of repeated implan-American Association of Veterinary Medicine*, **161**, 1333–8.

tation with anabolic agents on growth rate, carcass weight,

Walker, D.F. (1980) *Genital surgery of the bull. In Current testicular size and behaviour of bulls. Veterinary Record, Therapy in Theriogenology: Diagnosis, Treatment and Pre-113*, 531–4.

vention of Reproductive Diseases in Animals (ed. by D.A.

Palmer, A.C., Blakemore, W.F., Barlow, R.M., Frazer, J.A. & Morrow), pp. 370–401. W.B. Saunders, Philadelphia,

Ogden, A.L. (1972) *Progressive ataxia of Charolais cattle*

London, Toronto.

associated with a myelin disorder. Veterinary Record, **91**, Weaver, A.D. (1997) *Joint conditions. In Lameness in Cattle* 592–4.

(ed. by P.R. Greenough & A.D. Weaver), 3rd edn, pp.

Parkinson, T.J. (1987) Seasonal variation in semen quality in 162–80. W.B. Saunders, Philadelphia.

Chapter 39

Artificial Insemination and Diseases

Transmitted by Semen

G.H. Wentink

Introduction

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the ejaculate. Extenders are applied to improve the

Semen processing

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buffering action of the media surrounding the sperma-

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tozoa for protection against toxic metabolic substances

Disease transmissible by semen (and/or mentioned

(mainly lactic acid), and secondly to increase the

in trade certificates)

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volume in order to serve as many cows as possible with

Semen safety

one ejaculate.

Between 300 and at least 1000 straws can be produced from one ejaculate, depending on the concentra-

Introduction

tion needed in one dose. Fertility after insemination is

often expressed as the non-return rate (NR): the per-

Large scale artificial insemination (AI) for cattle was

centage of cows that are not rebred within a certain

started in Russia at the beginning of the twentieth

period (usually 56 days). This information can be used

century (1909) by the stimulating impetus of Professor

to optimize the concentration of spermatozoa per dose.

Ivanovich Ivanov. Verbal history says that it was used

Each dose is delivered in a straw (0.25 ml in Europe,

for genetic improvement, but mainly to avoid transmis-

0.5 ml in the USA) containing on average 15 × 10⁶

sion of venereal diseases. These diseases were trans-

sperm cells. However, fertility results differ between

mitted from infected cows to cows on different farms

bulls. The average NR in the Netherlands is 68 per cent;

by travelling bulls used for natural service. Using some bulls score above and others below the average. extended (diluted) semen the great number of offspring. Thus the semen concentration of each bull is altered to by one bull also produced more information on its produce the average NR and thereby allow an optimal breeding value. The main goal of AI is to improve the number of cows to be served and become pregnant with genetic potential of cattle, and considerable progress each ejaculate. Fertility of bulls can only be judged after has been made since its applications.

insemination based on NR. The criteria in the labora- Semen is shipped all over the world, and conse- tory do not have predictive value for fertility: very often quently must be free from infectious diseases. Relevant disappointing results are obtained with semen that was regulations apply in all parts of the world, but may considered of excellent quality in the laboratory, while differ slightly between countries. The EU regulations conversely excellent fertility has been obtained after concerning semen production are laid down in Direc-

natural service with bulls that did not fulfil the standard 88/407; semen imported into the EU from other dards for AI bull approval.

countries must also conform to this standard.

Semen is only safe beyond doubt if produced by bulls that were proved to be free of infectious diseases 21

Technique of artificial insemination

days after its production. International regulations current at the start of the twenty-first century do not The technique in itself is easy to perform, cheap and fulfil this requirement.

applied world-wide rather uniformly by technicians and veterinarians. Insemination is routinely performed by the recto-vaginal technique. One arm is introduced into

Semen processing

the rectum of the restrained cow in heat, grips the cervix and stretches the vagina. After careful cleaning of the After collection and application of extenders (specific vulval area, the AI gun is inserted into the vagina and diluents), semen must be cooled to 5°C over a period through the cervix for the deposition of three quarters

of 2–5 hours before freezing in liquid nitrogen. During of the semen into the corpus uteri (uterine horns are this period metabolism of the spermatozoa continues too deep) and, after limited withdrawal of the AI gun, and metabolic products are excreted into the liquid of the remaining quarter is deposited in the cervix at the

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first insemination after parturition. At the second or discharge and abortion in up to 30 per cent of pregnant higher number of inseminations the semen must be animals. The disease has a high morbidity but a low deposited in the cervix only, because a limited number mortality. However, the disease might follow a more of cows show signs of heat in the presence of an embryo. severe course if complicated by bacterial infections.

Introduction of the gun into the corpus uteri might IPV is characterized by erosion in the vagina or on the interrupt the integrity of the embryo and thus interfere penis, leading to adhesions. The disease is caused by with pregnancy.

bovine herpes virus type 1 (BHV1) which can be sub-

The proper time for insemination is in the last period divided in strains with preference for the respiratory or of the standing heat. Cows are selected for insemination genital tracts respectively. Diagnosis is made by demonstration by observation of the herd at least twice daily stration of the virus in secreta from respiratory and/or during moments of quiescence: observation during genital tracts up to 14 days after infection, and by serology. However, sensitivity and specificity of serological periods of even limited excitation (for example during collecting for milking) can lead to the insemination of tests applied in various countries differ considerably. cows not in heat and thus to disappointing fertility

The agent may survive in favourable conditions (high results. Several aids have been applied to improve heat relative humidity, low temperatures) for 30 days in the detection such as electronic pedometers, heat mount environment. BHV1 is easily transmitted by semen. AI detector pads, spotter bulls, etc. However, careful centres in the EU must be free of IBR: only serologi-

observation of the herd and proper records of heat
cally negative animals are allowed as BHV1 infections
periods undoubtedly form the backbone of good fertil-
lead usually to latency. Latently infected animals are
ity results (see Chapter 41b). Harmful side effects of
the source for new epidemics. Latently infected animals
improper insemination are infections and/or injuries of
are serologically positive for BHV1, but some seroneg-
the genital tract, which are dealt with in Chapter 36.
ative latently infected cows have also been reported.
However, the sensitivities of the tests applied in these
animals are doubtful. Straub (see Horzinek, 1990)

Diseases transmissible by semen

described seronegative latently infected animals after
(and/or mentioned in

the application of a one hour incubation serum neu-
tralization test with a limited sensitivity: the animals
trade certificates)

would probably have been positive with sensitive tests.

It is thought that seronegative latently infected animals

Table 39.1 summarizes the diseases in this category.

develop during the period of maternal immunity after an infection with a very low dose of BHV1 and they

Foot-and-mouth disease (FMD) (see also p. 700)

should exist in very low prevalence in endemic areas.

However, Switzerland, Denmark and Austria became

This is a highly contagious disease of cloven-hoofed

free of IBR: these countries eliminated the virus from

animals characterized by high fever followed by vesi-

their cattle populations without unexpected disease

cles on the mucosa of mouth and tongue, the feet and

outbreaks caused by serologically negative latently

the udder. FMD is caused by a picornavirus comprising

infected animals. The straightforward isolation of posi-

seven serotypes; infection with one serotype does not

tive animals was successful. So the problem of serologi-

induce immunity to other serotypes. In order to prevent

cally negative latently infected animals is very limited

spread of the disease in a country immediate recogni-

or even non-existent.

tion of the clinical picture is essential. For a positive

diagnosis the demonstration of FMD viral antigen by

indirect sandwich ELISA techniques, preferably in the Bovine virus diarrhoea (BVD) (see also pp. 578, 853) epithelium of unruptured or freshly ruptured vesicles, BVD is observed clinically as transient diarrhoea is sufficient. The agent is very resistant to environmental factors and may be spread over long distances by air 5 per cent of infected animals; in exceptional cases and/or vehicles and/or man. FMD virus is easily transmitted by severe diarrhoea, fever, ulceration of the buccal mucosa, haemorrhages and death occur. The remaining 95 per cent of the infections pass unnoticed. When infected in the first four months of pregnancy, cows may deliver persistently infected (pi) calves that shed the virus during their lifetime. The disease is caused by the pestivirus bovine virus diarrhoea virus (BVDV).

severe diarrhoea, fever, ulceration of the buccal mucosa, haemorrhages and death occur. The remaining 95 per cent of the infections pass unnoticed. When infected in the first four months of pregnancy, cows may deliver persistently infected (pi) calves that shed the virus during their lifetime. The disease is caused by the pestivirus bovine virus diarrhoea virus (BVDV). This is a disease of the upper respiratory tract or of the genital tract, respectively. IBR is characterized by

Bovine virus diarrhoea is endemic world-wide. Diagnostic signs such as high fever, nasal discharge, ocular nosis is by antigen ELISA or culture of the virus from

Table 39.1

The main characteristics of infectious diseases that could be transmitted by semen or artificial insemination.

Disease

Incubation

Reservoir

Excretion mainly

Period of

Transmission

period

by

transmission/

by semen/AIa

viraemia

FMD

2–8 days

Animals in the acute

Saliva

<14 days

++

phase

Other secreta

Carriers for

Chronic carriers

up to 2 years

(cattle)

IBR/IVP

2–5 days

Latently infected

Nasal discharge

2–20 days

++

animals

Rinderpest

4–15 days

Cattle in the acute

All secretions and

21 days or

++

phase

excretions

longer

BVD

2–15 days

Persistently infected

Saliva

Variable, usually

++

(pi) cattle

All secretions and

2–15 (but up to

Cattle up to 56 post

excretions of pi

56) days

infection

animals

MCF

From a few

Sheep during

Unknown

Unknown,

0

days to

lambing period

not cow to

year(s)

cow

Akabane virus

??

Biting insects

Viraemia

Viraemia

0

during

prolonged

periods

Bluetongue

3–6 days

Biting insects

Viraemia

Viraemia,

0

mostly <14

days

Enzootic

Up to 35 days

Chronically infected

Intact blood

During lifetime

0

bovine

cattle

Lymphocytes

after infection

leukosis

(EBL)

Genital

<3 days

Chronically infected

Copulation

During lifetime

++

campylobacteriosis

cattle: bulls without

after infection

symptoms

Bovine

14–120

Chronically (latently)

Vaginal discharge

Prolonged

+

brucellosis

days

infected animals

Fetal membranes

periods

Bovine

>3 weeks

Chronically infected

Nasal discharge

During lifetime

+

tuberculosis

cattle

after open

tuberculosis

Leptospira

<7 days

Chronically infected

Urine

Prolonged

+

hardjo

cattle

periods after

infection

Johne's

Occasionally Chronically

infected

Faeces

From

18

0

disease

12; usually 24

cattle

months before

months

overt clinical

disease

onwards

Mycoplasma

2–6 weeks

Chronically infected

Nasal discharge

During lifetime

+

mycoides

cattle

Urine

after infection

(contagious

bovine

pleuropneumonia:

CBPP)

Haemophilus

??

Harboured in genital

Nasal discharge

During lifetime

+

somnus

and respiratory tracts

Effluents from

after infection

in healthy animals

genital tract

Genital

<3 days

Chronically infected

Copulation

During lifetime

++

tritrichomoniasis

cattle: bulls without

after infection

symptoms

Query-fever

??

Ticks

Fetal membranes

Prolonged

0

(Q-fever)

Environment (dust)

Vaginal discharge

periods

Chronically infected

cattle (animals)

Bovine

4 years

CNS of

Contaminated food

No

0

spongiform

contaminated transmission

encephalopathy cattle

reported

(BSE)

a 0, unlikely; +, possible; ++, easily.

??, no experience and no information found in the literature.

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the peripheral blood or organs after death, and by serol-

can occur via the semen. The virus survives in the

ogy. Persistently infected animals transmit the virus

environment for limited periods.

during their lifetime and infect sentinel animals easily.

Transiently viraemic animals, however, transmit the

Malignant catarrhal fever (MCF) (see also p. 935)

disease to a limited extent. These primary infections

might lead to prolonged periods of viraemia (up to 40

MCF is a sporadically, almost invariably fatal disease of

days), the presence of virus in bronchoalveolar wash-

cattle of all ages. The disease is characterized by high

ings (up to 56 days after infection) and in semen even

fever, bright red coloration of all mucous membranes,

after seroconversion has taken place. Therefore, the risk enlarged superficial lymph nodes and very often diarrhoea. A herpes virus (BHV3) causes the disease.

exceptional bull has been described that shed BVDV

Diagnosis in live animals is by clinical signs, and by PCR

in the semen over prolonged periods (11 months),

on heparinized blood. Culture of the virus is not possible,

although the animal was serologically positive. This

ble, therefore information on the resistance of the virus

unusual pattern might be explained by a primary infection

against environmental influences is not available. Cow

tion during puberty or an intrauterine infection around

to cow transmission has never been reported. Transmission

the time of maturation of the immune system. There is

mission by semen is therefore very unlikely.

information that this phenomenon might occur more

frequently. The agent survives for some days in the environment.

Bluetongue (BT) (see also p. 691)

ronment. Transmission by semen is easy. AI centres

only accept bulls that are not persistently infected.

*BT occurs mainly in small ruminants but may occur in
However, because some infectious routes for the intro-
cattle as well. If clinically overt (many infections pass
duction of BVDV are not completely understood, in
unnoticed in cattle) the disease is characterized by
endemic areas BVDV might infect bulls in an AI
fever, facial oedema, haemorrhages and ulceration of
station. Where there is unintended infection of AI bulls
the mucous membranes. An orbivirus consisting of 24
the infection passes subclinically and, as this disease is
serogroups causes the disease. The virus is identified by
easily transmitted by semen, it is very important to test
culture and by PCR techniques on blood samples of
for BVDV. Monthly tests for serologically negative
febrile animals. The agent is transmitted by insects of
donor bulls and, in the case of seroconversion, ensur-
the genus *Culicoides*; transmission from cow to cow
ing the semen is free from BVDV might be the only
has not been reported. Transmission by semen is very
solution.
unlikely.*

Enzootic bovine leukosis (EBL) (see also p. 693)

Akabane virus (see also p. 580)

*EBL is found clinically by enlarged lymph nodes or
Infections of Akabane virus were shown to cause sporadic lymphosarcomas, or by lymphocytosis in the
radic epizootics of premature births and developmental peripheral blood in the minority (some 30 per cent) of
tal deformities in the new born (arthrogryposis-infected animals. Infections for the greater part pass
hydranencephaly) in cows, sheep and goats. The disease
completely unnoticed. The disease is caused by the
has been reported in Southeast Asia, the Arabian
retrovirus bovine leukemia virus (BLV), which may be
Peninsula and the Middle East and African countries.
demonstrated in the blood by PCR techniques. Routine
Diagnosis is done retrospectively by serology in adult
diagnosis is performed by serology (ELISA, AGID).
animals, and by culture techniques in abnormal fetuses
The agent does not survive in the environment. Only
and calves. Biting insects transmit the agent. After
intact lymphocytes are infective: the infection is trans-*

*experimental infection in bulls, Akabane virus was not
mitted by blood. Transmission by semen is very
demonstrated in the semen; transmission by this route
unlikely.*

is therefore unlikely.

Rinderpest (see also p. 707)

Bovine genital campylobacteriosis (see pp. 568, 582)

This is a very contagious disease of mainly cattle and

This is a venereal disease characterized by infertility,

buffaloes. The disease is characterized by high fever,

early embryonic death and abortion. In bulls, infections

erosions on the gums, tongue and palate together with

are inapparent. The disease is caused by Campylobac-

nasal and ocular discharge, and diarrhoea. Rinderpest

*ter fetus ssp. venerealis, which in bulls can be cultured is caused by a Morbelli
virus that can be demonstrated*

from preputial washings. Serological methods are unre-

in blood or lymph nodes and spleen, mainly by culture.

liable. The organisms survive in the environment for

Transmission is mainly directly by infected animals and

limited periods (hours). Mating and/or semen mainly

cause transmission. Virgin bulls are absolutely free of and may survive for several months and transmission this agent. This agent was the impetus for the world-by semen is possible. Tuberculosis is a zoonosis.

wide application of AI techniques and for the obligatory addition of antibiotics to the semen. If this

Leptospirosis (see also p. 735)

procedure is properly executed, semen is free from this bacterial agent. Bull stations should be free of this

Leptospirosis is a contagious disease of animals and agent, but the low transmission rate makes testing more

humans. Many Leptospira species exist, each with their often than once a year useless. However, when a posi-specific carrier host (reservoir) in which infection leads

tive result is obtained, all semen batches after the last to diseases of limited severity and prolonged excretion.

negative result should be checked for the presence of

Infection in other animal species (end hosts) leads to the agent and positive ejaculates destroyed.

very severe, life threatening disease symptoms. Clini-

A very similar agent, Campylobacter fetus ssp. fetus, cally, infections in

susceptible end hosts are characteris associated with abortion storms in sheep. In cattle,

ized by fever, icterus, haemorrhages, uraemia and blood after oral uptake, this agent infects the gastrointestinal tinged urine. After recovery, the Leptospira are contract and is shed by the faeces. In exceptional cases, oral pletely eliminated from the end host's body and trans-infections in adult pregnant cattle may lead to bacter-mission by semen is very unlikely. Infections in the aemia and in limited numbers to infection and expul-carrier hosts pass with minor clinical problems and may sion of the fetus. This agent is not transmitted even pass unnoticed.

by mating or semen. Giacoboni et al. (1993) isolated Cattle are carriers for Leptospira hardjo, but end Campylobacter fetus ssp. fetus from the faeces in 26.5 hosts for other Leptospira species. Infection with L. per cent of young calves, and in 15 per cent of older hardjo in cattle leads to prolonged or intermittent cattle. Excretion by the faeces leads inevitably to con-excretion with urine (and semen) and therefore may be

amination of the environment and thus to the risk of transmitted by semen. *Leptospira* may survive in the contamination of the prepuce. In The Netherlands, environment in humid and warm conditions (summer)

Campylobacter fetus ssp. *fetus* was isolated from the for longer periods. Attention in AI stations should be

prepuce of AI bulls ranging in age from 10 months to

concentrated on *Leptospira hardjo* infections. *L. hardjo* 5 years that had never served naturally.

is a zoonosis and causes milker's fever in man.

Brucellosis (see also p. 580)

Contagious bovine pleuropneumonia (CBPP)

(see also Chapter 49b)

This is manifested by abortions in the last third of pregnancy. The causative organisms are excreted in

CBPP is characterized by fever, dyspnoea, coughing, abundance with uterine discharges and with milk. The nasal discharges and anorexia. It is caused by

disease, which is a zoonosis, is caused by *Brucella* (B.)

Mycoplasma mycoides ssp. *mycoides*, which can be abortus, occasionally by *B. melitensis* (lower numbers of demonstrated in nasal secreta and bronchoalveolar aborting cows in the herd) and exceptionally by *B. suis*.

washings or in pleural fluids collected after puncture

The disease is diagnosed by culture of the causative

by culture in appropriate media. The agent is vulnera-

organism from uterine discharge and milk, by serology

ble for environmental influences and is transmitted by

(CFT, BUA, ELISA) or by a specific skin test. The

infective animals over limited distances. Transmission

organisms survive in the environment in favourable

by semen is possible.

circumstances for prolonged periods. Transmission by

semen is possible.

Johne's disease (paratuberculosis) (see also p. 857)

This disease is chronic enteritis of ruminants, charac-

Bovine tuberculosis (see also p. 862)

terized in cows by severe diarrhoea, emaciation and

This passes unnoticed in the early phases after infec-

submandibular oedema. It is caused by *Mycobacterium*

tion, but in advanced cases emaciation, coughing and

(*M.*) *avium* paratuberculosis, which only leads to clini-enlargement of the lymph nodes develop. It is caused

cal disease and excretion after infection at a very young

by *Mycobacterium (M.) bovis* and occasionally by *M. avium*. Infection leads to progressive thickening of the gut wall, causing a protein losing enteropathy with diarrhoea in the final stages after 24 months or longer. *M. avium* in live animals is diagnosed by a specific skin test, gamma-interferon assay using peripheral blood lymphocytes, and by culture from nasal secretions.

In the clinical stages, the agent can be demonstrated

The organism is resistant to environmental influences in faeces and exceptionally in milk and the fetus by

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culture and/or PCR techniques. The use of PCR on the diagnosed, no treatment is available and the bull and its faeces lacks sensitivity. For the detection of subclinically infected animals a skin test, ELISA and CFT are applied. Each method has the disadvantage of limited

Bovine spongiform encephalopathy (BSE)

specificity and sensitivity.

(see also p. 909)

Transmission by contaminated semen or semen from contaminated bulls has never been demonstrated.

BSE is a fatal neurological disease of cattle with a long incubation period. Clinically, behavioural changes and locomotion disturbances such as ataxia manifest the systems with low sensitivity lead inevitably to the introduction of infected calves into the AI centres. However, source of infection. After (oral) uptake of abnormal protein some of the body protein is stereometrically excretion by the faeces, and shedders can be traced by altered, making the protein invulnerable to proteases the application of annual faecal culture tests from the age of two years. Furthermore, there is no evidence that

This stereometrical change leads to death of brain cells, contaminated semen infects cows after insemination. resulting in the aforementioned. No diagnostic tests for Johne's disease might be a zoonosis (Crohn's disease in live animals are available, although some promising man).

results have been reported. The prions are extremely resistant to environmental factors. BSE prions have never been demonstrated in the genital organs of diseased bulls or in the semen and experimental insemination of susceptible cows with semen from Haemorrhagic septicaemia (HS) (see also p. 728) HS is a highly fatal disease characterized by initial fever, respiratory signs and terminal septicaemia BSE-contaminated bulls has not been shown to induce leading to recumbence. It has been reported from Asia BSE in the cows or progeny. There is no proof that and Africa, and is caused by certain strains of Pasteurella multocida. The agent can be cultured from risk of BSE transmission by semen is thought to be neg-

blood and from internal organs. *P. multocida* is moderately resistant to environmental influences. Transmission by semen is very unlikely.

at least partially resistant to environmental influences. Transmission should be minimal or none. BSE is considered to be a zoonosis (new variant CJD).

Query fever (Q-fever) (see also p. 586)

Other infectious agents

In cattle, Q-fever is characterized by abortion, retained

Ubiquitous agents that might be or are present in

placenta, metritis and infertility. The disease is caused

semen, e.g. *E. coli*, *Proteus* spp., *Pseudomonas* spp., by *Coxiella (C.) burnetti*.

Diagnosis is established by *Haemophilus* spp., *Campylobacter* spp. except *C. fetus* microscopy of fetal membranes or aborted fetuses, or

spp. venerealis, *Mycoplasma* spp., and *Ureaplasma* spp., by culture of this agent in embryonated eggs from

should not hinder the semen trade: the addition of

uterine effluents. Routine diagnostic tests are made by

appropriate antibiotics after proper procedures is a suf-

serology (CFT, ELISA). The agent is extremely resist-

ficient guarantee for safety.

ant to environmental influences. Transmission is caused

by biting insects or by inhalation of contaminated dust.

Transmission by semen is limited or unlikely. Q-fever is

Semen safety

a zoonosis.

For safety testing of semen, two approaches can be applied: examination of the end product or continuous

Bovine genital tritrichomoniasis (see also p. 584)

surveillance of the bulls before and after semen pro-

This is characterized by infertility and abortion. Bulls

duction. The tests for the presence of infectious agents

may be infected without clinical signs. The disease is

in the semen completely depend on one single investi-

caused by the protozoan Tritrichomonas fetus, which

gation and therefore rely on the sensitivity of the test

comprises three serotypes of equal pathogenicity. The

methods only. The continuous surveillance of the semen

agent can be cultured from uterine discharges and

donors for infectious diseases before and after semen

preputial washings. Tritrichomonas is rather resistant to collection is based on sequential investigations, increas-environmental influences. Mating and/or semen mainly

ing the reliability by the application of multiple tests. In

cause transmission. Virgin bulls are free of this agent.

terms of semen safety, testing the donor for the absence

However, if an infection with tritrichomoniasis is

of infectious diseases 21 days after semen collection is

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beyond doubt the best method. Therefore the bulls in

period is short. For diseases with high transmission

AI centres must be negative for the diseases mentioned

rates (e.g. IBR) monthly checks of 20 per cent of the

above. The very first step is to accept only virgin bull

donor bulls give a certainty of 99.9 per cent for semen

calves negative for these diseases. Most AI centres in

safety. This method is applied in one centre in The

Europe that release semen for the EU market collect

Netherlands.

their bull calves before the age of six months. There are

no concerns about diseases from which the area is offi-

cially declared free. An annual check on the health

References and further reading

status will suffice if the country in which the AI centre

is situated remains negative. However, if diseases are

Berchovich, Z. (1998) Maintenance of Brucella abortus free endemic, theoretical

risks exist for the introduction of herds: a review with emphasis on the epidemiology and the disease agents into AI studs. Preventive measures problems in diagnosing Brucellosis in areas of low prevalence. *Veterinary Quarterly*, **20**, 81–8.

entering the premises (showering and changing clothes), on 48-hour storage periods (including forage) bovine virus diarrhoea virus infections. Thesis, Utrecht. or on disinfection of materials before admittance into

Chen, S.S., Redwood, D.W. & Ellis, B. (1990) Control of the station, the use of mains water, and on the eradica-

Campylobacter fetus in artificially contaminated bovine semen by incubation with antibiotics before freezing.

tion of vermin. One risk factor remains, however infectious particles transported by air and/or birds. Only *British Veterinary Journal*, **146**, 68–74.

Council Directive (88/407/EEC) (1993).

large distances (more than 1 kilometre) between the AI

Den Daas, J.H.G. (1997) Prediction of bovine male fertility.

station and other cattle in the area will suffice.

Thesis, Wageningen.

The next step is to minimize the consequences of the

Eaglesome, M.D. & Garcia, M.M. (1997) Disease risks to unintentional introduction of agents into AI centres.

animal health from artificial insemination with bovine

Regular checks must be performed for the presence of semen. Revue Scientifique Office International des Epi-disease agents that might be transmitted by semen:

zooties, 16, 215–25.

animals found positive must be separated or destroyed

Giacoboni, G.I., Itoh, K., Hirayama, K., Takahashi, E. & immediately. The system for checking the disease-free

Mitsuoka, T. (1993) Comparison of fecal Campylobacter in status of the bulls depends on the transmission routes

calves and cattle of different ages and areas in Japan.

Journal of Veterinary Medical Science, 55, 555–9.

and transmission rate. Highly contagious diseases with

Herman, H.A., Mitchel, J.R. & Doak, G.A. (1994) The a high risk for transmission by semen should be checked

Artificial Insemination and Embryo Transfer of Dairy and

regularly (monthly). The rates at which diseases are

Beef Cattle, 8th edn. Interstate Publishers, Danville.

transmitted to other animals depend on a number of

Horzinek M.C. (1990) *Virus Infections in Vertebrates*. In *Viral characteristics of the pathogen, the host and of the*

Infections in Ruminants. (ed. by Z. Dinter & B. Morein) contact structure between animals. An important

Elsevier; Amsterdam, Oxford, New York, Tokyo.

parameter related to transmission is the basic repro-

Merkal, R.S., Miller, J.M., Hintz, A.M. & Bryner, J.H. (1982) duction ratio, i.e. the number of animals that are

Intrauterine inoculation of *Mycobacterium paratuberculosis* infected by one typical infectious animal during its

into guinea pigs and cattle. *American Journal of Veterinary entire infectious period*. This number depends greatly

Research, **43**, 676–8.

on the number of effective contacts between animals. A

OIE (1996) *OIE Manual of Standards for Diagnostic Tests and Vaccines*, 3rd edition. Office International des Epizooties, contact is effective when the pathogen is transmitted

1997. ISBN 92-9044-423-1.

from one infected animal to another (excretion titre

Thrusfield, M. (1995) *Veterinary Epidemiology*, (2th edn).

above the minimal effective dose). These contacts

Blackwell Science, Oxford.

might be either direct (animal–animal) or indirect (by

Wentink, G.H., Frankena, K., Bosch, J.C., Vandehoek, J.E.D.

air, people, equipment, etc.). High transmission rates in

& van den Berg, Th. (2000) Prevention of disease transmis-

AI stations will be achieved when the number of effec-

sion by semen in cattle. Livestock Production Science, 62, tive contacts is high and the length of the incubation

207–20.

Chapter 40

Embryo Transfer

A.K. Smith

Introduction

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- *Increased numbers of progeny from genetically*

The principles of embryo transfer

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superior dams.

Results

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- *The possibility of progeny of the other sex from*

Donors

637

dams that previously may have only produced

Recipients

637

calves of one sex.

Embryo collection: practicalities

638

- *Increased diversity for marketing herd genetics*

Donor selection criteria

638

including international trade.

Nutrition

638

Superovulation

638

- *Banking of frozen embryos to accommodate sea-*

Insemination

639

sonal breeding or longer term breeding strategies.

Embryo collection

641

- *As a diagnostic technique for infertility cases,*

Embryo processing

642

especially in diagnosing blocked oviducts.

Embryo freezing

642

- *As a method of continuing breeding from animals*

Embryo thawing

643

unable to do so naturally.

Embryo transfers: practicalities

643

- *Improved disease control applicable to high health*

Recipient selection criteria

644

status herds and international trade by reducing the

Nutrition and management

644

number of individual animal movements.

Disease control

644

Oestrous synchronization

644

Transferring the embryo

644

Hormone supplementation

645

Record keeping

645

The principles of embryo transfer

Legislation

645

Troubleshooting

646

As previously described (see Chapter 33), follicular

Advanced technologies

646

dynamics result in either two or three waves of follicle

Embryo splitting

646

development during the oestrous cycle. In each wave a

In-vitro fertilization (IVF)

647

dominant follicle suppresses the development of sub-

Ovum pick-up (OPU)

647

ordinate follicles until at the end of the cycle the corpus

Embryo sexing

648

luteum (CL) is lysed, allowing the dominant follicle of

Cloning

648

the last wave to become the ovulatory follicle.

Superovulation utilizes commercially prepared folli-

Introduction

cle stimulating hormone (FSH) to encourage a group

of developing follicles to overcome the dominant folli-

Although considered a recent technology, Walter Heap

cle suppression and continue developing to ovulation

performed the first successful embryo transfer (ET) in

with concomitant maturation of the oocytes. The FSH

rabbits over a hundred years ago. A further six decades

is commonly administered as a twice-daily injection elapsed before the first embryo transfer calf was produced in 1951, in the USA, by Willet and his colleagues. 9–14). Incorporated in this regime is a prostaglandin F2a (PGF2a) injection, usually given alongside the fifth FSH in cattle in 1969 by Rowson and his colleagues. In 1973 Wilmut and Rowson produced the first calf born from of oestrus and subsequent ovulation are accelerated in a frozen-thawed embryo and five years later Willadsen superovulated animals and oestrus occurs usually 48–56 hours after the PGF2a treatment, with ovulation occurring as early as the onset of oestrus (cf 12–18 hours post oestrus in non-superovulated cows). Accordingly it is breeding offering breeders a number of opportunities: usual to inseminate the donor cow soon after the onset

of oestrus (e.g. the afternoon of the day of oestrus, with

Box 40.1

Description of embryo grading. Source: IETS

a second insemination the following morning).

Manual, 3rd edn (Stringfellow & Seidel, 1998).

Embryo collection is performed on day 7 of the oestrous cycle (day 0 = day of oestrus). Embryos enter Grade 1: Symmetrical and spherical embryo mass with individual blastomeres (cells) that are uniform in size, colour and density. The embryo is consistent with day 4 of the oestrous cycle and, although embryos can be collected successfully from the uterus between days 5 and 9, a day 7 collection allows recovery of the most of the cellular material should be an intact, viable flexible stages of the embryo for both freezing and fresh embryonic mass. This judgement should be based

transfer. Collection (flushing) is a non-surgical procedure requiring catheterization of the uterine horns. by the extruded material in the perivitelline space.

Two types of embryo collection catheter exist:

The zona pellucida should be smooth and have no concave or flat surfaces that might cause the

- Two-way, which has two tubes within it. One allows embryo to adhere to a petri dish or a straw.

inflation of a latex cuff near the tip of the catheter,

Grade 2: Moderate irregularities in overall shape of the whilst the other enables the flushing media to enter embryonic mass or in size, colour and density of and leave the uterine horn. Greater volumes of individual cells. At least 50 per cent of the cellular media are required as a larger segment of the material should be an intact, viable embryonic uterine horn is flushed (Fig. 40.2).

mass.

- Three-way, which consists of three tubes. One allows

Grade 3: Major irregularities in shape of the embryonic mass

or in size, colour and density of individual cells. At inflation of the cuff situated 10 cm behind the tip.

least 25 per cent of the cellular material should be

Another tube allows media to enter the uterine an intact, viable embryonic mass.

lumen from the tip of the catheter and a one-way flow is created by the media returning down the third tube via its entry hole at the level of the cuff (Fig. 40.3).

injury, particularly when cooling is slower than

The flushing media, Dulbecco's phosphate buffered optimum.

saline (PBS), is infused into the uterine lumen to allow

- Intracellular ice: this results in physical damage slight distension before drainage by gravity flow along when cooling is faster than optimal.

with the embryos and ova into a collection filter. The filter contains a metal or plastic micromesh with a pore

Cooling rates of $0.3^{\circ}\text{C}/\text{min}$ to $0.6^{\circ}\text{C}/\text{min}$ are considered diameter approximately half that of an embryo

rapid whereas $0.1^{\circ}\text{C}/\text{min}$ is considered slow. The cryo- (embryo diameter 140–170 μm), allowing excess media

protectants lower the temperature at which intracellular ice forms, reduce the amount of ice present in

Embryo searching is performed once both uterine extracellular fluid, moderate the solute effect and help horns have been flushed. The contents of the filter are stabilize the cell membrane. Some cryoprotectants emptied into a petri dish and examined under a stereomicroscope at between $\times 6$ and $\times 10$ magnification.

to be added or removed gradually. The cryoprotectants Recovered embryos are placed in embryo holding most commonly used commercially are 10 per cent solution (Dulbecco's PBS with bovine serum albumin, glycerol and 1.5 M ethylene glycol. Stepwise addition or glucose and antibiotics), classified (Fig. 40.1; Plate 40.1) removal is required for 10 per cent glycerol in increasing and graded (Box 40.1). Embryo holding solution is also known as ovum culture medium (OCM). Embryo evaluation, whereas one-step thawing of embryos frozen in

uation is by subjective visual assessment under $\times 50$ ethylene glycol can be performed as it crosses cell stereoscopic magnification. Embryos can survive on the membranes more rapidly. Embryos are frozen in bench at ambient temperature (20°C) for up to eight 0.25 ml plastic straws which must be identified with the hours with little drop in pregnancy rate, but they are donor name, sire name, collection team code, breed best frozen or transferred within three hours of collection code and date code. The straws are sealed either by a tion (Wright, 1985).

plastic plug or are heat-crimped.

Embryo freezing involves, initially, partial dehydration. The cooling curve to be used is preprogrammed into the freezing machine and the straws are subjected to a controlled rate of freezing as this progresses to ensure optimal survival of the embryo. A process called 'seeding' is performed when the freezer chamber tem-

- *Solution effects: the solute concentration within a perature is at -6°C (some programmes -7°C). This cell increases due to dehydration and causes cell nucleates the freezing medium with an ice crystal when*



(1) 1-cell

(4) Late morula

(day 1)

(day 6)

(2) 2-cell

(5) Early blastocyst

(day 2)

(day 7)

(2) 4-cell

(6) Blastocyst

(day 3)

(day 7–8)

(2) 8-cell

(7) Expanded blastocyst

(day 4)

(day 8–9)

(2) 16-cell

(8) Hatching blastocyst

Fig. 40.1

The code for stage of develop-

(day 5)

(day 9)

ment is numerical. Number 1 identifies an unfertilized oocyte or a one-cell embryo.

Number 2 identifies embryos with 2 to 16 cells (approximately day 2 to day 5).

Number 3 identifies an early morula, and numbers 4 to 8 (inclusive) identify post compaction stage embryos as illustrated above.

(3) Early morula

(day 5–6)

In commercial embryo transfer, embryos are collected on days 6 to 8 of the oestrous cycle (morula to blastocyst).

1 or 2°C below its freezing point, preventing super-cooled and then they are reloaded into straws for cooling and excessive temperature rise from the release transfer.

of latent heat in the cooling process. At the end of the

The embryo transfer procedure for the recipient is the freezing programme the

straws are plunged directly

*same for fresh or frozen embryos. Recipients need to
into liquid nitrogen before being placed in plastic
have been synchronized to display oestrus on the same
goblets and stored under liquid nitrogen in cryogenic
day as the donor cow or within a 24-hour period either
flasks.*

*side of that day. For frozen embryos the recipients are
Thawing of the straws is a reversal of the previous
synchronized to be in oestrus six, seven or eight days
procedures. The straws are thawed in a flask of warm
prior to the day of transfer. Before transfer of the
water and if frozen in 1.5 m ethylene glycol are then
embryo the recipient's ovaries are examined by manual
ready for immediate transfer. Those frozen in 10 per
rectal palpation or ultrasound scanner for the presence
cent glycerol must first have this extracted from the
of a CL. Note is also made of which ovary has the CL.
embryo. The embryos are removed from the straws they
Recipients receive an epidural anaesthetic and, after
are frozen in and passaged through decreasing concen-*

*the perineum has been cleaned, the transfer gun is
trations of glycerol until finally they are placed in
passed through the cervix and guided along the uterine
embryo holding solution, re-examined, their grades
horn ipsilateral to the corpus luteum before depositing
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Table 40.1

Analysis of results achieved from 1110 commercial

Table 40.2

*Analysis of pregnancy results for 3820 embryos
embryo collections. Source: Ovaflo Embryo Transfer.
transferred commercially. Source: Ovaflo Embryo Transfer.*

Overall results per collection

By classification

Total ova (includes unfertilized ova,

10.08 (mean)

Blastocysts

62.3%

degenerate embryos and viable embryos)

(grade 1)

Total viable embryos

5.03 (mean)

Early blastocysts

62.4%

Fertilised ova

58.6%

(grade 1)

(includes viable and degenerate embryos)

Morulas

62.4%

(grade 1)

By classification

Blastocysts

11.3%

By procedure

Early blastocysts

15.2%

Fresh transfers

64.8%

Morulas

73.5%

(grade 1)

Frozen transfers

58.8%

By grades

(grade 1, 10% glycerol)

Grade 1 embryos

88.0%

Grade 2 embryos

11.7%

By freezing method (purchased embryos) Grade 3 embryos

0.3%

10% Glycerol

53.4%

1.5 M Ethylene glycol

55.4%

By embryo grade

the embryo. Historically embryos were transferred

Grade 1

62.4%

surgically, involving paravertebral anaesthesia and

Grade 2

44.5%

laparotomy of the sublumbar fossa, but for welfare reasons surgical transfers have been superseded by the

By dam breed

non-surgical technique.

Beef

59.3%

(grade 1)

Dairy

64.5%

Results

(grade 1)

Donors

In superovulated animals an estimate can be made of anomalies were found in day 7, 8 or 9 embryos, suggest the likely number of ova by counting the corpora lutea gesting embryos with abnormal karyotypes were non-on the ovaries. Manual estimation is poor if there are functional by that stage. Whilst embryos from donors more than ten CLs on an ovary, but if there are over 15 years old have shown lower pregnancy rates fewer then estimates have a higher accuracy. Some dis-

(Hasler et al., 1987), the biggest single embryo factor crepancies can be accounted for by luteinized follicles

affecting pregnancy rates is embryo quality. Good

which histologically show an enclosed oocyte with the

quality embryos give better pregnancy rates

cumulus cells dispersed and luteinization in the granu-

(Donaldson, 1985). By day 16 well-developed embryos

losa cells (Monniaux et al., 1983). Heifers can exhibit produce significantly more interferon, thus fully inhibit-large responses to superovulation (based on palpated

ing luteolytic PGF2a, than do poorly developed embryos

CLs) with high fertilization rates of the recovered ova

at that stage.

but lower recovery rates than parous animals (Hasler et

al., 1983). Examples of results achieved commercially

Maternal factors

are given in Table 40.1.

The best pregnancy rates are achieved when the syn-

chrony is close (± 24 hours) between the embryo's stage

Recipients

of development and the recipient's uterine environ-

Successful pregnancy rates are influenced by a number

ment (Donaldson, 1985; Newcomb & Rowson, 1975).

of factors, both embryonic and maternal.

Transfer of the embryo to the uterine horn ipsilateral

to the corpus luteum with deep placement of the

embryo, rather than placement at the level of the

Embryonic factors

intercornual ligament, gives the best pregnancy rates

Although some young embryos (days 3–6) have been

(Christie et al., 1979). The CL ensures a source of prog-shown to have abnormal karyotypes (7 per cent) no

esterone but maternal progesterone concentrations can

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show considerable diurnal fluctuations. Progesterone is

this relationship, but since diet supplementation can

required in the first half (approximately 12–14 days) of

raise insulin levels nutrition has an important role.

the oestrous cycle to suppress the oxytocin receptors.

Donors should be on a rising plane of nutrition for six

Inhibition of the oxytocin receptors is less effective in

to eight weeks before flushing. All adjustments must be

cows with a low progesterone concentration, meaning

gradual without any sudden changes. The timing of that the luteolytic mechanism develops earlier allowing housing and turn-out needs to be planned carefully as less time for the conceptus to produce sufficient trophoblastic interferon to create an adequate block to luteolysis. Remsen et al. (1982) and Northey et al. (1985) demonstrated maximal pregnancy rates (PR) when plasma progesterone concentrations were in the range 2–5 ng/ml (PR 74 per cent) and 2–6 ng/ml (PR 68 per cent), respectively. However Hasler et al. (1980) and molybdenum diet (15–20 mg Mo/kg dietary DM) produce greater numbers of abnormal embryos, pregnancy rates and progesterone concentrations. A review suggesting that elevated dietary molybdenum will

of mean pregnancy rates that can be achieved compared to embryo development. In addition, diets containing 5 mg Mo/kg DM decrease conception rates and cause anovulation and anoestrus in cattle. Protection against molybdenum toxicity is achieved by providing adequate supplies of copper. Although copper can be supplied by injection or orally as powdered mineral, Forward planning is the key. Careful animal preparation and attention to detail will improve success.

the use of intraruminal boluses is more efficacious (p. 298).

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the use of intraruminal boluses is more efficacious (p. 298).

the use of intraruminal boluses is more efficacious (p. 298).

Donor selection criteria

Donor heifers:

Superovulation

- Body condition score approximately 21/2 (assessed over the range 1–5; Lowman et al., 1976; see also gonadotrophin (eCG) and human menopausal gonadotro-

Chapter 2);

phin (hMG) have been used in the past to superovulate

- Regular oestrous cycles;

cows, but due to the undesirable effects from extended

- Age 15–18 months.

activity of the luteinizing hormone (LH) fraction they

Donor cows:

have been superseded by purified FSH. This is avail-

able as a freeze-dried product with diluent which,

- At least six weeks calved;

when mixed, produces a solution with controlled

- Clean uterus and returned to regular oestrous

amounts of LH activity. These commercial FSH prod-
cycles;

ucts are sourced principally from ovine and porcine

- Preferably two heats since calving;

pituitaries.

- Maintaining or increasing liveweight (body condi-

It is best to start superovulation from a known refer-
tion score 2–3).

ence heat so that the injections commence at a known

point in the oestrous cycle. A superovulated response can be achieved starting anywhere in the cycle, given

Nutrition

good control of the follicular wave, but the most repeat-

It is now recognized that the time required for a pri-

edly consistent results are achieved when the FSH

mordial follicle to develop to an ovulatory follicle is

injections commence between days 9 and 14 (Hasler et

60–90 days and the environment that the cow is in

al., 1983). This same review found no differences in the during that time can influence not only the vitality of

number of ova (11.6 versus 10.2) or viable embryos (6.2

that oocyte but also the subsequent embryo (Webb et

versus 6.4) recovered from donors superovulated fol-

al., 1999). In particular there is a correlation between lowering prostaglandin-induced reference heats versus

follicular development and plasma levels of insulin and

natural reference heats, respectively. The use of intra-

peripheral levels of insulin-like growth factor-1 (IGF-

vaginal devices to mimic the hormone levels found mid

1), with elevated levels of insulin being associated with

cycle can supply an exogenous source of progesterone.

enhanced ovulation rates. Research continues into
Although less consistent than starting from a known
identifying the mechanisms and pathways involved in
reference heat, the results from this method are satis-
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Table 40.3

Examples of commercial superovulation regime for cattle.

Maiden heifer

Mature cow

day 12

am

2.25 ml FSH solution

3.0 ml FSH solution

pm

2.25 ml

3.0 ml

day 13

am

1.75 ml

2.5 ml

pm

1.75 ml

2.5 ml

day 14

am

1.25 ml + double dose of PGF2a

2.0 ml + double dose of PGF2a

pm

1.25 ml

2.0 ml

day 15

am

0.75 ml

1.5 ml

pm

0.75 ml

1.5 ml

Total dose

12 ml

18 ml

Note:

At the time of publication there is one licensed FSH product in U.K. This is an

ovine FSH (Ovagen, ICP, NZ) containing 0.88 mg/ml NIADDK-oFSH-17 ovine pituitary FSH.

PGF2a = Prostaglandin F2a

These protocols are included as examples of regimes used in commercial embryo transfer practice. Alternative protocols do exist with variations in total volume, volume of individual injections and rate of decline of dosage regime.

factory even if few or no behavioural signs are seen at

is to improve the superovulatory response without

the time of the reference heat.

creating an over-response, because as the number of

When a twice-daily injection regime is used the injec-

follicles stimulated increases there is a negative effect

tion interval is ideally 12 hours, but as long as the inter-

on the viable percentage of the recovered ova. Gener-

val is no less than eight hours satisfactory results will

ally this happens once the total CL count rises above

still be achieved. Product manufacturers advocate that

20. As numbers increase above this level there is a ten-

the total dose be split into eight equal injections, but the

endency for a rise in numbers of unfertilized and degen-

use of a declining regime with higher dose volumes at

erate embryos and a decline in viable embryos, often to

the start gives superior results. Responses to the same zero. However, some donors can still perform successfully with CL counts in the range of 20–30. It is, and there are also breed differences in sensitivity to the treatment with the Simmental breed being particularly sensitive. In addition, body condition, nutrition and the mid teens, which can result in 8 to 12 viable embryos environmental factors will all influence the response to (fertilization rate 50–60 per cent). As Table 40.1 demonstrates real results are lower than this. This can be partly attributed to some donors being consistently successful, heifers will respond satisfactorily to a lower total dosage of FSH than that required for a mature cow. but others being consistently poorer performers. Although aged cows can be successfully superovulated Studies of donor populations corroborate this, but such the oocyte quality may be compromised in these cows,

profiles can only be built up over time and on the especially if over 15 years old, so that fertilization rates strength of a single flush a donor cannot be categorized of these ova or pregnancy rates of subsequent embryos simply as good or bad other than for that single event. may be poorer. An example of a standard commercial Cleanliness at the time of injection is important as programme used for maiden heifers and mature cows contamination of the stock solution can result in loss of is shown in Table 40.3.

potency of the FSH. New sterile syringes and needles Between animals there can be a substantial variation should be used for each injection. In animals that have in their superovulatory response to a given dose of high body condition scores the superovulatory response FSH. What can be an effective dose in one animal may is improved if the injections are given into the neck result in either too low or too high a response in muscle.

another. Animals which fail to respond sufficiently (fewer than two ova is a non-response and fewer than

Insemination (see also Chapter 39)

five ova is a low response) can usually be improved by increasing the dose on a subsequent occasion. Increasing the number of straws of semen recommended can vary the total dose in 2 ml increments (0.25 ml per injection) from operator to operator, but given good quality semen three straws given in two insemination periods

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Balloon cuff

Uterine horn

Oviduct

Superovulated ovary

Cervix

Vagina

Flexible PVC catheter

(catheter stiffened with steel stylet for insertion)

Cuff

Inflation

Fluid outlet

3 way

Tap

Embryo collection

Fluid inlet

filter

Fig. 40.2

Two-way embryo collection

catheter.

are usually adequate, firstly the afternoon of the day the previous experience, the semen is known to be of poor donor is in oestrus and again the following morning.

quality then extra straws may be used (e.g. three

The precise timing of this action can be dependent on sessions utilizing two, two and one straws of semen).

the time of onset of oestrus. If the donor is in standing

A mix of semen from two different bulls can also be

oestrus in the morning then artificial insemination (AI)

used when quality is questionable. It should be con-

at mid afternoon that day with two straws and again the

firmed that the Breed Society rules permit this practice

following morning with one straw is sufficient. If the

and all progeny produced will need to be either blood-

donor only comes into standing oestrus later in the day typed or DNA tested to confirm parentage. Equivocal then the first AI can be delayed. This is easily done if evidence exists as to whether heterospermic interaction in this scenario will synergistically enhance fertilization rates. Published data suggest enhancement in proportion to the size of the superovulatory response a range of 2 to 10 per cent, dependent on whether response. If a cow is in standing oestrus the night before two bulls (same breed) or three bulls (different breeds) were used respectively. Natural service can also be assumed and an extra AI session using an AI gun can be used and the bull should be allowed to serve the additional two straws of semen should be planned donor as soon as she comes into oestrus. Consideration for the morning of the first day of AI. If, through must be given to the health status of bulls used

Uterine horn

Oviduct

Balloon cuff

Drainage holes

Cervix

Superovulated ovary

in catheter

Flexible PVC

catheter

Vagina

Stainless steel

intraducer

Embryo

Fluid inlet

collection

filter

Cuff inflation

Fig. 40.3

Three-way embryo collection

catheter.

for natural service to avoid transmission of venereal

A successful recovery rate requires good catheter and other diseases.

position and good media flow. These conditions are most easily met if the epidural completely abolishes rectal peristalsis. The ideal position for a two-way

Embryo collection

catheter is to have the cuff inflated at least 5 cm past the

Cleanliness of the epidural site and the perineum is

level of the intercornual ligament (ICL) (Fig. 40.2). In

important and cotton wool swabs dampened with sur-

the three-way system the stainless steel cannula, down

gical spirit are ideal for this. All instruments, catheters,

which the catheter passes, is inserted to the level of the

tubing and filters must be sterile. Metal instruments,

ICL. Upon removal of the centre, the catheter is passed

silicone tubing and glass tubing can be autoclaved,

down the cannula and along the uterine horn until the

whereas catheters and filters are usually gas sterilized

tip is about 10–15 cm from the end of the horn (Fig.

with ethylene oxide. No disinfectants or harsh cleans-

40.3). Inflation of the cuff should be enough to anchor

*ing solutions that may persist on surfaces and be
it in position without damaging the endometrium;
harmful to the embryos should be used. All equipment
usually 2 ml of air and 4 ml of sterile water will be suf-
sterilized by ethylene oxide must be aired for a
ficient in most donors. The two-way system will use
minimum of seven days before use.*

more media than the three-way because a larger

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(a) 10% Glycerol

Seeding Point

Embryo

Seal

Plug

Air

Air

Air

10% Glycerol

10% Glycerol

10% Glycerol

(b) 1.5 M ethylene glycol

Seeding Point

Embryo

Seal

Plug

Air

Air

Air

Air

Air

OCM

EG

EG

EG

OCM

OCM = Ovum culture media

Fig. 40.4

Methods of loading straws for

EG = 1.5 M ethylene glycol

freezing.

volume of uterine horn is being flushed. Generally the

Embryo processing

two-way system will require about 350–500 ml per horn
Examination of the embryos requires a good quality
compared to the three-way system using 180–200 ml per
stereoscopic dissecting microscope with at least ¥10, ¥25
horn. Care must be taken not to overdistend the horn
and ¥50 magnification. Disposable plastic Petri dishes
and aliquots of approximately 30 ml in a three-way
should be used. Once retrieved from the flushing solu-
system would be sufficient. To aid successful drainage
tion, embryos should be kept in embryo holding solution
of the media down the catheter it is important to lift the
at a constant ambient temperature (approximately 20°C)
tip of the uterine horn gently so that it is not curled
and out of strong light until the next procedure. Ideally,
under the body of the uterus. The bovine uterus is a
embryos should be frozen or transferred within three
fragile organ and rough handling or excessively force-
hours of collection. Although the law requires embryos
ful actions can result in damage to the endometrium or
to be washed ten times prior to freezing it is good prac-
perforation of the wall.

tice to wash all embryos whether destined for freezing or Best practice is to return the donor animals to normal fresh transfer. An outline of this procedure is given in cycles after the flush. This ensures termination of Box 40.2 and a more detailed account can be found in skulking embryos and reduces opportunities for the Manual of the International Embryo Transfer Society development of cystic ovarian disease after superovulation. (IETS) (Stringfellow & Seidel, 1998). The ten-times washing regime will remove a number of viruses from the using a combined progesterone releasing intravaginal zona pellucida whilst other more persistent ones can be device with PGF2a. When using PGF2a alone a higher removed by including a wash with trypsin in the procedure than normal is required to lyse the luteal tissue dure. Box 40.3 lists viruses and bacteria which can be present on superovulated ovaries. Consequently a cessfully removed by standard washing procedures and one-and-a half times or double normal dose of PGF2a trypsin washing.

is used. If donors are to be repeat flushed they should be allowed two oestrus events between flushes to opti-

Embryo freezing

mize the repeat results (Hasler et al., 1983). In practice this translates to an eight-week interval between

Prior to freezing the embryos are partially dehydrated flushes.

in cryoprotectant. Ready-to-use 10 per cent glycerol

Embryo

Plug

Air

Air

Air

OCM

OCM

OCM

OCM

Fig. 40.5

Method of loading straws for

OCM = Ovum culture media

transfer.

Box 40.2

Embryo washing procedure. Source: IETS

Box 40.3

Pathogens not found after washing of embryos

Manual, 3rd edn (Stringfellow & Seidel, 1998).

previously exposed to these agents. Source: IETS Manual, 3rd edn (Stringfellow & Seidel, 1998).

-

Only embryos from a single donor should be washed together

Normal ten-times washing:

Akabane virus

-

Ten or fewer embryos should be washed at one time

Bovine leukaemia virus

-

Only zona pellucida intact embryos should be washed

Bluetongue virus

-

Only embryos free of adherent material should be washed

Bovine viral diarrhoea virus

-

A minimum of ten washes is necessary, ensuring gentle

Foot-and-mouth disease virus

agitation in each wash

Brucella abortus

-

Use a new sterile micropipette each time embryos are

Trypsin washing:

Bovine herpes virus 4

moved from one wash to the next

Infectious bovine rhinotracheitis

-

Regulate volumes so that each wash is at least a 100-fold

virus

dilution of the previous wash, achieved by using 1 ml

Vesicular stomatitis virus

droplets of Dulbecco's PBS and a 10 ml micropipette to

move embryos

and 1.5 M ethylene glycol are available commercially,

straws are still submerged. If long-term storage is envis-

which have the benefit of being quality controlled and aged, separate into two groups and store in different screened for viruses. Prior to freezing the embryos need cryogenic flasks.

to be allowed time to equilibrate in the cryoprotectant.

This state is achieved after 5 to 10 minutes immersion

Embryo thawing

in 1.5 m ethylene glycol, but with the 10 per cent glycerol the embryo needs to be passaged step-wise through

Thawing of the straws is a reversal of the previous proce-

increasing concentrations of the solution. A common

dures. Once the straws have been removed from the

method is to allow 5 minutes in 5 per cent glycerol

liquid nitrogen they are placed in a flask of warm water.

(10 per cent glycerol diluted with an equal volume of

Some protocols require a short period of air thawing

embryo holding solution) followed by 15 minutes in

prior to entering the water. The water temperature

10 per cent glycerol. After the required equilibration

(22–37°C), the duration of submersion (7–30 seconds)

time in the cryoprotectant the embryos are loaded into

and the inclusion of air thawing (none–10 seconds) are all straws (Fig. 40.4), sealed and labelled before being put dependent on the method and programme protocol used in the freezing machine. It is important not to greatly for freezing. Most embryo transfer companies provide overextend the equilibration time as the cryoprotec- recommended thawing protocols along with the tants themselves are embryo-toxic if overexposure is embryos. Labels must be checked and the details allowed. At the appropriate time in the freezing proto- recorded. Straws with embryos frozen in 1.5 m ethylene col the cooling process is paused to allow seeding. glycol can be loaded into the transfer gun for immediate Seeding is easily performed using a metal rod, e.g. transfer. Embryos frozen in 10 per cent glycerol must be brass; the rod is supercooled in liquid nitrogen then removed from the straws they were frozen in and pas- gently and briefly touched against the straw (Fig. 40.4). saged through decreasing concentrations of glycerol; The seeded medium immediately becomes opaque as it they are then placed in embryo holding solution, re-

nucleates. Embryo damage can occur if too long a period of time elapses at seeding (overseeding) or if too large an area is seeded. The use of a brass rod is therefore preferable to tweezers for this reason. Once all straws are seeded the cooling process continues and at an ingress of water to the cells. One brand of ready-to-use solutions passages the embryo from 10 per cent glycerol through 5 per cent glycerol, 0.5 M sucrose (5 minutes) then 2.5 per cent glycerol, 0.5 M sucrose (5 minutes) and finally 0.6 M sucrose (5 minutes) before immersion in liquid nitrogen and should be kept submerged in liquid nitrogen and should not be exposed to air until thawing. The straws embryo holding solution/OCM prior to reloading. should be handled with tweezers, not fingers. The

easiest method to check identities on the straw labels is to place the whole goblet containing the straws in

Embryo transfers: practicalities

a wide-mouthed insulated flask containing liquid nitrogen. Thus the labels/plugs can be read while the For successful results forward planning is essential.

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Recipient selection criteria

ence of virus from the live IBR vaccines which may cause problems for ET progeny destined for export (including Recipient heifers should be:

Northern Ireland) or for entry to semen collection

- *At least 15 months old;*

centres. IBR marker vaccines (p. 1010) may alter the

- *At least 370kg (dairy heifers);*

situation but this will depend on recognition by the

- *At least 400kg (beef heifers);*

importing country and importer. If BVDV is known to be

- *Of a suitable frame size to carry to term and to*

a problem on a farm then screening of the recipients prior

calve naturally an embryo calf of the breed to be

to vaccination may be desirable to identify and remove transferred;

any persistently infected animals. When leptospirosis is a

- *Body condition score 2–21/*

known problem on a farm then treatment of the recipients

2; (assessed over the

range 1–5; Lowman et al., 1976; see also Chapter 2);

with 25 mg/kg of dihydrostreptomycin (British Cattle

- *Displaying regular oestrous cycles.*

Veterinary Association (BCVA), 1992) at the time of first

vaccination may be required to reduce the infective load

Recipient cows should be:

of this bacterium. As the epidemiology of Neospora

- *No more than a fourth calver;*

caninum becomes better understood (see Chapters 19,

- *Of a good breeding history;*

37) it may be prudent to screen potential recipients for

- *At least six weeks calved;*

this agent, especially if vertical transmission of this proto-

- *Clean and returned to regular oestrous cycles post*

zoan from recipient to fetus could affect the reproductive

calving;

performance of any resulting ET heifers.

- *Maintaining or increasing liveweight;*
- *Body condition score 2½–3;*

Oestrous synchronization

- *Of a suitable frame size to carry to term and to*

(see also Chapter 42)

calve naturally an embryo calf of the breed to be transferred.

For successful pregnancy to be achieved the recipients need to be tightly synchronized to come into oestrus one week before the embryo is transferred. Therefore a tight

Nutrition and management

synchrony period is required. A good method is to use a

It is important that recipients are on a rising plane of progesterone releasing intravaginal device or progestage nutrition for six to eight weeks before transfer and that implant in combination with a prostaglandin injection.

this continues for at least another six weeks post trans-

The device chosen can be inserted for 9, 10 or 11 days and fer. This is of particular relevance around the time of

the prostaglandin injection given 24, 48 or 72 hours housing and turn-out and ideally these two events before removal. This gives a tight synchrony with the should not occur in the middle of a programme but peak of activity 28–56 hours after device removal. In the should be completed either two months before the author's opinion a double prostaglandin injection proto-transfer date or delayed until at least six weeks post col will not provide a tight enough synchrony to maximize recipient usage. The stockperson should observe it is providing an acceptable balance of energy and recipients for oestrus the week before transfer. Observation should be performed at least three times a day for a donors, a suitable supply of minerals is important. If minimum duration of 20 minutes per observation period. molybdenum toxicity is likely to be a problem then a Observations should occur for the three days following suitable supply of protective copper should be made the day the synchrony devices are removed.

available (e.g. intraruminal boluses) (see pp. 254, 298).

Purchasing recipients, mixing groups and routine

Transferring the embryo

procedures, such as worming, bolussing, vaccination,

freeze-branding and foot-trimming, should all be com-

Having checked for the presence of a CL, and recorded

pleted at least six to eight weeks before the transfer

which ovary it is on, the recipient is prepared for the

date to minimize stress.

embryo transfer. Cleanliness of the epidural site and

the perineum is important and cotton wool swabs

dampened with surgical spirit are ideal for this. After

Disease control

traversing the cervix the transfer gun is guided along

A number of infective agents can severely affect the

the uterine horn ipsilateral to the corpus luteum. Preg-

success of an ET programme. The principal culprits are

nancies can be achieved if the embryo is placed at the

bovine viral diarrhoea virus (BVDV), leptospirosis and

level of the ICL, but results improve if the site of trans-

infectious bovine rhinotracheitis (IBR). Vaccination is

fer is further up the horn. Ideally a position of 10 cm or possible for all three agents but there is a risk of persist- more past the ICL is desirable. It is important to be as Embryo Transfer • 645

gentle as possible and avoid manoeuvres which result suppression was transient. Thus if the GnRH analogue is in endometrial damage. Sometimes in fatter animals given too soon the luteolytic mechanism may have time with large dependent tracts it is necessary to accept that to recover, resulting in a temporary benefit, but with the site of placement is not ideal in preference to over- some pregnancies being lost at a later date.

handling the tract, resulting in inflammation or physical These observations indicate that although hormonal injury to the endometrium.

supplementation has a tendency to improve pregnancy rates, the concept of a blanket treatment for groups of cattle needs to be considered carefully as inappropriate

Hormone supplementation

or mistimed treatments may at best have no benefit but Exogenous hormonal supplementation has taken three

at worst could be detrimental to pregnancy results.

main forms in an effort to provide support to the pregnancy. Firstly, exogenous progesterone or progestagen has been used. Progesterone injections have improved

Record keeping

pregnancy rates in asynchronous recipients. However, the use of progesterone releasing intravaginal devices Box 40.4 lists the important records that should be kept produced non-significant negative effects on pregnancy for donors, recipients and embryos. Legislation requires rates when these devices were in situ from day 7 to 19 that records are maintained for 12 months.

and had direct deleterious effects on embryo development when two devices (double dose) were used from day 7 to day 21. The use of exogenous progestagen

Legislation

treatments (Norgestomet), inserted on day 7, increased pregnancy rates (57.3 per cent control vs 67.8 per cent Trade in embryos between member states of the European Union (EU) and importation of embryos from

support pregnancies, but by day 38 of pregnancy in approved third countries, including the associated collection, processing, production and storage, is governed between treated and control groups. It has been observed that too much exogenous progesterone can

Box 40.4

List of necessary records to be maintained by licensed ET teams for 12 months after collection/production of an embryo. Source: Ministry of Agriculture, Fisheries and Food (1995).

sufficient progesterone for continued support of the pregnancy when the devices are removed. The conclu-

Donor

sion is that only discrete (as yet unidentifiable) categories of animal may benefit from this treatment and

Date of collection, place of collection

careful consideration will need to be given to the

Superovulatory response, number and category of embryos

dosage levels and timing of progesterone supplementa-

tion before applying it as a blanket treatment.

recovered, number and grades of viable embryos

Details of processing procedures for embryos (including

The second form of hormonal supplementation

freezing, transfer, micromanipulation and in-vitro fertiliza-
has been the administration of human chorionic

tion and culture as appropriate)

gonadotrophin (hCG) given at various times and for

Recipient

varying durations. Christie et al. (1979) used multiple Breed, age and ID no. of
recipient

injections between days 13 and 35 of pregnancy,

Date and place of transfer

whereas others used a single injection given on either

Identification of embryo transferred and its source (if known) the day of transfer,
day 14 or day 15. All showed a non-Storage

significant improvement in pregnancy rates in the

Identification of every embryo stored

treated animals over controls.

Date of entry/departure of every embryo

The third method of supplementation has been the use

Place of origin of embryo prior to arrival

of a GnRH analogue (buserelin) given between days 11

Destination of embryo on departure

and 13. This has been shown to increase pregnancy rates

significantly (MacMillan et al., 1986). In cyclic cows IVF storage

In addition to above:

treated with buserelin on days 11 and 13 of the cycle the

IVF embryos can be identified on a batch basis

plasma oestradiol concentrations were significantly

Must contain details of the date and place of collection of

reduced, suggesting that the mode of action is to reduce

the ovaries/oocytes

the strength of the luteolytic mechanisms, so improving

Must identify the herd of origin of donor animals

the embryo's chance of inhibiting luteolysis;however,the

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Box 40.5

Troubleshooting: in the event of poor superovulatory responses or embryo

recovery.

Problem

Issues to consider

Solution options

No reference heat

Poor heat detection

Abandon programme and restart

Poor behavioural signs

Provide artificial CL through exogenous

progesterone supplementation

Poor synchrony

Re-attempt

Poor superovulatory response

Nutrition

Check ration and adjust nutrition

Mineral status

Check mineral status and adjust supplementation

Type of FSH

Try alternative type of FSH

Drug dose/regime

Increase drug dose or alter regime

Unfertilized ova

Semen quality

Check bull history/try different sire

Insemination technique

Check technique

Semen handling/thawing

Check technique

Donor/bull interaction

Try different sire

Insemination timing

Add additional AI session and extra straws

Poor ovum quality

Check nutrition/mineral status/body condition loss

Overstimulation

Reduce FSH dose

Defective uterine environment

Infection – antibiotic therapy and/or return to oestrus

Hormonal – difficult to identify/resolve

Superovulation drug

Try alternative product

Poor recovery

Catheter position

Check technique

(normal breeding history)

Leakage past cuff

Check cuff inflation/integrity of cuff

Inadequate flow

Raise tip of uterine horn higher than cuff position

If above fails reposition catheter

(Note: risk of embryo loss in this procedure)

Luteinized follicles

Poor recovery

As above plus

(‘problem breeder’ history)

Blocked oviducts

Defective gamete transport

Ovarian/bursal adhesions

by EU Council Directive 89/556 of 25 September 1989, should be considered in assessing poor results in super-amended as appropriate by the relevant Council Directive of donor cows or pregnancy rates in embryos and Commission Decisions. Within most member

recipients.

states this European legislation is supported by domestic legislation.

In the UK the statutory instrument is the Bovine

Advanced technologies

Embryo (Collection, Production and Transfer) Regulations 1995. This covers the licensing of ET teams and

Embryo splitting

premises, legislation relating to disease control

This technology was promoted in the early 1990s as a means of improving reproductive health, fertility, and welfare and also record keeping. The above EU commercial application. Its appeal was that it could give a more accurate picture of fertility and UK legislation along with the Manual of the Inter-Professional Society for the Study of Fertility (a collection of proportionately more pregnancies from a national Embryo Transfer Society (a procedural guide of embryos than could be achieved by simply transferring and general information for the use of embryo transfer ring the embryos whole. It involved bisecting the technology emphasizing sanitary procedures) are embryo with a microblade or a specially designed glass essential reading for anyone planning to become

microneedle. These instruments were operated via a involved in practical embryo transfer.

micromanipulator which converts gross movements by the operator at the controls to very fine micromove-

Troubleshooting

ments at the level of the embryo. One disadvantage is that this is a relatively expensive piece of equip-

Embryo collection results will vary between donors and ment. Inevitably the production of these demi-embryos

pregnancy results can fluctuate between batches of resulted in some blastomere damage of the embryo so

recipients. Boxes 40.5 and 40.6 summarize factors that that split embryos tended to have lower pregnancy rates

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Box 40.6

Troubleshooting: in the event of poor pregnancy rates.

Recipients

Check:

Nutrition

Mineral status

Body condition gain/loss

Health

Risk of disease challenge

Vaccination status

Management

Check:

Correct completion of synchrony programme

Heat detection

Recipient handling/stress

Embryos

Check:

Grades used

Freezing protocol

Thawing protocol

Quality control of procedures at each step

Miscellaneous

Consider donor effects:

- *oocyte quality*
- *donor/bull interaction (genetic effect)*
- *donor uterine environment*
- *a percentage of donors are consistently poor candidates*

Consider operator technique/experience

than contemporary whole embryos (split embryos 57
able variation between laboratories, but the incidence
per cent versus whole embryos 65 per cent). Even
in some cases was up to 20 per cent. The syndrome man-
though an increased number of calves could be pro-
ifested itself as giving large calves at birth (up to 75 kg)
duced (an increase of the order of 13 per cent) the tech-
with increased birthweights greater than could be
nique was never really accepted commercially by either
accounted for by extended gestation length alone. This
the ET operators or the cattle breeders.

was associated with an increased incidence of dysto-
cia and a higher mortality rate. Another feature was
enlarged placentae in some of these pregnancies, on

In-vitro fertilization (IVF)

occasion with a mean weight 25 per cent greater than
This technology involves the creation of embryos in the
those of untreated contemporaries. Additionally, in
laboratory. A source of immature oocytes is required.
some cases, there were congenital malformations and
These can be harvested from ovaries removed from a

problems with altered allometric coefficients for the slaughtered animal as long as the ovaries are submitted liver, kidney and heart which eventually left these to the laboratory within four to six hours. Alternatively calves unable to survive. Interestingly, despite differences in birthweight, the mean liveweight at one year sound-guided ovum pick-up (see later). The immature oocytes can be collected from the live animal by ultrasound-guided ovum pick-up (see later). The effects for these animals can be similar to controls. The effects oocytes must first be matured, which takes approximately 18 hours. After this fertilization takes place on the pregnancies did vary depending on the laboratories from which the embryos came. Therefore it by mixing capacitated sperm with the embryo. Follow- was deduced that a factor in the maturation or culture ing fertilization the embryos are cultured to a stage method was significant. The resultant worldwide of development equivalent to day 7 in-vivo embryos, research effort is helping to elucidate the mechanisms when they can then be transferred fresh or frozen. Pregn- involved. To date, one widely observed finding is that

nancy results with IVF embryos tend to be lower than from those originated in vivo (IVF 40–50 per cent as opposed to serum reduces, or eliminates, the incidence of large calves. Another potential

problem linked to IVF is that of the ‘large calf syndrome’. Although it can also be associated with asyn-

Ovum pick-up (OPU)

chronous timing of embryo transfer and progesterone supplementation early in the oestrous cycle of the dam,

This is the technique that allows the collection of oocytes for IVF from the ovaries of live donor animals.

It was documented in the early 1990s as being associated with IVF calves. The results of various IVF laboratories worldwide showed that a significant number of

ET pregnancies could be affected. There was considerable manual manipulation of the ovaries. The ultrasound

The operator places one hand in the rectum to allow

ET pregnancies could be affected. There was considerable manual manipulation of the ovaries. The ultrasound

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probe is placed in the vagina so that the ovary can be

cytometer/cell sorter utilizing a laser can then sort the visualized with only the vaginal wall between the ovary sperm on the basis of their DNA content (Cran and the head of the probe. Usually a 7.5 MHz curvilinear- et al., 1993). At present the technique is 90 per cent accurate. A probe is used. On visualization of a follicle on the rate, but only small quantities of sperm can be processed ovary a 60 cm needle (18–21 gauge) is passed down a at a time and the resulting sexed semen is more successful special channel in the probe. It punctures the vaginal ful in fertilizing ova if used for deep uterine AI (Seidel et al. and enters the follicle on the ovary. A dotted line al., 1998). New freezing protocol advances for sexed on the scanner screen indicates the line down which the semen may obviate the need for deep uterine AI.

needle will transect the ovary, allowing the operator to line up the follicle with the intended path of the needle.

Cloning

At the point of entry the operator uses a foot-pedal to apply a vacuum pressure to the collection line attached Cloning results in the production of genetically identical to the needle. Too high a pressure results in stripping

cal offspring. The simplest form of cloning is twinning of the cumulus cells from the oocyte and these de- (bisecting an embryo). Further advances in the technology resulted in the technique of nuclear transplantation. On average, ten oocytes can be collected from both ovaries per session and it is possible to perform a collection enucleation of ova and the electrofusion of blastomeres session weekly or twice weekly (van der Schans et al., from donor embryos. In essence, unfertilized ova are 1991). This usually translates, after developmental losses and fertilization failures, to two transferable genetic material is supplied, in the case of embryos, embryos per collection session. On average, 70–80 per cent of oocytes recovered are suitable for further cell stage (32 blastomere stage at approximately day 5). processing. Of these, 70 per cent will be successfully These blastomeres are disassociated and an individual

fertilized and start to cleave and ultimately 40 per cent blastomere (genetically identical to the other 31 blastomeres) is introduced into the enucleated recipient oocyte. With an average pregnancy rate of 50 per cent this means that approximately one pregnancy will result from each OPU session.

embryo is cultured to day 7 prior to transfer to a recipient animal. Alternatively, at day 5 the whole procedure can be repeated using blastomeres from the cloned embryo to create another generation. There are reports

Embryo sexing

of this recloning process being used to produce second and third generation embryos. The difference between simple, easy-to-use way of sexing embryos. The most this procedure and that used to produce the late Dolly the sheep is that, for Dolly, the donated nuclear mate-

Finland (Bredbacka et al., 1995). It involves taking a rial was recovered from an adult somatic cell which was

biopsy of a few cells from the embryo and lysing them then 'reprogrammed' to be capable of differentiation to release the DNA content. This DNA material is then and subsequent organogenesis.

mixed with enzymes and multiplied using a polymerase

*Currently, however, these procedures are too ineffi-
chain reaction (PCR). A stain is included in the solu-
cient for commercial production of large numbers of
tions which binds to the Y chromosomes, making them
cloned animals and are only justified for biomedical
fluoresce under UV light. On completion of the PCR
applications. This has resulted in some commercial
cycle the tubes containing the biopsies from male
companies utilizing the technology to produce cloned
embryos are identified by this fluorescence. Non-fluo-
animals for production of specific proteins for biomed-
rescing samples are deemed to be female. This tech-
ical purposes.*

nique is 95 per cent successful in identifying females

and 100 per cent in identifying males. The discrepancy is due to errors when biopsies are not placed correctly

Acknowledgement

in the sample tubes.

New technology allows the production of correctly

The author would like to thank all who assisted with sexed embryos through the use of sexed semen. X chromosome research for this chapter and particularly David McNee for the illustrations, George Seidel Jr and Richard Bowen of Colorado State University for the pictures and fluoresces in a laser beam. X chromosomes with higher DNA content will fluoresce more brightly. A flow cytometer is used to sort sperm with a Y chromosome. The semen is mixed with a stain which is taken up by DNA. The Y chromosome fluoresces more brightly than the X chromosome. The author would like to thank Seonaid Grimmer, Mike Kerby and Arthur Redpath for constructive comments on the manuscript.

Embryo Transfer • 649

References

ient heifers at day of transfer. Theriogenology, 17, 105 (abstract).

Schans van der, A., van der Westerlaken, L.A.J., de Wit,

Bredbacka, P., Kankaanpää, A. & Peippo, J. (1995) PCR-
A.A.C., Eyestone, W.H. & de Boer, H.A. (1991) Ultra-
sexing of bovine embryos: a simplified protocol. *Theri-
sound-guided transvaginal collection of oocytes in the cow.
ogenology*, **44**, 167–76.

Theriogenology, **35**, 288 (abstract).

BCVA (1992) *Guidelines for the diagnosis and control of* Lep-Seidel Jr, G.E.,
Herickhoff, L.A., Schenk, J.L., Doyle, S.P. & tospira hardjo infection in cattle.
British Cattle Veterinary Green, R.D. (1998) Artificial insemination of heifers
with

Association, Frampton-on-Severn, pp. 13–14.

cooled, unfrozen sexed semen. *Theriogenology*, **49**, 365

Christie, W.B., Newcomb, R. & Rowson, L.E.A. (1979)
(abstract).

Embryo survival in heifers after transfer of an egg to the

Smith, A.K., Broadbent, P.J., Dolman, D.F., Grimmer, S.P.,

uterine horn contralateral to the corpus luteum and the

Davies, D.A.R. & Dobson, H. (1996) Norgestomet implants,

effect of treatments with progesterone or hCG on preg-

plasma progesterone concentrations and embryo transfer

nancy rates. Journal of Reproduction and Fertility, **56**, 701–6.

pregnancy rates in cattle. Veterinary Record, **139**, 187–91.

Cran, D.G., Johnson, L.A., Miller, N.G.A., Cochrane, D. &

Stringfellow, D.A. & Seidel, S.M. (eds) (1998) *Manual of the Polge, C. (1993) Production of bovine calves following*

International Embryo Transfer Society, 3rd edns. Interna-separation of X- and Y-chromosome bearing sperm and in-

tional Embryo Transfer Society, Sary.

vitro fertilisation. Veterinary Record, 132, 40–1.

Webb, R., Gosden, R G., Telfer, E.E. & Moor, R.M. (1999)

Donaldson, L.E. (1985) *Matching of embryo stages and grades*

Factors affecting folliculogenesis in ruminants. Animal

with recipient oestrous synchrony in bovine embryo trans-

Science, 68, 257–84.

fer. Veterinary Record, 117, 489–91.

Wright, J.M. (1985) *Commercial freezing of bovine embryos*

European Community (1989) Council Directive 89/556 EEC

in straws. Theriogenology, 23, 17–29.

of 25 September 1989. In Official Journal of the European

Community No. L 302/10, 19.10.89.

Hasler, J.F., Bowen, R.A., Nelson, L.D. & Seidel, G.E. Jr (1980) **Further reading**

Serum progesterone concentrations in cows receiving

embryo transfers. Journal of Reproduction and Fertility, 58,

Factors affecting pregnancy rates

71–7.

Hasler, J.F., McCauley, A.D., Lathrop, W.F. & Foote, R.H.

Armstrong, D.T. (1993) Recent advances in superovulation of

(1987) Effect of donor–embryo–recipient interactions on

cattle. *Theriogenology*, **39**, 7–24.

pregnancy rate in large-scale bovine embryo transfer

Arreseigor, C.J., Sisul, A., Arreseigor, A.E. & Stahringer, R.C.

program. Theriogenology, **27**, 139–68.

(1998) Effect of cryoprotectant, thawing method, embryo

Hasler, J.F., McCauley, A.D., Schermerhorn, E.C. & Foote,

grade and breed on pregnancy rates of cryopreserved

R.H. (1983) Superovulatory responses of Holstein cows.

bovine embryos. Theriogenology, **49**, 160 (abstract).

Theriogenology, **19**, 83–99.

Beal, W.E., Hinshaw, R.H. & Whitman, S.S. (1998) Evaluating Lowman, B.G.,

Scott, N.A. & Somerville, S.H. (1976) Condi-embryo freezing method and the site
of embryo deposition

tion scoring of cattle. East of Scotland College of Agricul-

on pregnancy rates in bovine embryo transfer. Theri-

ture, No. 6.

ogenology, **49**, 241 (abstract).

MacMillan, K.L., Taufa, V.K. & Day, A.M. (1986) Effects of an Gayerie de Abreu, F., Lamming, G.E. & Shaw, R.C. (1984) A

agonist of gonadotrophin releasing hormone (buserelin) in

cytogenetic investigation of early stage bovine embryos –

cattle III, pregnancy rates after a post-insemination injec-

relation with embryo mortality. In Proceedings of the 10th tion during metoestrus or dioestrus. Animal Reproduction

International Congress on Animal Reproduction and AI,

Science, **11**, 1–10.

Illinois, paper 82.

Ministry of Agriculture, Fisheries and Food (1995) The Bovine

Lamming, G.E. & Mann, G.E. (1993) Progesterone concen-

Embryo (Collection, Production and Transfer) Regulations

tration affects the development of the luteolytic mechanism

1995: Statutory Instrument 1995 No. 2478 Agriculture,

in the cow. Journal of Reproduction and Fertility Abstract Livestock Industries. Her Majesty's Stationery Office,

Series, No. 11, abstract 8.

London.

Mann, G.E., Mann, S.J. & Lamming, G.E. (1996) The inter-

Monniaux, D., Chupin, D. & Saumande, J. (1983) Superovula-

relationship between the maternal hormone environment

tory responses of cattle. *Theriogenology*, **19**, 55–81.

and the embryo during the early stages of pregnancy.

Newcomb, R. & Rowson, L.E.A. (1975) Conception rate after

Journal of Reproduction and Fertility Abstract Series, No. 17, transfer of cow eggs in relation to synchronisation of oestrus abstract 55.

and age of egg. *Journal of Reproduction and Fertility*, **43**, 539–41.

Nutrition

Northey, D.L., Barnes, F.L., Eyestone, W.E.H. & First, N.L.

(1985) Relationship of serum progesterone, luteinising

Black, D.H. & French, N.P. (1999) Copper supplementation

hormone and the incidence of pregnancy in bovine embryo

and bovine pregnancy rates; three types of supplementation

transfer recipients. *Theriogenology*, **23**, 214 (abstract).

compared in commercial dairy herds. In *Proceedings of the*

Remsen, L.G., Roussel, J.D. & Karihaloo, A.K. (1982) Preg-

Nottingham Cattle Fertility Conference, 1999, Nottingham pregnancy rates relating to plasma progesterone levels in recipient-University, pp. 29–36.

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O’Gorman, J., Smith, F.H., Pook, D.B.R., Boland, M.P. &

Massey, J.M., Oden, A.J., Voekel, S.A. & Godke, R.A. (1983) Roche, J.F. (1987) The effect of molybdenum-induced

Pregnancy rate following hCG treatment of bovine embryo

copper deficiency on reproduction in beef heifers. Theriogenology, **19**, 140 (abstract).

Theriogenology, **27**, 265 (abstract).

Peterson, A.J. & McMillan, W.H. (1996) The effect of progestins-Phillippo, M., Humphries, W.R. & Atkinson, T. (1987) The

effect of progesterone supplementation on actual and potential pregnancy

rates and embryo survival following the single or twin

puberty, fertility and oestrous cycles in cattle. Journal of transfer of cattle

blastocysts. Theriogenology, **45**, 228

Agricultural Science (Cambridge), **109**, 321–36.

(abstract).

Rosmarin, M.L., Lock, T.F., Dahlquist, T.G., Nash, T.G.,

Faulkner, D.B. & Kesler, D.J. (1998) Norgestomet implants

Superovulation

enhance embryo survival in post-partum cows. Theriogenology, **49**, 355 (abstract).

Bo, G.A., Adams, G.P., Pierson, R.A. & Mapletoft, R.J. (1996) Santos-Valdez de los, S., Seidel Jr, G.E. & Elsdon, R.P. (1982) Effect of progestagen plus oestradiol-17 β treatment on

Effect of hCG on pregnancy rates in bovine embryo trans-

superovulatory response in beef cattle. Theriogenology, **45**, 85 (abstract). Theriogenology, **17**, 85 (abstract).

897–910.

Sheldon, I.M. & Dobson, H. (1993) Effects of gonadotrophin Broadbent, P.J., Tresgaskes, L.D., Dolman, D.F. & Smith, A.K.

releasing hormone administered 11 days after insemination

(1996) The effects of varying dose and pattern of adminis-

on the pregnancy rates of cattle to the first and later

tration of ovine FSH on the response to superovulation in

services. *Veterinary Record*, **133**, 160-3.

performance tested, juvenile Simmental heifers. *Animal*

Tribulo, R., Nigro, M., Bury, E., Caccia, M., Tribulo, H. & Bo, Science, **62**, 181–6.

G.A. (1997) Pregnancy rates in recipients receiving CIDR-

Garcia, G.J.K., Elsdon, R.P. & Seidel Jr, G.E. (1983) Efficacy B devices immediately following embryo transfer. Theri-of PGF2a, for reducing the return to estrus interval in super-

ogenology, **47**, 372 (abstract).

ovulated cattle. *Theriogenology*, **19**, 129 (abstract).

Greve, T., Callesen, H., Hyttel, P., Høier, R. & Assey, R. (1995) The effects of exogenous gonadotrophins on oocyte and

Embryo splitting

embryo quality in cattle. *Theriogenology*, **43**, 41–50.

Gray, K.R., Bondioli, K.R. & Betts, C.L. (1991) The commer-Tonhati, H., Lobo, R.B. & Oliveira, H.N. (1999) Repeatability cial application of embryo splitting in beef cattle. Theri-and heritability of response to superovulation in Holstein

ogenology, **35**, 37–44.

cows. *Theriogenology*, **51**, 1151–6.

Kippax, I., Christie, W. & Rowan, T.G. (1991) Effects of

Warfield, S.J., Seidel Jr, G.E. & Elsdon, R.P. (1986) A com-method of splitting,
stage of development and presence or

parison of two FSH regimens for superovulating cows and

absence of zona pellucida on fetal survival in commercial

heifers. *Theriogenology*, **25**, 213 (abstract).

bovine embryo transfer of bisected embryos. *Theriogenol-
ogy*, **35**, 25–35.

Hormonal supplementation

In-vitro fertilization

Broadbent, P.J., Sinclair, K.D., Dolman, D.F., Mullan, J.S. & McNally, J.R.
(1992) The effect of a norgestomet ear implant

Sinclair, K.D., Broadbent, P.J. & Dolman, D.F. (1995) In-vitro (Crestar) on
pregnancy rate in embryo transfer recipients.

produced embryos as a means of achieving pregnancy and

*In Proceedings of the 12th International Congress on Animal improving
productivity in beef cows. Animal Science*, **60**, Reproduction, The Hague, Vol. 2,
pp. 782–4.

55–64.

Diskin, M.G. & Sreenan, J.M. (1986) Progesterone and

Wagtendonk-de Leeuw van, A.M., Aerts, B.J.G. & Daas

embryo survival in the cow. In *Embryo Mortality in Farm*

den, J.H.G. (1998) *Abnormal offspring following in-vitro Animals* (ed. by J.M. Sreenan & M.G. Diskin) pp. 142–58.

production of bovine preimplantation embryos: a field
Martinus Nijhoff, Dordrecht.

study. Theriogenology, **49**, 883–94.

Geisert, R.D., Fox, T.C., Morgan, G.L., Wells, M.E., Wette-

Walker, S.K., Hartwich, K.M. & Seamark, R.F. (1996) *The*

mann, R.P. & Zavy, M.T. (1991) *Survival of bovine embryos*

production of unusually large offspring following embryo

transferred to progesterone treated asynchronous recipi-

manipulation: concepts and challenges. Theriogenology, **45**, ents. *Journal of*
Reproduction and Fertility, **92**, 475–82.

111–20.

Greve, T. & Lehn-Jensen, H. (1982) *The effect of hCG administration on*
pregnancy rate following non-surgical transfer

Ovum pick-up

of viable bovine embryos. Theriogenology, **17**, 91 (abstract).

Looney, C.R., Oden, A.J., Massey, J.M., Johnson, C.A. &

Pieterse, M.C., Vos, P.L.A.M.; Kruip, Th.A.M., Wurth, Y.A.,

Godke, R.A. (1984) *Pregnancy rates following hCG admin-*

Beneden van, Th.H., Willemse, A.H. & Taverne, M.A.M.

istration at the time of transfer in embryo-recipient cattle.

(1991) Transvaginal ultrasound guided follicular aspiration

Theriogenology, **21**, 246 (abstract).

of bovine oocytes. *Theriogenology*, **35**, 19–24.

Mann, G.E. & Lamming, G.E. (1995) Effects of treatment with Scott, C.A., Robertson, L., de Moura, R.T.D., Paterson, C. & buserelin on plasma concentrations of oestradiol and prog-Boyd, J.S. (1994) Technical aspects of transvaginal ultra-

esterone and cycle length in the cow. *British Veterinary*

sound-guided follicular aspiration in cows. *Veterinary*

Journal, **151**, 427–32.

Record, **134**, 440–3.

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Cloning

Takano, H., Kozai, C., Shimizu, S., Kato, Y. & Tsunoda, Y.

(1997) Cloning of bovine embryos by multiple nuclear

Campbell, K.H.S., McWhir, J., Ritchie, W.A. & Wilmut, I.

transfer. *Theriogenology*, **47**, 1365–73.

(1996) Sheep cloned by nuclear transfer from a cultured cell

Wolfe, B.A. & Kraemer, D.C. (1992) Methods in bovine

line. *Nature*, **380**, 64–6.

nuclear transfer. *Theriogenology*, **37**, 5–15.

Chapter 41

Herd Fertility Management

(a) Beef herds

input and low output enterprises. In the UK, consumer concerns about food safety and the BSE crisis mean

S. Borsberry

that cull cows are of relatively low value and so the main income derives from the progeny, from either sale as store or finished cattle (see Chapter 3). Calving inter-

Introduction

652

val is thus one of the most important measures of repro-

Determinants of reproductive performance

652

ductive performance. One of the main effects on

Factors affecting reproductive performance

654

reproductive performance in beef cows is the adverse

Infectious disease

654

effect of marginal nutrition. Inefficient suckler herds

Nutritional management

655

are unlikely to survive in the present economic climate

Controlling the length of the calving season

656

and, in Britain, marketing of progeny may become

Feeding policy

656

increasingly difficult following the 2001 foot-and-mouth

Culling policy

656

Post partum onset of ovarian cycles

656

disease (FMD) outbreak. There is likely to be increased

Factors affecting the post partum acyclic period

656

legislation concerning the movement of FMD suscepti-

Suckling

656

ble stock which may well affect public auction at live-

Nutrition, body weight and condition and the

stock markets, thereby reducing the potential number

post partum period

657

of outlets for farm animals.

Season

657

In some beef herds cows and bulls are together all

Induction of ovulation in beef cows

657

the year round and there is little attempt to control

Gonadotrophin-releasing hormone

658

fertility or the season of calving. In better-managed

Oestrus synchronization and AI in beef cattle

658

herds a seasonal calving pattern is adopted and the

Methods

659

calving pattern itself becomes an important assessment

Management of heifers

661

of reproductive performance. An excellent review of

Monitoring reproductive performance in the beef herd

661

Analysing herd problems

662

suckler cow management in the UK has been published (Lowman, 1988) and this paper is recommended if the reader requires further detail after reading this chapter.

Introduction

Beef cattle fertility must be considered as a separate

Determinants of reproductive

entity from dairy cattle as they are managed under

performance

entirely different circumstances. Usually they are

managed as a herd or group and so do not receive the

In the UK, beef cows traditionally calve either in spring

same individual attention as do dairy cows. In general,

or autumn. The trend in gross margins has decreased

clinical infertility problems do not appear to occur to

over recent years (1995 to 1999); although there has

the same extent. However, with the dairy herd supply-

been a marginal increase in stocking rate, sale price has

ing fewer cross-bred female replacements and the ever reduced significantly (see Table 41.2).

increasing importance of biosecurity, many herds are Autumn-calving herds tend to produce higher gross breeding their own replacements. This results in a need margins than spring-calving herds under UK conditions for careful planning of breeding in order not to lose (see Table 41.3). Spring-calving herds tend to be fertility and longevity that is achieved with heterosis stocked more heavily, but there is greater financial (Table 41.1).

output from autumn-calving herds.

Veterinary involvement in most commercial suckler

The calf is essentially the sole product of the beef herds is often limited, as these herds are run as low herd, therefore the rate of calf production (calves born

652

Herd Fertility Management • 653

Table 41.1

Traits affected by hybrid vigour (Reproduced with

Table 41.3

Gross margins and performance for upland

permission from Meat and Livestock Commission, 2000).

sucklers (1999) (Reproduced with permission from Meat and

Livestock Commission, 2000).

Trait

Predicted hybrid

vigour effect

Spring Autumn

calving

calving

Conformation

Skeletal size

0–5%

Low

}

Number of herds

43

43

Mature weight

Gross margin (£)

Per cow

208

227

Growth rate

Per hectare

355

404

Weaning weight

5–10%

Medium

}

Calf age at sale (days)

212

314

Milk production

Calf weight at sale (kg)

268

335

Fertility

Stocking rate (cows/ha)

1.7

1.5

Health

10–30%

High

}

Longevity

Table 41.4

Performance of upland suckler herds (1999)

(Reproduced with permission from Meat and Livestock Commission, 2000).

Table 41.2

Trends in gross margin results for upland suckler

herds, 1995–99 (Reproduced with permission from Meat and

Bottom Top

Livestock Commission, 2000).

third

third

Sale price

Stocking rate

Gross margin

Gross margin (£)

(p/kg lw)

(cows/ha)

Per cow

151

278

(£/head)

(£/ha)

Per hectare

253

493

Cow performance (per 100 mated)

1995

124

1.4

313

438

Calving period (weeks)^a

12

10

1996

112

1.5

295

445

Number barren

6

6

1997

107

1.7

308

505

Calves born alive

91

92

1998

87

1.5

214

316

Calf mortality

3

2

1999

95

1.5

217

380

Calves purchased

1

2

Calves reared

89

92

lw, liveweight.

Calf performance

Age at sale/transfer (days)

238

287

Weight at sale/transfer (kg)

276

329

Daily gain (kg)

alive and reared) is even more critical than it is in the

Male

1.0

1.1

dairy herd. In order to illustrate this a comparison

Female

0.9

1.0

between the worst and best performing (in terms of

Stocking rate (cows/ha)

1.5

1.6

gross margin/cow) suckler herds recorded by the Meat

and Livestock Commission (MLC) (2000) is shown in

a For 90 per cent of calvings.

Table 41.4. The most important factors causing the

differences were

Also, the gestation period and hence the calving

(1)

The number of calves reared/100 cows and

interval can be influenced, to a limited extent, by the

(2)

The calf weight at sale/transfer.

breed of sire.

Put more simply, the most successful herds sold more

When breeding replacements for the suckler herd,

heavier calves than the worst.

two aspects must be considered: mature body size and

Calf weight and age at sale/transfer are reflections of

breed composition. Mature body size must be matched

health and growth rate; obviously, calf weight is further

by feed resources. The larger the cow, the more difficult

dependent, to a major extent, on the breed of the sire

it is to maintain good body condition and this will

and breeding programme used. The heavier breeds

reduce fertility. In order to maximize hybrid vigour the

such as Charolais and Simmental produce calves with

cross breeding system can be worth up to 23 per cent

a high weight. However, this has to be balanced

more weight of weaned calf per cow put to the bull

against a higher risk of dystokia and possible calf

(Meat and Livestock Commission, 2000).

mortality (see Table 41.5), but it must be remembered

*An important index of reproductive performance in
that there is considerable variation within a breed.
the suckler herd is the length of calving season. A 365-
654 • Chapter 41*

Table 41.5

*Pure bred results (Reproduced with permission from Meat and Livestock
Commission, 2000).*

Birth

200 day

400 day

Easy

weight

average

average

calvings

average

(kg)

(kg)

(%)

(kg)

M

F

M

F

M

F

M

F

Aberdeen Angus

37

35

267

238

516

398

92

95

Blonde Aquitaine

41

39

274

238

548

421

80

88

Belgian Blue

45

41

269

245

494

404

17

32

Charolais

44

41

325

281

652

466

70

80

Hereford

41

39

248

223

447

362

87

92

Limousin

39

36

274

242

545

405

89

94

South Devon

46

42

271

245

499

389

73

86

Simmental

42

38

321

278

628

449

81

89

Sussex

39

36

232

214

411

331

94

94

Welsh Black

38

36

227

215

376

329

81

89

M = male, F = Female.

Table 41.6

Theoretical relationship between conception rate and the calving period in a 100 cow herd (adapted from ICI computer simulation by Allen & Kilkenny, 1980).

Herd conception

Service period for

Length of

No. of cows

rate (%)

90% of cows to be

calving period

calving in

pregnant (days)

(days)

first month

30

245

260

12

40

140

155

28

50

100

110

41

60

70

85

54

day calving interval is optimal, but a compact calving theoretical relationship between the conception rate, season is also desirable because:

the service period and the calving period was calculated by ICI and is shown in Table 41.6.

- *More cows calve early in the period, therefore the age and weight of calves are higher at the time of weaning and sale.*

- *The impact of calf disease and mortality may be*

Factors affecting reproductive

reduced if there is little variation in calf ages.

performance

- *Cows are all at a similar stage in the production cycle, therefore feed can be rationed more precisely*

Infectious disease

and the cows managed more conveniently.

In some cases of low herd reproductive performance

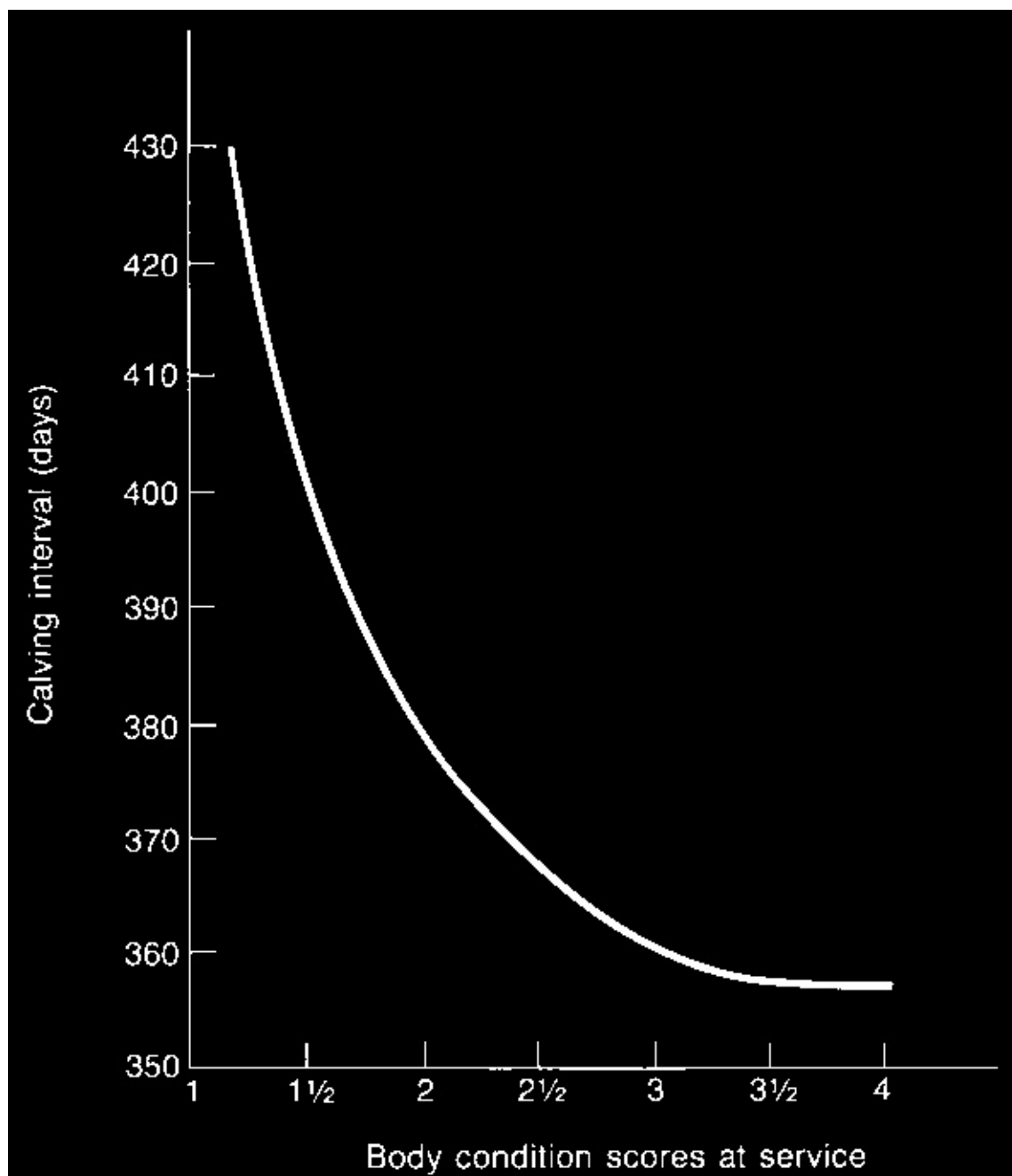
- *When cattle are sold they can be batched to there may be an underlying infectious disease.*

produce groups of similar size, weight and

Although the UK is officially brucellosis-free there is condition.

always the potential for its re-introduction; indeed, this

The calving season is almost directly dependent on the has happened. Following the use of artificial insemination (AI), campylobacteriosis and Tritrichomonas fetus pregnancy rate to service and the length of the breeding or service period. Obviously, the higher the pregnancy rate, the shorter the service period required to be a problem in some suckler herds where a bull is used ensure pregnancy of all or the majority of the cows. A (Caldow & Taylor, 1997). Additionally, some viral dis-



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Table 41.7(a)

Recommended target body condition scores of

Table 41.7(b)

Relationship between body condition and repro-

beef cows at various stages of the reproductive cycle (Lowman, ductive performance (Kilkenny, 1978).

1988).

Herd average

Calving interval

Calves weaned per

Calving

Mating

Weaning

mating scores

(days)

100 cows bulled

Autumn-calving

3.0

2.5

2.5

1–2

418

78

suckler cows

2

382

85

Spring-calving

2.5

2.0

3.0

2–3

364

95

suckler cows

3+

358

93

eases may result in poor reproductive performance, for example bovine herpes I (BHVI) infection (see pp. 289, 579) and bovine viral diarrhoea (BVD) (see pp. 578, 853).

Obviously, it is essential to rectify any infectious problem before attempting to improve herd reproductive performance by other means.

Nutritional management

Nutritional status is of major importance in the maintenance of a high rate of reproductive performance. The nutrition of cattle is covered more comprehensively in specialized texts and only the basic principles are described here.

Whilst specific deficiencies of micronutrients are common under particular circumstances and can affect fertility, under normal conditions dietary energy appears to be the main factor limiting reproductive performance. However, other nutritional deficiencies occur but are usually confined to localities or even individual herds. These include problems with manganese, selenium and cobalt (see Chapter 18) and some vitamins,

Fig. 41.1

The relationship between calving interval and body condition score at service in beef cows (Kilkenny, 1978). For a fuller account of this problem the reader is referred to Chapters 9 and 21. The technique of body condition scoring has been developed as a simple semi-objective monitor of cows'

energy status. The principle depends on the manual operator can assess the body condition of cows to palpation of the thickness of subcutaneous fat cover on within one half-unit. Optimum body condition scores various parts of the body (see p. 10).

for beef cows have been worked out for the various Methods have been developed for both dairy stages of the reproductive cycle and these are shown in (Mulvaney, 1978) and beef cows (Lowman et al., 1976) Table 41.7a. The target body condition score at service and although the finer details vary slightly the overall is the most critical as this is very closely related to principle is the same. The thickness of fat cover over overall reproductive performance. The calving interval the lumbar and tailhead area is estimated and assigned has been shown to be negatively correlated with body a score, usually from 0 (emaciated) to 5 (very fat), condition at the time of mating in beef cows, although although different scales are sometimes used. Describe the true relationship is probably curvilinear (see Fig. tions of the various categories on the 0–5 scale are given

41.1) and affects the number of calves weaned (Table on p. 10.

41.7b). This target score is also most difficult to achieve

An additional guide to body condition can be

in autumn-calving cows as they are mated during mid-

obtained by palpating over the hip bones, ribs and

winter when they are lactating and when good quality

either side of the tailhead. With a little experience an

feed is expensive. In a survey, approximately half of the

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Table 41.8

Financial effects of a high culling rate (after Allen & Kilkenny, 1980).

Normal

High culling rate

culling

policy

Year 1

Year 2

Year 3

No. of cows

78

86

88

No. of replacements

13

36

20

16

Calving spread (days)

150

100

100

100

Average calf sale

Weight (kg)

285

285

305

315

Gross margin/ha (£)

219

174

233

257

autumn-calving beef cows failed to reach the target necessitate the achievement of calving intervals in late-condition score at mating (Kilkenny, 1978). In contrast, calving cows of well below 365 days. However, it is the nutritional drain of lactation is offset in spring important to remember that proper nutritional management by the plentiful supply of grazing.

agement is vital for the maintenance of a compact Cows should be fed to calve at a condition score of seasonal calving pattern.

2.5–3 and should lose minimum condition until conception (see Table 41.7a). Cows calving in fatter condi-

Culling policy

tion may have calving difficulties, which in turn may lead to delayed involution, reproductive tract damage, This is the most extreme but probably the most effective susceptibility to infection of the tract or a combination tive method of modifying a herd's calving pattern. As

of these problems. Also, cows with a score of 4 or more discussed above, the length of the calving period is are likely to mobilize their fat reserves excessively highly dependent on the herd pregnancy rate. For during the early postpartum period.

example, a five-month calving period is approximately Beef cows calving in a low condition are also likely equivalent to a pregnancy rate of 40 per cent. In this to undergo a prolonged period before the re-situation approximately 70 per cent of cows will calve establishment of ovarian cycles, undernutrition being in the first two months and 30 per cent in the next three one of the major causes of failure to ovulate after months. Therefore, the 30 per cent late calvers should calving. Consequently, pregnancy is likely to be considered culled and replaced. Many producers are reluctantly delayed in such cows.

to adopt such a high culling rate due to the high cost of As pregnancy progresses the lactational demand for purchasing or rearing replacement heifers, particularly a high level of dietary energy decreases. This enables

during the first year or so. However, such a policy can the cow to replace body energy reserves that were lost be of eventual financial advantage due to a decrease in during early lactation. Thus the cow can be brought the spread of calving and the consequent management back towards the target body condition for the next economics as discussed above (see Table 41.8). calving and subsequent mating.

Post partum onset of ovarian cycles

Controlling the length of the

A delay in the onset of ovarian cycles can lead to

calving season

extended calving intervals and possibly increased variation in calving intervals between cows within a herd.

There are a number of ways in which a compact sea-

*This will result in increases in the length of the calving
sonal calving pattern may be established and main-
season.*

*tained in a healthy seasonally calving beef herd. These
include feeding policy, culling policy and postpartum
induction of ovulation.*

Factors affecting the post partum

acyclic period

Feeding policy

Suckling

It is very difficult to restore compact seasonal calving patterns by nutritional management in herds that have
Many studies have shown that the onset of ovulation
a grossly extended calving season, since this would
and/or oestrous behaviour is delayed in either dairy or
Herd Fertility Management • 657

beef-type cows that suckle calves, relative to milked
suggested that photoperiod might play some role in
animals, particularly where more than one calf is
seasonality of reproductive activity in the cow and a
suckled per cow. In suckling beef cows kept under UK
negative correlation between daily photoperiod during
conditions the average time to resumption of ovarian
pregnancy and the onset of ovarian cycles after calving
cycles has been reported as 59.9 ± 2.5 days after calving
has been demonstrated (Peters & Riley, 1982b). It is
(Peters & Riley, 1982a), but there was considerable

possible that a vestigial sensitivity to photoperiod may variation both within and between herds. Weaning of be present in the domestic cow and that in feral cattle calves, either temporary or permanent, or at least the this pattern would predispose towards calving during prevention of suckling has been reported to shorten the the late spring to early summer, the optimal time for acyclic period.

food supply.

In summary, a variety of factors affect the onset of ovarian cycle in the post partum beef cow. The order of

Nutrition, body weight and condition

importance is probably nutrition, suckling and season.

and the post partum period

However, it is normally impossible in the practical

Long post partum acyclic periods in suckling cows may situation to quantify these effects so that the time to be reduced by the provision of increased dietary energy first ovulation can be predicted.

(e.g. Dunn et al., 1969). Energy intake appears to be more critical than protein intake in the maintenance of

Induction of ovulation in beef cows

reproductive function as positive relationships between

energy intake and reproductive performance have been

Most evidence to date suggests that delay in ovulation

demonstrated in several studies. Low energy intake in

during the postpartum period is mediated by a low rate

prepartum and post partum cows increases the length

of gonadotrophin release; most work has been done on

of the anoestrous period and in heifers it has been

luteinizing hormone (LH), but follicle stimulating

shown to result in fewer ovarian follicles, lower pro-

hormone (FSH) is probably equally important. Such

gesterone levels and lower conception rates (Hill et al., endocrine changes occur as a result of external factors

1970).

(some discussed above) acting via the hypothalamus

Nutritional status at and before calving appears to be

to suppress the release of gonadotrophin-releasing

more important than that during the post partum

hormone (GnRH).

period, since Peters and Riley (1982a), using body

A reliable hormonal method of inducing ovulation in weight as an index of nutritional status, found a significant negative correlation between body weight at calving and the length of the acyclic period in beef cows, been devised.

whilst body weight change after calving had no effect.

Gonadotrophins of non-bovine origin in the form of Also, an increase in energy supply to pregnant beef either pregnant mare's serum gonadotrophin (PMSG) cows has been shown to accelerate the return of ovarian and human chorionic gonadotrophin (hCG) have been cycles after calving. Target body condition scores are extensively used for this purpose in the past but results given in Table 41.7a.

have been variable; hCG has largely LH-like activity whereas PMSG has mainly FSH-like activity and may result in the development of multiple follicles.

Season

However, Penny (1998) suggests a dosage of PMSG

In the temperate latitudes seasonal variations in conception rates and a longer interval between parturition (see Table 41.9).

and first oestrus in the winter and early spring have

The injection of oestrogens in cows results in pre-

been reported (e.g. Thibault et al., 1966). Furthermore, ovulation surges of gonadotrophin release (depending

spring-calving beef and dairy cows have been reported

on dosage) and oestrous behaviour. However, ovula-

to undergo longer periods between calving and first

tion may or may not follow such treatment. The

ovulation than autumn calvers (Bulman & Lamming,

response is generally too unpredictable for this to be a

1978; Peters & Riley, 1982a). Most authors have sug-

gested that such seasonal effects are related purely to

Progestagens have been used extensively to synchro-

nutritional management; however, strong effects of

nize ovulation in cyclic cows, but may also be used

season have been demonstrated after adjusting statisti-

to induce ovulation after calving. Roche et al. (1981) call for the effects of body weight at calving (Peters &

reported that 10-day treatment of beef cows with the

Riley, 1982b). Evidence for seasonality in cattle has

progesterone-releasing intravaginal device (PRID)

now been accumulated from Europe, North America,

resulted in ovulation in about half the cows treated,

Canada and New Zealand. Thibault et al. (1966) have

whereas Bulman and Lamming (1978) reported a 75

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Table 41.9

Dose of PMSG at implant removal (Penny, 1998). Reproduced with permission of The British Cattle Veterinary Association.

Type of animal

Conditions

Dose of PMSG

at progesterone

withdrawal

Beef suckler cow

Calved <55 days

400 iu

Calved >55 days

Poor body condition score

(<2) 400 iu

Good body condition

score (>2) 250 iu

Beef heifer

None required under

normal conditions

PMSG, pregnant mare's serum gonadotrophin.

per cent success rate with a 12-day PRID treatment in

this treatment regime has been particularly successful

dairy cows. However, in the latter study the conception

in large-scale field trials with beef cattle (Mawhinney

rate in the responding cows was only 50 per cent and

et al., 1979). A short-lived rise in progesterone concen-treatment did not affect the mean calving-to-

trations following GnRH injection has been reported

conception interval. Other workers (Ball, 1982; Drew et

by several authors and may be compared to that occur-

al., 1982) have reported a reduction in the calving-toring naturally in some cows prior to the onset of normal

conception interval of up to 14 days following the use

ovarian cycles. There has also been interest in longer-

of PRID; however, the calving-to-conception interval of acting administration of GnRH, but no product is yet PRID treated beef cows was reduced only if used commercially available.

before day 30 after calving (Peters, 1982).

Mulvehill and Sreenan (1977) have reported the best success in induction of ovulation in beef cows by injecting 750 iu PMSG at the time of progesterone with-

Oestrous synchronization and AI in

drawal. However, as a small number of twin and triple **beef cattle** (see also p. 678)

ovulations did occur, Penny (1998) suggests a lower dose and a maximum of 400 iu (Table 41.9).

The full exploitation of genetic progress cannot be made in beef cattle without the use of AI. AI is not very widely used in beef cattle, but as oestrus synchroniza-

Gonadotrophin-releasing hormone

tion techniques have improved the number being

The injection of GnRH in cattle induces release of both artificially inseminated has steadily increased. There are LH and FSH. There have been many attempts to induce

several advantages of fixed-time AI (Penny, 1998) in ovulation in post partum cows by single intramuscular that it:

injections of 100–500 mg GnRH and these have given

- *Eliminates the practical problems of heat detection variable results. In order to apply these treatments in in beef cattle;*

practice, more consistent responses would be necessary.

- *Allows the use of superior sires with estimated*

At the above dose levels, LH release of pre-ovulatory breeding values (EBVs) (see p. 71) for important surge magnitude usually occurs, depending on the traits to improve the quality of the calf crop;

responsiveness of the pituitary. However, ovarian folli-

- *Allows the use of tested sires to breed replacement cles appear to require a two to three day period of rising heifers;*

plasma LH concentrations in order to mature fully

- *Allows the use of sires selected on ease of calving, prior to ovulation. Therefore, a pre-ovulatory LH surge thus reducing the likelihood of dystokia;*

will induce ovulation only if a follicle at the appropri-

- Helps to create a compact calving pattern;*

ate stage of development is already present. Alterna-

- Can be used to eliminate venereal disease.*

tively, the induced LH release might cause premature

luteinization of an unovulated follicle and transient

Anecdotal evidence suggests that AI pregnancy rate

secretion of progesterone sufficient to initiate ovarian

results on farm have been poorer than expected. Beef

cycles.

cattle tend to be less handled than their dairy counter-

Two injections of 500 mg GnRH at an interval of 10

parts. Mann (2001) suggests poor pregnancy rates in

days were advocated by Webb et al. (1977). Moreover,

dairy cattle were in part due to impaired ovulation as a

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Table 41.10

Pregnancy rates after various treatments and

Table 41.11

Summary of oestrous synchronization and breed-

insemination regimens (after Smith et al., 1984, with permission ing protocol

(Penny et al. , 1997).

from the American Society of Animal Science).

Day

Procedure

Treatment

Timing of AI

Percentage

after last

pregnant

-12

Insert crestar implants

treatment

-4

Inject prostaglandin

-2

Remove Crestar implant \pm PMSG

Control

Observed heat

72

0

First AI

6 day PRID plus PG day 6

Observed heat

82

12

Insert crestar implants

7 day PRID, PG day 6

Observed heat

73

21

Remove Crestar implants, take milk samples

2 ¥ PG

80 hours

52

23

Second AI if milk progesterone <3–5 ng/ml

7 day PRID, PG day 6

84 hours

66

35

Insert Crestar implants

44

Remove Crestar implants, take milk samples

PRID, progesterone-releasing intravaginal device; PG, prostaglandin.

48

Third AI if milk progesterone <3–5 ng/ml

PMSG,

pregnant mare's serum gonadotrophin; AI,

artificial

*result of 'stress'. In order to maximize the success of
insemination; Crestar, 3 mg norgestomet implant.*

synchronized AI, Penny (1998) advises the following:

- *Ensure that the animals are handled and that the
handling facilities are safe and efficient.*

Table 41.12

Results of the triple synchrony breeding pro-

- *As nutrition has probably the greatest influence on
gramme over two years (Penny et al. , 1997).*

*the success or failure of oestrous synchrony, ensure
that target calving condition scores are achieved*

Year 1

Year 2

and minimize body condition score loss between

calving and first service. This will result in the

Pregnancy rate 1st AI

56% (27/48)

58% (40/69)

majority of cows cycling prior to the synchrony pro-

Pregnancy rate 2nd AI

69% (11/16)

48% (11/23)

gramme. Sudden changes in the plane of nutrition

Pregnancy rate 3rd AI

40% (2/5)

33% (4/12)

Pregnancy rate to all

58% (40/69)

53% (55/104)

may reduce pregnancy rates due to nutritional

services

‘stress’.

Calving rate to all services

58% (40/69)

53% (55/104)

- Avoid major management changes during the pro-

Barren rate

17% (8/48)

20% (14/69)

gramme, e.g. turning out, housing, mixing groups and treatments such as vaccination.

AI, artificial insemination.

- Ensure that adequate bull power is available for repeats and, to reduce the risk of introducing venereally transmitted diseases, avoid hired or shared bulls.

inations 56 hours post progestagen removal were to cows which had a milk progesterone concentration of <3.5ng/ml. Overall pregnancy rates, for the two years,

Methods

were 58 per cent and 53 per cent. However, the barren

Both prostaglandins and progestagens may be used in rate was 17 per cent and 20 per cent (Table 41.12).

beef cows (Peters, 1986). Fixed time AI is not generally

Stevenson et al. (2000) compared combinations of

recommended, however. Programmes which have pro-

GnRH, Norgestomet and prostaglandin F2a. Fig. 41.2
duced acceptable pregnancy rates utilize a combination
shows the experimental procedure for oestrous syn-
of progesterone and prostaglandin (Table 41.10).
chronization. They found that prior to 60 days post
Prostaglandin administered 24 hours prior to progesta-
partum, programmes involving GnRH produced a
gen removal has the effect of synchronizing the decline
higher percentage of cows in oestrus and higher con-
in progesterone concentrations from endogenous and
ception rates compared to the programme involving
exogenous sources. Progestagen may either be in the
only two prostaglandin injections (Table 41.13).
form of a synthetic progestagen implant,
e.g.

Martinez et al. (2000) compared oestrous synchro-
Norgestomet, or a progesterone intravaginal release
nization and pregnancy rates in beef cattle given prog-
device.

esterone in the form of a controlled internal drug
Penny et al. (1997) described a triple synchrony pro-

release device (CIDR-B), prostaglandin F2a and oestradiol (Table 41.11) in a spring-calving, single-suckled beef herd used over a two year period. Natural service was not used and AI was at fixed times. Repeat insemination given a CIDR-B device plus prostaglandin F2a

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(PGF) at CIDR-B removal, and oestradiol or 100 mg GnRH, 54 hours after PGF, and concurrently gonadotrophin releasing hormone (GnRH). In experiment 1, cross-bred beef heifers received a CIDR-B

after onset of oestrus. The oestrus rate was lower ($p < 0.01$) in the GnRH group (55 per cent) than in either the progesterone (E + P group; $n = 41$), 100 mg gonado-

E + P (100 per cent) or control (83 per cent) groups. The mean interval from CIDR-B removal to oestrus was

further treatment (control group; $n = 42$), on day 0. On shorter ($p < 0.01$) and

less variable ($p < 0.01$) in the day 7, CIDR-B devices were removed and heifers

were

E + P group than in the GnRH or control groups. Pregnant treated with PGF. Heifers in the E + P group were given pregnancy rate in the E + P group (76 per cent) was higher 1 mg EB, 24 hours after PGF, and then inseminated 30

($p < 0.01$) than in the GnRH (48 per cent) or control hours later. Heifers in the GnRH group were given

(38 per cent) groups. In experiment 2, 84 cows were treated similarly to the E + P group in experiment I.

Cows received 100 mg progesterone and either 1 mg EB

Select Synchron

or 5 mg oestradiol-17 β (E-17) on day 0 and either 1 mg

GnRH

PGF

Detection of oestrus

2a

of EB or 1 mg of E-17 β on day 8 (24 hours after CIDR- and AI

B removal), in a 2 \times 2 factorial design, and were inseminated 30 hours later. There were no differences among

–7

0

1

2

3

4

5

6

groups for oestrus rates or conception rates. The mean interval from CIDR-B removal to oestrus was 44.2

Select Synch = NORG

hours, sd = 11.2. Conception rates were 67 per cent, 62

GnRH

PGF

Detection of oestrus

2a

per cent, 52 per cent, and 71 per cent in groups E-17b/E- and AI

17b, E-17b/EB, EB/E-17b and EB/EB, respectively. In cattle given a CIDR-B device and estradiol plus pro-

–14

–7

0

1

2

3

4

5

6

gesterone, treatment with either EB or E-17b effectively synchronized oestrus and resulted in acceptable

2 ¥ PGF2a (Control)

conception rates to fixed-time AI (Table 41.14).

GnRH

PGF

Detection of oestrus

2a

Penny and Lowman (2002) have designed a triple and AI

synchrony system for beef cows which relies on fixed-timed AI and not on natural service. The programme

–14

–7

0

1

2

3

4

5

6

allows cows up to three service opportunities over a

Days from PGF2a

46 day period (Table 41.14). Double insemination

improves pregnancy rate by 10 per cent compared to a

PGF2a: 25 mg Lutalyse (Pharmacia and Upjohn)

single insemination. GnRH (10 mg Buserelin) at the

GnRH: 100 mg Factrel (Fort Dodge) or Cystorelin (Merial Animal Health)

time of CIDR insertion gave an improvement in preg-

NORG (indicated by the shaded box): 6 mg Norgestomet

nancy rate of 12 per cent in treated cows. Cows which

– Syncro-Mate-B (Merial Animal Health)

were in a body condition score <2 (1–5 scale) or calved

<

Fig. 41.2

Experimental protocol for oestrous synchronization

55 days at first AI were injected with 400 iu PMSG.

(Reproduced from Stevenson et al. , 2000 with permission from PMSG was administered to maiden heifers and pro-the American Society of Animal Science).

duced a first service pregnancy rate of 74 per cent,

Table 41.13

The effect of oestrous synchronization treatment and number of days post partum on the percentage of cows detected in oestrus and conception rate (Reproduced from Stevenson et al. , 2000 with permission from the American Society of Animal Science).

Days post partum

Treatment

Select Synch +

Select Synch

Norgestomet

2 Ȳ PGF2 α

% Oestrus

% CR

% Oestrus

% CR

% Oestrus

% CR

£60

44.2

50.0

44.1

45.8

25.0

8.3

61–70

62.7

74.4

72.4

60.5

50.6

65.8

71–80

65.1

60.3

84.1

58.9

56.6

61.7

>80

60.0

73.8

72.4

64.0

46.0

72.4

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Table 41.14

Triple synchronization programme for beef cows (Penny & Lowman, 2002).

Day

Day

Action

0

Thursday

Insert progesterone + GnRH

7

Thursday

Inject prostaglandin PGF2a

9

Saturday

Progesterone outa (am) + PMSGb

11

Monday

1st AI (am)

12

Tuesday

1st AI (am)

22

Friday

Insert progesterone in late calversc + GnRH

27

Wednesday

Insert progesterone

29

Friday

Inject prostaglandin PGF2a (late calvers)

32

Monday

Progesterone out (am) and tail paint (\pm PMSG late calvers)

33

Tuesday

Heat detect carefully

34

Wednesday

2nd AI (am) any cows seen bulling + late calvers

35

Thursday

2nd AI (am) any cows seen bulling + late calvers

46

Monday

*Scan cows assumed pregnant and insert new progesterone in any non-pregnant
+ GnRH*

50

Friday

Insert progesterone in 2nd AI group

53

Monday

Inject Prostaglandin PGF2a (scanned empty cows)

55

Wednesday

Progesterone out and tail paint

56

Thursday

Heat detect 2nd AI group

57

Friday

3rd AI (am) any cows seen bulling + non-pregnant group

58

Saturday

3rd AI (am) any cows seen bulling + non-pregnant group

a Rinse briefly in dilute antiseptic then dry with paper towels and store in cool dark conditions; wear disposable gloves when handling progesterone.

b 400 iu PMSG given to thin cows CS < 2 or calved >55 days.

c Late calving cows enter the programme for the second AI and therefore only have 2 opportunities for AI. Progesterone CIDR InterAgART.

GnRH 10 mg Buserelin Receptal Intervet.

which was better than untreated heifers (pregnancy

advantage in heifers along with fixed-time insemination

rate not published). The authors postulate that

if required, although a bull would still be necessary to

anoestrus may be a real problem in 15 month-old

serve non-pregnant animals subsequently.

heifers of later maturing beef breed crosses. They con-

clude that a target of 90 per cent calving rate is achievable and repeatable in a 46 day breeding period and

Monitoring reproductive

leads to a sustainable compact calving period.

performance in the beef herd

As beef cattle are less used to being handled they can become fractious when being confined and restrained.

This is very much more difficult in beef cattle than in

At times of hormonal treatments it is essential to

dairy cows because of the nature of the management provide suitable handling facilities and sufficient experience systems.

rienced labour. If these are not provided there is con-

Even in the best-managed units, records of repro-

siderable danger to both animals and humans. Where

ductive events may be quite rudimentary, possibly

programmes involve progestogen implants, either as ear

including only date of calving. Since bulls are widely

implants or intravaginal devices, it must be remem-

used few records of service may be kept. The bull would

bered that these may be lost prior to their planned

normally be left with the cows for a length of time that removal. There are also welfare considerations and corresponds to the aimed length of the calving season. consumer concerns when subjecting animals to injections and implants. Whilst it is advantageous to identify barren cows at the

end of the bulling period, in many cases it is not desirable to cull those animals because they are still suckling calves. However, methods of early pregnancy diagnosis

Management of heifers (see Chapter 5)

are highly advantageous in the beef herd so that non-pregnant cows may be identified quickly and appropriate action taken. It is unlikely that the milk

Heifers should be bred to calve three to four weeks before the adult herd. Heavy breeds of bull likely to progesterone test will become popular in beef herds, cause dystokia, e.g. Charolais and Simmental, should be even though a 'cow-side' test is available, because of avoided. Oestrous synchronization may be used to potential difficulties in sample collection. However, the

use of real-time ultrasound where embryos/fetuses can

- An assessment of bull capacity including their ages
be detected by day 30 (White et al., 1985) or earlier
and clinical histories;*

offers very exciting possibilities.

- Evaluation of the other farm enterprises so that the
It would be desirable for the practitioner to design a
best calving season can be chosen.*

*‘herd plan’ to monitor reproductive performance in
Beef cow stockworkers are capable of achieving rates
beef herds. The following could be used as a guide.
of heat detection equal to those of dairy stockworkers.*

(1)

Condition-score cows at least two months before

*If reproductive performance is to be optimized then the
the start of the calving season, e.g. at weaning.*

stockworker must be prepared to observe and record

Those below target (Table 41.7a) should receive

*the herd. For those who do so, techniques such as AI,
supplementary rations to bring them up to target.*

oestrous synchronization/induction and induction of

Any cows above target should have their condition reduced.

The veterinarian should be able to assess the potential
(2)

Condition-score cows at calving; those below target should receive supplementary rations.

should be on a long-term basis, i.e. five years, by which
(3)

Special attention should be paid to first and second calvers since these are most likely to be

time optimal targets should have been reached and

advisory input can be reduced to a surveillance role.

vulnerable to problems, particularly extended

Where herds have an extended calving pattern it may

periods of anoestrus after calving.

take several years of planning and reproductive management
(4)

The producer should begin to observe and record

pupulation before a herd achieves a two month calving

oestrus three weeks before the bull is introduced.

period:

(5)

Oestrus periods and services should be recorded

- *Calve heifers to an easy-calving bull one month throughout the breeding season. Particular attention should be paid to the dates when cows are*

tion should be paid to the dates when cows are

- *Body condition-score cows regularly; if they are too expected to return to oestrus. The use of chinball thin increase feed and/or wean calves early.*

markers may assist oestrous observation with

- *For late calving cows, manipulate oestrus and use cows at grass.*

AI soon after calving.

(6)

Pregnancy diagnosis should be carried out six

weeks after the bulls are removed. Barren cows

can then be culled when weaned. The findings at

pregnancy diagnosis in conjunction with the

(b) Dairy Herds

*service records will enable the farmer/veterinarian
to estimate the likely calving dates and to plan the*

D.C. Barrett and H. Boyd

nutritional management.

Fertility problems arising in beef herds are likely to

Introduction

662

result from causes similar to those in the dairy herd.

Reproductive performance in the UK national dairy herd

663

However, investigation is often hampered by the lack

Components of a fertility control programme

664

of adequate records. Clinical problems such as retained

Identifying herd-specific goals

664

placenta, cystic ovaries and metabolic disease are much

Planning and setting targets

664

less likely to occur, but otherwise the more common

Cow identification, records, data analysis and

*problems are similar to those in dairy herds. Similarly,
interpretation*

667

when problems occur they are likely to reflect herd

Regular veterinary visits

668

status rather than just involving individual cows.

Periodic review and regular evaluation of performance

669

Maintaining motivation

672

*Advice for veterinarians who wish to start herd fertility
control programmes*

672

Reproductive record keeping and analysis

672

Analysing herd problems

If the client is aiming for a 12-month calving interval

673

If the client is aiming for a tight calving season

673

Future developments in herd fertility management

675

The following information is likely to be of value in understanding and rectifying problems.

- *Cumulative frequency curve of calvings;*

Introduction

- *The age distribution of cows;*
- *The calving dates of first-calving heifers;*

A fertility control programme is a system to monitor

- *The culling rate and reason for culling;*

and manage reproduction, and is a well-established

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element of preventive medicine in dairy herd manage-

Reproductive performance in the UK

ment that is successfully practised in many parts of the

national dairy herd

world. In a successful programme the farmer gains an

improved income more than the cost of implementing

There has been a general decline in fertility in the UK

the programme (Esslemont et al. , 1985). For the veterinal dairy herd over recent years, emphasizing the

narian, a well-managed fertility control programme growing need for cost-effective veterinary intervention should represent a profitable outlet for specialist professional skills. In addition, regular farm visits to carry out fertility related work allow the veterinarian the opportunity to promote other aspects of preventive medicine, both within the dairy herd and also to other livestock enterprises on the farm.

Analysis of approximately 9000 herds (National Milk Records (NMR)) representing data on around 1 million cows, between January 1997 and September 2000, shows that the average herd size has risen from 97 to 112 cows, and that the herd average yield has risen from below 5500 kg to approach 7000 kg per annum. Over the same period the mean calving to first service interval has risen from 28 to 32 days, and the mean calving to first service interval has risen from 28 to 32 days, and the mean calving to first service interval has risen from 28 to 32 days.

Reproduction is particularly suitable for a preventive approach because if intervention is delayed too long there will be an irretrievable loss of production time and thus a financial loss to the producer.

risen from 84 to 88 days, the mean calving to conception interval from 113 to 121 days (Fig. 41.3) and the and the veterinarian, and is based on good husbandry mean calving interval from 391 to 399 days (Fig. 41.4). and consistently efficient work by the veterinarian.

There also seems to be a downward trend in first service Because of the effect of husbandry (feeding, housing pregnancy rate of approximately 3 per cent over the and breeding management) and general health on same period (Barrett, 2001). Royal et al. (2000) and reproduction, a fertility control programme should also Esslemont and Kossaibati (2000a) have also reported incorporate other aspects of preventive medicine and similar trends of falling fertility performance in the encourage the practice of good husbandry. The devel- UK and O'Farrell and Crilly (1999) have documented opment of herd health plans such as the British Cattle Veterinary Association (BCVA) Herd Health Plan over recent years (Anon, 1999) has been a significant step forward in this regard. The instigation of a herd health

Box 41.1

The main components of a successful fertility plan allows the veterinarian to influence all aspects of control programme.

husbandry and preventive medicine within the herd.

Furthermore, as most aspects of husbandry and health

Identifying herd specific goals

care will impact in some way on fertility and reproduc-

Planning and setting targets

tion, this holistic approach to preventive medicine

Cow identification, records, data analysis and interpretation

will aid and support the fertility control programme.

Regular veterinary visits

Periodic review and regular evaluation of performance

Box 41.1 lists the main components of a fertility control

Maintaining motivation

programme.

124

122

120

118

116

interval (days)

114

Mean calving to conception

Fig. 41.3

NMR data from approximately

112

9000 UK dairy herds showing the trend in

mean calving to conception interval

110

between January 1997 and September

Jan-97

Jul-97

Jan-98

Jul-98

Jan-99

Jul-99

Jan-00

Jul-00

Jan-01

2000.

Date

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400

398

396

394

Mean calving interval (days) 392

Fig. 41.4

NMR data from approximately

390

9000 UK dairy herds showing the trend in

Jan-97

Jul-97

Jan-98

Jul-98

Jan-99

Jul-99

Jan-00

Jul-00

Jan-01

mean calving interval between January

Date

1997 and September 2000.

lowered fertility in Irish dairy herds. This declining fer-

gramme for the herd to ensure milk is produced at the

tility may be due to a number of factors such as high

most profitable time, maximizing financial margins

levels of peri-parturient disease (Peeler et al. , 1994a, b), per litre as well as ensuring an optimal culling rate

inadequate nutrition (Webb et al. , 1997, 1999; Wathes with minimum involuntary culls and the production of

et al. , 1998), increasing genetic merit for milk produc-sufficient replacement heifers.

tion (Nebel & McGilliard, 1993, Lamming et al. , 1997; Given that producers will sell their milk to different

Lucy & Crooker, 1999), infectious diseases or numer-

milk purchasers who have different price structures, it

ous other independent or inter-related factors. Specific

is important to ascertain by discussion with the farmer

breeding management changes such as the increase in

and his other advisors what reproductive pattern will

the use of do-it-yourself artificial insemination (AI),

deliver the best overall profitability. For example, a low

with variable amounts of training of personnel, may

input, low to moderate yielding herd of 5000 to 6000

also be significant (Howells et al. , 1999). Increasing milk l/cow per year looking to maximize the utilization of

yields and increasing herd sizes are almost certainly

grazed grass may be looking for a tight seasonal calving

contributing to the problem. It is also highly likely that

pattern with a calving index (mean calving interval) of

the decreasing availability of labour on farms is result-

365 days. In contrast, a farmer attempting to produce a

ing in reduced labour hours per cow per year. The

uniform supply of milk each month, purchasing a large

overall effect is greater difficulty in getting cows in calf

proportion of the feed used and running an intensive,

and increased costs of trying to do so.

high yielding herd of 10 000 l/cow per year may desire a

year-round calving pattern and a calving index of 420

days. On the other hand a pedigree breeder producing

embryos for sale may have general goals for the major-

Components of a fertility

ity of his herd, but very specific reproductive objectives

control programme

for his embryo donor cows. Having agreed the overall reproductive strategy that suits a given herd, herd

Identifying herd specific goals

specific reproductive targets can then be set.

It is no longer possible to assume that every herd owner's reproductive targets are the same. Targets will

Planning and setting targets

be determined by various factors fundamental to the production system employed, for example contractual

A fertility control programme requires reproductive agreement with milk purchaser, seasonal milk price, targets. As outlined above, these are usually chosen for milk constituent prices, feed costs, availability of labour, economic reasons but are limited by what is physiologically possible. It is the collective responsibility of the producer will almost always be to maximize financial farmer, his veterinarian and others in the farm advisory return within the constraints of milk quota or production economics. Fundamental to achieving this is having

should ensure as far as possible that the targets are a predetermined, and managed, reproductive pro-realistic and achievable within an agreed time span.

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There is no point producing targets that do not take into

Once the age of calving has been decided, other con-

account bovine reproductive physiology or that are

sequential targets have to be considered. If a heifer has

unduly optimistic concerning pregnancy rate, oestrous

to calve at 24 months old she must become pregnant at

detection rate or the standard of husbandry on the

15 months old. To do this she has to be cycling and well

farm. It is better to set a realistic target, achieve it and

grown (see p. 58). She also has to keep growing to be

then set a more demanding target than to begin with a

in a fit condition to calve down and be big enough to

target that is unrealistic and totally out of reach, as this

thrive in the dairy herd in competition with adult cows,

reduces stockman motivation. The farmer and his staff

or at least able to lactate, continue growing and get back

have to be in full agreement with the targets set, as only

in calf at the optimum time when managed in a group if they really want to achieve the selected targets are of first lactation cows. This means the farmer has to plan they likely to be met. The veterinarian can only help the growth rate from birth to meet the targets at these and advise the stockmen on the farm, and cannot critical times (see p. 56). The veterinarian has an important role in reducing calf and growing cattle diseases to alone. A team spirit has to be engendered and each allow planned growth rates to be achieved.

member of the team must play his or her part fully. Other breeding targets have to be established, for example an acceptable culling rate and the choice of sires used. One aim is to avoid dystokia and this should

Heifers

be considered in choosing the breed of bull and Dairy farmers often aim for seasonal calving for the perhaps, more importantly, the individual bull to be whole herd. To achieve this, it is essential that the used for service. Under conventional breeding pro-

heifers calve early in the chosen season. As age at first
grammes the generation interval is one of the major
calving has a major effect on the proportion of a cow's constraints to genetic
gain. There are advantages
life that is productive, there is an optimal age for first
in breeding replacement heifers out of heifers as this
calving and, for modern dairy breeds in Britain and
minimizes the generation interval and thus maximizes
elsewhere, this is usually put at 24 months old (see
genetic gain within the herd (see p. 59). If proven dairy
Chapter 5). However, farmers have to consider condi-
sires are used by artificial insemination (AI) they
tions in their own herds before they decide at which age
should be selected for ease of calving and be of higher
the heifers should calve. Seasonal calving herds may
genetic merit than the heifers being bred. At the
have to calve heifers at approximately 24 months of age,
present time health traits are gaining in importance
as the alternative of 36 months is usually not economi-
in the selection of dairy cattle, and in the future they
cally viable (see Chapter 5). However, year-round

may be seen as equally, or even more important, than calving herds or herds with two main calving periods production traits, so in this context genetic merit can be more flexible. For example, some may elect to encompass both health and production traits.

calve their heifers at 27 or 30 months of age.

It may also be a target to use beef sires on heifers, to Having chosen the desired season of first calving and allow the use of natural service or to avoid replacement the desired age at first calving, various problems arise dairy animals being produced from dams whose pro- that have to be considered. If, for example, the calving ductivity is unknown. This approach may also be used season chosen is September, October and the beginning to avoid too many dairy calves being born to the herd, of November and the age at first calving is to be 24 particularly as pure bred dairy bull calves are of little months, what is to be done with heifers that are born or no value.

late in December, January or February? Will they be left until the next calving season? Will they be sold

Cows

either as calves or perhaps later as in-calf heifers? Will they be made to calve even younger than 24 months old

The most important targets are a controlled calving or will they be allowed to calve in January or February interval and season of calving, as these will impact and extend the herd's agreed calving pattern outside directly on profitability by influencing the mean yield the optimum time period? These are important and difficult decisions that have to be considered when the milk produced. In addition, minimizing the involuntary overall goals of the fertility control programme are culling rate and allowing strategic voluntary culling are agreed. Strategic decisions of this type have to be necessary.

thought through, for example the above scenario can be

In fertility control work, the calving interval is the avoided completely if replacements are only bred from

number of days from the immediately past calving to cows and heifers served during the agreed breeding

the next. Farmers are very conscious of the need to have season, and repeat breeders and late calving cows are a controlled calving interval, but the ideal target varies either culled or bred to a bull of a beef breed.

from country to country, from farm to farm and indeed

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to derive the cost-benefit of any alterations in fertility

Box 41.2

Factors influencing the choice of calving interval.

parameters including an extended calving interval; the

Age and breed of the cow

principles behind this are discussed more fully by

Yield and shape of her lactation curve

Dijkhuizen et al. (1997).

Month of the year

Once the target calving pattern and target mean

Feeding

calving interval (calving index) have been chosen a

Cost

series of consequential targets emerge. If the target

Availability

calving index is, for example, 365 days, then the target

Calving and milking facilities

for calving to conception must be 85 days, which is 365

Milk sale contract and differential pricing structures

Milk quota

days minus a mean gestation length of 280 days. It

Calf value

should be noted that if the most profitable calving inter-

val for an individual cow is 365 days, then the aim in

theory would be to have every cow calve with that inter-

val. In practice what happens is that a herd calving

from lactation to lactation. In Britain the normal target

index becomes the target. However, care should be

for average yielding herds is a 12-month calving inter-

taken to minimize the distribution around this 365-day

val although, as discussed above, this may on occasions

mean figure, as cows with both longer and shorter

be extended for strategic reasons. Box 41.2 lists factors

calving intervals will be performing below their

that influence the choice of calving interval.

optimum. As the calving index is the mean interval

There are two reasons why calving interval is between two calvings it only relates to those cows that important.

calve again. In a herd with a 25 per cent replacement rate only 50 per cent of the herd will contribute to the (1)

Typically, milk yield reaches a peak about five calving index, as it takes no account of the fertility of weeks after calving and starts to decline at about heifers or cull cows, and can be manipulated greatly by eight or nine weeks after calving. This is why the culling. It is important therefore always to interpret the first part of a lactation is more profitable than later calving index alongside the culling rate for failure to parts and the dry period. Short calving intervals conceive. The calving index as a measure of fertility in result in a greater proportion of a cow's produc- a herd is also too historical to be of much use in monitoring life being spent in this early, more profitable toring fertility, as it relates to the fertility of the herd part of lactation.

over a year before. A better and more contemporary

(2)

Short intervals produce more calves over a given measure is the calving to conception (more correctly period of time than long intervals. In a 100-cow termed calving to establishment of pregnancy) interval herd with a 12-month calving index there will be as it relates to the current year rather than the previous (approximately) 100 calves born per year; in a year.

similar herd with a 14-month calving index the Account must be taken of the fact that many cows number of calves per year will be about 86.

require more than one service per pregnancy when cal-

However, longer calving intervals should not necessarily culating the target interval from calving to first service.

ily be considered a bad thing. If lifetime production

In an individual herd this depends on conception could be maintained while reducing the frequency of rate, oestrous detection rate and culling rate. In many calving of dairy cows there may be benefits. As many of

herds a target for calving to first service of 65 days is the disease problems seen in dairy cows occur around satisfactory.

the time of calving or in early lactation, having a system

It is clear that cows must be cycling by the target date

of extended lactation that reduces both disease in the

for first service. A target that is often taken for the start cows and the numbers of low value calves produced

of cyclicity based on the physiology of the dairy cow is

may bring significant welfare benefits to the dairy

42 days after calving.

industry (Knight & Sorensen, 1998).

It is quite obvious that a target of first service 65 days

It is tempting when marketing fertility control to put

after calving has to be modified to become the oestrous

a money value on the loss occasioned by every day the

cycle that straddles 65 days, often taken as 56–76 days

calving interval is extended beyond the target. A figure

inclusive (eight to eleven weeks) after calving. This

that has remained remarkably constant over the

modifies the target for calving to conception to the

last decade (to 2002) is £2.50 to £3.00/day. It should be three-week period around 85 days, i.e. up to 96 days realized that this (or any other generalized figure) is after calving. It follows therefore that cows should be not necessarily applicable to any individual herd.

served to the first observed oestrus after 56 days post

Where possible, herd specific costings should be done calving. In other words, the voluntary waiting period in Herd Fertility Management • 667

this example should be 55 days. Shortening this period systems there is still the need for a stockman to be able will allow cows to be served more quickly, but in general to identify each animal readily and rapidly in the herd it will result in poorer conception rates.

in all housing and pastures used without risk of error or

Many farmers have as their prime target the season the need to resort to specialist equipment.

of calving, which tends to dictate a 365-day target calving index within a predefined service and calving

Record systems

period. The emphasis tends to be different from farmers

aiming for a different calving index, although the
A great deal has been written about record systems,
method of calculation of target indices is in essence the
ranging from simple, individual cow card-based
same. The start and end of the service season is con-
systems, other paper-based systems and wall mounted
trolled by the targeted calving season. It is essential that
breeding boards (Anon, 1984) to detailed state of
as many cows as possible are served in the first three
the art computerized systems such as National Milk
weeks (i.e. one oestrous cycle) of the breeding season.
Records' 'InterHerd' software. Simple systems for
It is particularly important to have all the cows cycling
beginners are described at the end of this chapter
in time and therefore the target for cyclicity is the three
(see p. 673).

weeks before the start of the service period. Submission
rates (defined as the proportion of cows eligible for

Why are records needed?

service served in a defined time period, usually 21–23
days) during the breeding season of seasonal calving

(1)

Records are needed for efficient day-to-day herds must be optimized, therefore oestrous detection running of the herd, even in herds with no is paramount and every effort has to be made to ensure thought-out plans and targets. They tell the stock-that cows are served.

worker whether a cow that is in oestrus is due for service or should be left until the next oestrus; they indicate when a cow is expected to come in season from the recorded date of last oestrus or

Cow identification, records, data analysis

service and they allow the accurate selection of and interpretation cows for the veterinary visit or for sampling.

A clear, reliable cow identification system is needed to (2)

Records are needed to monitor progress. It is necessary to run a fertility control scheme. Good identification is necessary to keep an eye on herd progress as well as needed for accurate records.

individual cow status. Monitoring will indicate

Without proper identification there will be no clear how many cows have started cycling on time after system to ensure that cows are served when planned, calving, how many cows have been served on time no way of knowing which animals fail to meet targets and it will show the success rate of services and and dangerous confusion when animals are presented the incidence of long and short interservice intervals for veterinary examination. As an example of the latter point, consider a cow that has an unrecorded service, is veterinarian the chance to deal with herd problems at an early stage.

seen in oestrus'. She will have a corpus luteum, and the

(3)

Accurate records analysed and interpreted correctly are an essential aid to the diagnosis of herd fertility problems. Records may be analysed for

sented for pregnancy diagnosis on the basis of a known the whole herd, or if cows are grouped according service date had an unrecorded service at a later date, to factors that may be related to fertility it is some- even the most experienced veterinarian could make an times possible to pinpoint aetiological factors erroneous diagnosis of 'not pregnant'.

which may have temporal or other links. Exam-

Although it is now compulsory within Europe that all ples include where one bull has a poor pregnancy cattle are tagged in both ears with a unique identifica- rate, or the mean calving-to-first-service interval is tion number, this may not allow rapid identification of very long in first calvers or if a seasonal effect on cattle at pasture or within a large group during routine fertility is noted.

handling. The veterinarian should encourage the farmer to choose an identification system suitable for the con- What are records?

ditions in which the herd is kept. Under most situations this will mean freeze branding, although other methods

Records consist of the following information written such as numbered collars may be utilized in some down by a farm worker: a cow number, a date and an systems. Even with the advent of electronic tagging event. Errors of two sorts occur: items are written down

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Box 41.3

The main fertility/reproductive/production events

Box 41.4

Selection of cows for routine visit (the stage for to record.

examination or intervention figures are in brackets).

Calving, oestrus, service, result of service, abortion

Cows with abnormalities at or after calving (21 days after

Veterinary reproductive findings and treatment

calving); some will examine all cows about 21 days after

Other diseases

calving, although this is probably unnecessary

Production information

Cows not seen in oestrus (≥ 42 days) after calving

Milk yield

Cows in target service period but not yet served (56–76 days

Feeding

after calving)

Cows overdue for service (≥ 77 days after calving)

Cows due for pregnancy diagnosis (≥ 42 days after last service or earlier if ultrasound examination used)

incorrectly and events occur and are not recorded. The

Cows seen at the previous visit and not yet served

main events that are recorded in variable detail are

Other problem cows, e.g.

listed in Box 41.3. Even on farms with the poorest

Purulent vaginal discharge

records, many of the required data should be available.

Aberrant sexual behaviour

For example, the number of cows in the herd, culling

Repeat breeders

records and calving records have to be recorded for

statutory purposes (data required by the British Cattle

Movement Service). If AI is used the AI company can

provide data on service dates. This, along with a veteri-

Regular veterinary visits

nary examination of each cow to determine pregnancy status, will provide enough information to calculate, or At each farm visit, the owner will have selected animals estimate, basic fertility parameters for the herd.

for examination based on an 'action list' generated using a system agreed between the client and veteri-

Problems in record analysis: There are two intrinsic, narian. Where a computerized data management

insurmountable problems in relation to data analysis.

system is in place the computer software may generate an action list based on predetermined agreed criteria.

(1)

With very few exceptions the amount of data

The animals presented for examination will vary from available, when taking into account the variability

herd to herd and between practices. The groups of cows of all fertility measurements, is not enough to give

shown in Box 41.4 are examples of cows that may be statistically valid information. This is particularly

presented at the regular visit.

the case when the records are subdivided into age

Methods of examination, diagnosis and treatment

groups, seasonal groups and so on.

have been discussed in Chapter 35. At each visit the

(2)

Information that is up-to-date is practically always

clinician should spend a few minutes looking over

incomplete (see below) and as a result may

the records with the client to pick up any problems at

produce incorrect conclusions; complete informa-

an early stage, to modify targets, to give information to

tion is nearly always out-of-date.

the farm staff and to give them the chance to discuss

Up-to-date, incomplete information will give a more

any veterinary problem that is worrying them.

favourable result than is true in the measurement of the

time from one reproductive event to another. Consider

Records kept at the visit

the calculation of the herd mean and spread of the

interval from calving to first service. If the figure is cal-

Apart from the breeding records the veterinarian

culated before all the cows in the calculation have been

should keep a simple record sheet that records every-served (or marked as 'not to be served') the result will thing done and advised, an example is shown in be biased because it will include only cows served soon Fig. 41.5.

after calving and will exclude those problem cows that Whatever method is used to transfer the veterinary will eventually be served after a long calving to first findings to the records, care should be taken with cows service interval. Incomplete pregnancy data based on that are found to be non-pregnant to make sure that it non-return rates or on extremely early pregnancy diagnosis is clear to which service 'non-pregnant' applies. Errors arise when the cow is served later and the result 'non-real picture.

pregnant' may be taken to apply to that subsequent These errors matter because decisions can be based service. Where a bull is present on the farm, a diagnosis on false assumptions. The actual analysis of records is of 'pregnancy not detected' is safer than non-pregnant.

further discussed in the section on periodic review and

The quality of the work done at the farm visit

the regular evaluation of performance.

together with the informal discussion of progress and

Record of veterinary visit.

problems is very important. It acts not only to detect interest and with the apparent success rate as noted at and treat problem cows and gather fertility and related each visit. The basis of the periodic review is an analysis of the herd breeding records and an assessment of the advice given at the periodic review.

the effectiveness of all interventions. Cows in the milking herd will be considered separately from heifers.

There are so many ways of recording and analysing

Periodic review and regular evaluation

herd breeding records that this simple exercise can be of performance

unnecessarily confusing. There are two reasons for

Detailed descriptions of the many different ways of analysing herd records in these periodic reviews:

gathering and analysing breeding records are easily

- Assessment of current status to establish whether obtained in other publications (Anon, 1984; Esslemont there is a problem that needs attention or whether et al. , 1985; Brand & Varner, 1996a, b; Bailey et al. , 1999).

things are progressing satisfactorily.

Instead of repeating them here, attention is concen-

- *Discovery of any particular areas of weakness.*

trated on what the analysis is trying to achieve and on some of the problems of interpreting records.

In the analysis of purely reproductive data (not

While at each visit an eye is kept on the progress of factors which affect reproduction) there are five analyses that can be carried out, as listed in Box 41.5. The most important of these are marked with an asterisk.

herd. The intervals between periodic reviews will vary

Even in herds where overall performance is adequate with the calving pattern, with the intensity of the client's there may well be subsections of the herd where both

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Box 41.5

Analysis of reproductive data.

Box 41.6

Measurement of success rate of service.

Intervals in days between calving and subsequent reproduc-

*Percentage (pregnancy rate): 'successful services' of 'all
tive events*

services', e.g. 55%

Calving to first observed oestrus

Ratio (services per pregnancy): 'all services' to 'successful

*Calving to first service**

services', e.g. 1.82

*Calving to conception**

Visually: as a cumulative sum (Anon, 1984) – visual and

Pregnancy (conception) rate or services per pregnancy

allows a degree of temporal analysis

*(conception)**

*Intervals in days from service to service (interservice
intervals)*

Involuntary culling rate (reasons for culling)

*Submission rate (in herds with short breeding season)**

*The mean is of limited value because it may be made
up of a wide range of values. For example, a mean*

** Most important reproductive data.*

calving to first service interval of 65 days may consist of

very short and very long intervals (unsatisfactory) or it may be the result of most intervals being close to the target (satisfactory). If the mean is greater than the farmer and the veterinarian should be concentrating their efforts. Analysis of those data relating to herd mean does give some idea of the seriousness of the subsections may reveal these areas of weakness.

situation. However, calculation of a mean \pm standard deviation for each parameter would be better, with the number of animals.

target standard deviation as small as possible.

The first step is to define clearly which cows are to be included in the analysis. The ideal baseline is all cows been developed. For example, instead of a 65-day target that calve in a specified period. This period has to end for a mean interval from calving to first service, the long enough before the day of analysis to allow every target changes to having as many cows as possible

cow to have completed her reproductive cycle to the
served in the oestrous cycle period around 65 days, i.e.
stage of pregnancy diagnosis or to the decision to cull.
from 56 to 76 days (8–11 weeks) after calving. The
Once the group to be analysed has been selected, it
target for conception is the period around 85 days, i.e.
is important that the analysis makes it clear what has
76–96 days (11–13 weeks) after calving. For first oestrus
happened to every cow in the group.

it is best to aim for as many cows as possible seen in
oestrus by 42 days.

Information about the number of animals that fall

Calving to subsequent reproductive events

into the target period, before the target period and after
(to first oestrus, to first service, to conception)

it can be presented in histogram form. This is very

It is simple to calculate the number of days from calving

clear, easily understood and puts the extent of the

to first observed oestrus, to first service and to concep-

problem into exact numbers. This is particularly easy

tion (the date of the service that resulted in a confirmed

with modern software spreadsheet programs such as pregnancy). It is more difficult to express this information in a clear way and to interpret the results correctly. Microsoft (MS) Excel and some of the specialist software available for herd record analysis.

The first thing to note is how many cows actually had a first observed oestrus, had a first service or became pregnant, in relation to the number of animals that started in the group. Culling rates of about 25–30 per cent are common in dairy herds. If the rate is much greater than this, the veterinarian should establish whether something is going wrong in the herd or in Box 41.6. The difficulty arises because of the different ways of defining ‘all services’ and ‘successful services’.

The success rate of services is again quite simple to work out, but to avoid misinterpretation it is essential to understand the different ways of doing this, as shown in Box 41.6. The difficulty arises because of the different ways of defining ‘all services’ and ‘successful services’. ‘All services’ can mean literally that; it can also

The next thing to calculate is the mean number of services’. ‘All services’ can mean literally that; it can also

days from calving to oestrus (target: cycling by 42 days) mean all services up to a certain number of services, say

to service (target: 65 days) and to conception (target:

five, or it can mean 'all first services' (first service is

85 days). Targets assume that the object is to have a

defined as the first service after calving or in a heifer's

12-month calving interval. While the means are not

life). 'Successful services' can mean services that result

completely satisfactory numbers they are worth

in calving, or that result in positive pregnancy diagno-

knowing and comparing with targets.

sis by rectal palpation or ultrasound examination, or

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positive pregnancy diagnosis by milk progesterone or

of 18–24-day returns is quite useful for assessing

by non-return to service at various intervals after

levels of oestrous detection.

calving.

• Observation of the incidence of irregular intervals,

Depending on the definitions used the success rate

such as <18 days and between 25 and 36 days, gives

calculated from the same group of animals can vary an indication of the frequency of inaccurate heat enormously.

detection. However, it should be recognized that In most herds that are under veterinary supervision these irregular intervals may be brought about by 'success' will be defined as positive pregnancy diagnosis late embryonic death or may be artefacts due to sis undertaken by the veterinarian using ultrasound veterinary intervention to manipulate the oestrous examination or rectal palpation. There are several cycle.

points in favour of basing the success rate on first services The proportion of cows presented for pregnancy ices rather than on all services, in particular:

diagnosis that are not pregnant also gives an indication

- The result can be obtained earlier than if one has of the number of missed oestrous periods because most to wait for the result of the last repeat breeder in non-pregnant cows have been in season once or twice the group.*

before examination and have not been observed in

- *Each cow contributes the same weight to the oestrus. Another indicator of oestrous detection efficiency is the submission rate, defined as the proportion of cows eligible for service served in a defined time*

Therefore, in herds controlled by a veterinarian, success period, usually 21–23 days. This measure is particularly

rate is best defined as the first service pregnancy rate, useful in seasonal calving herds.

i.e. the percentage of cows that are pregnant to first service. It should be borne in mind that an accurate figure will not be obtained until the result of the last

Culling rate

first service is known, which may be about six weeks after that last service.

The incidence of culling in relation to the total starting

Once the first service pregnancy rate has been

population and the stage at which the decision to cull

worked out it is compared with the target rate of

is taken is presented as a percentage and an actual

between 45 and 60 per cent, the target depending on a number. It is desirable to record the reason for culling. variety of factors.

In addition to first service pregnancy rate, at the end

Effectiveness of treatment and accuracy of

of a breeding season it is valuable to calculate an all-

pregnancy diagnosis

service pregnancy rate as it is this that will dictate the number of straws of semen used and the total insemination-Treatment, including conservative treatment, takes

nation charges. The all-service pregnancy rate will give

place:

a measure of the overall fertility within the herd, but

- At and after calving

should be interpreted in light of other information such

- In the preservice period and

as the culling policy for failure to conceive.

- At and after service.

Lists should be prepared with appropriate dates, diag-

Interservice intervals

nosis, treatment and outcome following treatment.

Many veterinarians who have not previously

The number of days between services is worked out. analysed their results will find the effectiveness of fertility treatment disappointing. Better results require lengths (18–24 and 36–48 days), or less than one, more accurate diagnosis, careful selection of cases for between one and two or more than two cycles (<18, 25–35 and >48 days). Alternatively, the figures are set out as a histogram with each day represented separately, which is satisfactory for visual presentation but difficult to describe in a report. The object of doing this is to assess the efficacy of heat detection.

Heifers

- *The assumption is made that most cows that do not become pregnant return to service at a normal cycle length of 18–24 days and therefore that returns*

where natural service is employed. Where AI is used longer than this represent a missed oestrus. While analysis is to some extent as for adult cows. The aspects this is not strictly correct in all cases the proportion shown in Box 41.7 should be borne in mind.

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Advice for veterinarians who

Box 41.7

Analysis of heifer breeding records.

wish to start herd fertility control

Starting population

Percentage cycling when put to bull

programmes

First service pregnancy rate

Percentage that become pregnant (in defined breeding

Problems on the dairy farm that limit herd productivity and profitability such as suboptimal fertility usually

Length of calving period

have multiple causes and require an integrated, multi-

Range of ages at first calving (available from milk records)

disciplinary approach (de Kruif, 1998). The veterinary practitioner will be most effective when he or she is part of a dairy farm consultant team that includes, in addition to the veterinarian, personnel such as the nutritionist, financial consultant and other agricultural professionals who can contribute to a team approach is to correlate the fertility results with factors that could interfere with reproductive efficiency.

for problem solving. If all the key advisors meet regularly on the farm and collaborate with developing solutions for farm problems, benefits will outweigh those seen if each advisor acts independently. This considered appropriate.

team approach has been advocated for many years (O'Connor et al., 1985; Kelly & Whitaker, 1999), but has

Maintaining motivation

been slow to be recognized by some and barriers are

sometime found between the different professionals.

It is essential if a breeding programme is to be success-

*The veterinary surgeon should work to break down
ful for all interested parties to remain focused and moti-
these barriers for the benefit of his client.*

vated at all times. This requires good communication

*Many veterinarians who are not carrying out fertility
between all involved and regular review of the herd's
control work for any of their clients find it difficult to
fertility performance within an economic framework.*

get started. One problem is lack of confidence as

*There are considerable benefits to be gained from
regards skills in rectal palpation or ultrasound exami-
improvements in herd health; for example, a farmer
nation. If this is the case it has to be overcome by*

*with 77 cows, who adopted a planned approach to herd
self-education and by undertaking suitable continuing
management, saw his farm profits improve by £13 000
professional development (CPD) training.*

in 2 years. It was estimated that £6000 of this came from

Marketing of the service is best done via practice

improved fertility management alone (Esslemont & newsletters and a special client meeting to explain to Kossaibati, 2000b). This in itself should be enough to clients all that is involved in control programmes, motivate farmers to undertake schemes of this type. preferably with the help of a client who is convinced of

The economic appraisal of herd health schemes has the benefits talking to others who are more sceptical. been extensively reviewed elsewhere and will not be

Help to arrange a meeting can be obtained from many discussed in depth here (Esslemont & Peeler, 1993; sources, such as other (specialist) practitioners who run Esslemont, 1995). However, it is important to realize fertility control schemes, commercial firms or col- that the financial benefits are often hidden and not leagues in the veterinary schools.

immediately apparent to the farmer unless the herd

Beginners often have difficulty in knowing what to do records are analysed regularly and benefits quantified on regular farm visits and particularly how to set up and in monetary units.

use a recording system.

Given the economic pressure on the dairy industry, farmers cannot afford to pay for services that do not bring financial benefits, nor can they afford to tolerate

Reproductive record keeping

less than optimum fertility within their herds. In the and analysis

past the uptake of herd fertility programmes has been poor in the UK (Wassell & Esslemont, 1992). If they are

The first decision to be made on reproductive record to become a regular feature on the majority of dairy keeping and analysis is who is to undertake this. Is it to be farms then it is the responsibility of the veterinarian to the farmer, the veterinarian (or his staff) or a third party? ensure that they are cost-effective and to be able to

There are distinct advantages if the veterinarian offers illustrate this fact to his client. The use of spreadsheet a complete package to the farmer including record programs containing herd specific economic parame- keeping. This gives the veterinarian control over which ters and easily interpreted graphics would make this

cows are presented at the regular farm visits. However, if task much easier.

the farmer already keeps adequate records this may result

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in duplication of effort and the farmer may be unwilling

rate records for more than one or two months prior to

to pay for something he considers he already has. What-

starting (except those data which are required to be

ever system is agreed upon the farmer will be required to

recorded by law) so that recording should be started at

collect most of the data and will need some records on

the same time as the rest of the programme. The best

farm to make day-to-day management decisions.

recording is likely in herds where there is some other

It is essential that accurate herd records be maintained

recording system used, such as individual cow milk pro-

and that they are analysed and acted upon regularly

duction records. Data kept in this way will require

(Varner & Brand, 1996; Bailey et al., 1999). A computer-regular analysis which, if done by hand, will be time

based system of record keeping offers many advantages,

consuming and thus costly.

including rapid data analysis and the rapid production of

Many farmers find that a circular breeding board is

action lists and high quality reports. Many dedicated

useful to supplement this sheet (Anon, 1984; Fig. 4.29).

herd fertility record software packages such as DAISY

Figure 41.6 could also be used as a template for the con-

(Agrisoft, NMR), InterHerd (Agrisoft, NMR), Dairy-

struction of a computer spreadsheet for data analysis.

WIN (Massey University) and DairyCHAMP (Univer-

sity of Minnesota) are now commercially available.

If the client is aiming for a 12-month

These offer distinct functions, which have been pro-

calving interval

duced specifically to handle herd fertility data. In

addition, software packages such as Herd Browser

Depending on the calving pattern and herd size,

(Livestock Services UK Ltd) and Herd Management

arrange to visit the herd say every 14 days and at each

(Sum-it Computer Systems) offer some fertility record-

visit examine all cows that:

ing and analysis functionality within more general soft-

- *Are more than 56 days after calving and have not
ware packages that have wider usage to the farmer.
been served;*

While computers are invaluable in data handling and

- *Were served 42 days or more previously and have
analysis it is important to realize that specific software
not returned to service (if unsure of early preg-
is not essential. It is perfectly possible with limited com-
nancy diagnosis, start later);*

puter skills to maintain and analyse herd reproductive

- *Have had any problems.*

data in a spreadsheet package such as MS Excel,

although it will not easily generate action lists and some

Keep a record of everything you do on the farm and

of the other output available from dedicated herd fer-

find out what has happened to all the cows you saw at

tility software. The interpretation and understanding of

the last visit (see Fig. 41.5).

herd records can be greatly enhanced by the use of

It takes a year before the records are complete

computer generated graphs and charts, such as those enough for sensible analysis. Make an initial analysis quickly and easily produced by computer programs like after six months, bearing in mind the problems discussed above (see p. 672 onwards). After that, analyse MS Excel (Hendry, 1999).

Computerized recording will allow more in-depth the records every six months, or other suitable interval, and frequent analysis of data. However, it is vital that to show the following as a minimum:

the veterinarian (and the farmer) understands fully the
(1)

The number of cows that calved.

output of these systems and recognizes that the output
(2)

Calving to service interval:

is only as good as the quality of those data being
(a)

The number that have been served since
entered into the system.

calving;

If for any reason the veterinarian is unwilling or

(b)

The number not served;

unable to use a computer-based system a written record

(c)

The mean calving-to-service interval;

system is perfectly adequate for small herds, although

(d)

A simple, small histogram of distribution of

it lacks the ability to rapidly produce regular record

calving-to-service intervals.

analysis, action lists and reports containing graphical

(3)

The same information for calving-to-conception

representation of data. The following simple, paper-

interval.

based system, which can be made up in the practice and

(4)

First service conception rate (and all service con-

photocopied, is suggested as a starting point. One sheet

ception rate).

of A4 paper is marked across the top with headings that
(5)

Culling rate (and reason for culling).

cover one reproductive cycle for each cow from calving
to confirmation of pregnancy or disposal/death. The

If the client is aiming for a tight

sheet is lined horizontally and each sheet will take
calving season

records from about 20 cows (see Fig. 41.6).

This sheet is filled in by the farmer in chronological

Use the same record system. Discuss oestrous detec-
order of calving. It is usually impossible to obtain accu-
tion. Make sure the farmer records all heats in the

674

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Chapter 41

Herd breeding record:

Farm:

Sheet no.:

Cow

Calving

Normal

Lactation

Pre-service heat

Target

Service dates/bull used

PD

Days from calving to:

date

no.

dates

1st

2nd

1st

2nd

3rd

4th

5th

1st heat

1st serve

Pregnancy

36

6-3-2001

Y

6

8-5-2001

1-5-2001

25-5-2001

P+

63

80

80

GS

20

7-3-2001

Dead calf

3

2-5-2001

12-5-2001

11-7-2001

P+

—

66

126

DH

Char

38

9-3-2001

Y

6

16-4-2001

4-5-2001

3-5-2001

P+

38

55

55

DH

186

10-3-2001

Y

4

5-5-2001

1-7-2001

¥

To be sold

P-

—

114

—

DH

148

8-7-2001

CS

1

2-9-2001

13-9-2001 —

67

DH

148

11-8-2001

Y

I

6-10-2001

219

12-8-2001

Y

2

15-9-2001

7-10-2001

34

Fig. 41.6

A simple herd fertility recording system. Y, yes; CS, Caesarean section.

Herd Fertility Management • 675

three-week period before the first day of the service

Caldow, G.L. & Taylor, D.W. (1997) Experiences with venereal period.

real Campylobacter infection in suckler herds. Cattle Prac-Examine, and treat where needed, all animals that

tice, 5, 327–34.

have not been in season at the end of the three-week

Drew, S.B., Gould, C.M., Dawson, P.L.L. & Altman, J.F.B.

preservice period. Visit at weekly or fortnightly inter-

(1982) Effect of progesterone treatment on the calving-to-

conception interval of Friesian dairy cows. Veterinary

vals thereafter to examine all cows not yet seen in

Record, **111**, 103–6.

oestrus and to follow up treatments. Monitor the

Dunn, T.D., Ingalls, J.E., Zimmerman, D.R. & Wiltbank, J.N.

submission rate.

(1969) Reproductive performance of two year old Hereford

Carry out pregnancy diagnosis nine weeks after the

and Angus heifers as influenced by pre- and post-calving

first day of the service season. Remember this simpli-

energy intake. Journal of Animal Science, **29**, 719–26.

fied advice covers the first steps to help beginners to get

Hill, J.R., Lammond, D.R., Hendricks, D.M., Dickey, J.F. & started.

Niswender, G.D. (1970) The effects of undernutrition on

ovarian function and fertility in beef heifers. Biology of Reproduction, **2**, 78–84.

Future developments in herd

Kilkenny, J.B. (1978) Reproductive performance of beef cows.

fertility management

World Review of Animal Production, **14**, 65–74.

Lowman, B.G. (1988) Suckler Cow Management. In Practice, **10**, 91–100.

The decreasing availability of staff on farms in the UK

Lowman, B.G., Scott, N.A. & Somerville, S.H. (1976) Condi-

is presenting a real challenge to many farmers and vet-

tion scoring of cattle. East of Scotland College of Agricultural Sciences as they attempt to maintain and improve

ture, Bulletin No. 6.

herd fertility. Over recent years there have been numer-

Mann, G.E. (2001) Pregnancy rates during experimentation in

ous technological developments that can help, particu-

dairy cows. *The Veterinary Journal*, **161**, 301–5.

larly aids to oestrous detection such as the KaMaR heat

Martinez, M.F., Kastelic, J.P., Adams, G.P., Janzen, E., McCartmount detector, pedometers and milk progesterone

ney, D.H. & Mapletoft, R.J. (2000) Estrus synchronization

assays (Peters & Ball, 1995). It is probable that in the

and pregnancy rates in beef cattle given CIDR-B,

prostaglandin and estradiol or GnRH. *Canadian Veterinary*

future accurate and reliable automated systems to

Journal, **41**, 786–90.

assess the cow's physiological state using biosensors will

Mawhinney, S., Roche, J.F. & Gosling, J.P. (1979) The effects be developed (Koelsch et al., 1994; Frost et al., 1997; of oestradiol benzoate (OB) and gonadotrophin releasing

Mottram, 1997; Scully et al., 2000). New developments hormone (GnRH) on reproductive activity in beef cows at

in this area of technology may allow automatic milk

*different intervals post partum. Annales de Biologie
progesterone assays, and other assays, to be performed
Animale Biochimic et Biophysique, 19, 1575–87.*

*while cows are being milked, allowing the timing of
Meat and Livestock Commission (1986) Beef Year Book, p. 55.
ovulation to be accurately predicted and removing the
MLC, Milton Keynes.*

*need for oestrous detection. In addition, technologies
Meat and Livestock Commission (2000) Beef Year Book pp.
such as automated milking may become more wide-
23–4, 31, 44–47. MLC, Milton Keynes.*

*spread, freeing stockmen from routine mundane tasks
Mulvaney, P. (1978) Dairy cow condition scoring. Paper
No. 4468. National Institute for Research in Dairying,
and allowing them to concentrate on improving cow
Reading.*

*care. New methods of data recording, such as the use of
Mulvehill, P. & Sreenan, J.M. (1977) Improvement of fertility palm held
computers and mobile phones with internet
in post-partum beef cows by treatment with PMSG and
capability, will allow data collected on the farm to be*

progestagen. *Journal of Reproduction and Fertility*, **50**, logged direct to remote computer databases in the farm

323–5.

office or at distant sites.

Penny, C.D. (1998) Practical oestrus synchronisation techniques in beef suckler herds. *Cattle Practice*, **6**, 169–73.

Penny, C.D. & Lowman B.G. (2002) Mating beef cows without

References

natural service – a triple synchronisation system. *Cattle Practice*, **10**, 23–5.

Beef herds

Penny, C.D., Lowman, B.G. & Scott, P.R. (1997) Repeated oestrus synchrony and fixed-time artificial insemination in

Allen, D. & Kilkenny, B. (1980) Planned Beef Production, beef cows. *Veterinary Record*, **140**, 496–8.

p. 183 , Granada, London.

Peters, A.R. (1982) Calving intervals of beef cows treated with Ball, P.J.H. (1982) Milk progesterone profiles in relation to

either gonadotrophin releasing hormone or a progesterone

dairy herd fertility. *British Veterinary Journal*, **138**, 546–51.

releasing intravaginal device. *Veterinary Record*, **110**, Bulman, D.C. & Lamming, G.E. (1978) Milk progesterone

515–17.

levels in relation to conception, repeat breeding and factors

Peters, A.R. (1986) Hormonal control of the bovine oestrous

influencing acyclicity in dairy cows. Journal of Reproduction cycle. II. Pharmacological principles. British Veterinary

and Fertility, 54, 447–58.

Journal, 142, 20–9.

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Peters, A.R. & Riley, G.M. (1982a) Milk progesterone profiles R.S. Morris), pp. 41–58. Post Graduate Foundation in Vet-and factors affecting post partum ovarian activities in beef

erinary Science, University of Sydney.

cows. Animal Production, 34, 145–53.

Esslemont, R.J. (1995) Economic appraisal of herd health

Peters, A.R. & Riley, G.M. (1982b) Is the cow a seasonal

schemes. The Veterinary Annual, 35, 243–80.

breeder? British Veterinary Journal, 138, 533–7.

Esslemont, R.J., Bailie, J.H. & Cooper, M.J. (1985) Fertility Roche, J.F., Ireland, J. & Mawhinney, S. (1981) Control and Management in Dairy Cattle, pp. 1–143. Collins, London.

induction of ovulation in cattle. Journal of Reproduction

Esslemont, R.J. & Kossaibati, M.A. (2000a) Trends in fertility and Fertility, Supplement 30, 211–22.

in 52 dairy herds over 11 seasons. In Proceedings of the XXI Smith, R.D., Pomerantz, A.J., Beal, W.E., McCann, J.P.,

World Buiatrics Congress, Uruguay, December, 2000, pp.

Pilbeam, T.E. & Hansel, W. (1984) Insemination of Holstein 5221–32.

heifers at a preset time after oestrous cycle synchronisation

Esslemont, R.J. & Kossaibati, M.A. (2000b) Dairy farming

using progesterone and prostaglandin. Journal of Animal

systems: husbandry, economics and recording. In The

Science, 58, 792–800.

Health of Dairy Cattle (ed. by A.H. Andrews), pp. 299–327.

Stevenson, J.S., Thompson, K.E., Forbes, W.L., Lamb, G.C.,

Blackwell Science, Oxford.

Grieger, D.M. & Corah, L.R. (2000) Synchronizing oestrus

*Esslemont, R.J. & Peeler, E.J. (1993) The scope for raising and (or) ovulation in
beef cows after combinations of*

margins in dairy herds by improving fertility and health.

GnRH, Norgestomet, and prostaglandin F2a with or without

British Veterinary Journal, 149, 537–47.

timed insemination. Journal of Animal Science, 78, 1747–58.

*Frost, A.R., Schofield, C.P., Beulah, S.A., Mottram, T.T., Lines, Thibault, C.,
Courot, M., Martinet, L., Mauleon, P., De Mesnil J.A. & Wathes, C.M. (1997) A
review of livestock monitor-du Buisson, F., Ortovant, R., Pelletier, J. & Signoret,
J.P.*

ing and the need for integrated systems. Computers and

(1966) Regulation of breeding season and oestrous cycles

Electronics in Agriculture, **17**, 139–59.

by light and external stimuli in some mammals. *Journal of*

Hendry, M.A.W. (1999) Practitioner use and graphical display

Animal Science, **25** (Suppl.), 119–39.

of dairy cow fertility records to give the farmer/herdsman

Webb, R., Lamming, G.E., Haynes, N.B., Hafs, H.D. & Manns, incentives to improve efficiency of output. *Reproduction in J.G. (1977) Response of cyclic and post-partum suckled*

Domestic Animals, **34**, 203–12.

cows to injections of synthetic LH-RH. *Journal of Repro-*

Howells, H.M.J., Davies, D.A.R. & Dobson, H. (1999)

duction and Fertility, **50**, 203–10.

Influence of the number of days spent training in an

White, I.R., Russel, A.J.F., Wright, I.A. & Whyte, T.K. (1985) abattoir with access to live cows on the efficiency of do-itReal time ultrasonic scanning in the diagnosis of pregnancy

yourself artificial insemination. *Veterinary Record*, **144**, and the estimation of gestational age in cattle. *Veterinary* 310–14.

Record, **117**, 5–8.

Kelly, J.M. & Whitaker, D.A. (1999) A multidisciplinary

approach to dairy herd health and productivity manage-

ment. *BSAS Occasional Meeting: Fertility in the High-*

Dairy herds

Producing Dairy Cow, Galway, Ireland, September, 1999, pp.

25–6.

Anon (1984) Dairy Herd Fertility. Ministry of Agriculture, Knight, C.H. & Sorensen, A. (1998) Fertility parameters of Fisheries and Food, Reference Book 259, pp. 1–80. Her

cows with extended lactations. Cattle Practice, 6, 379–82.

Majesty's Stationery Office, London.

Koelsch, R.K., Aneshansley, D.J. & Butler, W.R. (1994) Milk Anon (1999) BCVA Herd Health Plan for the Purposes

progesterone sensor for application with dairy cattle.

of Farm Assurance, pp. 1–21. BCVA Office, The Green,

Journal of Agricultural Engineering Research, 58, 115–20.

Frampton-on-Severn, Gloucester, UK.

Kruif de, A. (1998) Modern health care in food producing

Bailey, T.L., Dascanio, J. & Murphy, J. (1999) Analyzing

animals. Irish Veterinary Journal, 51, 588–90.

reproductive records to improve dairy herd production.

Lamming, G.E., Darwash, A.O. & Woolliams, J.A. (1997) Sub-

Veterinary Medicine, 94, 269–76.

fertility in dairy cattle: potential genetic and environmental Barrett, D.C. (2001) Cattle Fertility Management in the UK.

influences. Cattle Practice, 5, 357–60.

Cattle Practice, 9, 59–68.

Lucy, M.C. & Crooker, B.A. (1999) Physiological and genetic Brand, A. & Varner, M. (1996a) Monitoring reproductive per-differences between low and high index dairy cows. BSAS

formance: objectives, reproductive indices and materials

Occasional Meeting: Fertility in the High-Producing Dairy

and methods. In Herd Health and Production Management

Cow, Galway, Ireland, September, 1999, pp. 27–8.

in Dairy Practice (ed. by A. Brand, J.P.T.M. Noordhuizen & Mottram, T. (1997) Automatic monitoring of the health and

Y.H. Schukken), pp. 283–92. Wageningen Pers, Wageningen.

metabolic status of dairy cows. Livestock Production

Brand, A. & Varner, M. (1996b) Monitoring reproductive

Science, **48**, 209–17.

performance: execution. In Herd Health and Production

Nebel, R.L. & McGilliard, M.L. (1993) Interactions of high Management in Dairy Practice (ed. by A. Brand, J.P.T.M.

milk yield and reproductive performance in dairy cows .

Noordhuizen & Y.H. Schukken), pp. 293–311. Wageningen

Journal of Dairy Science, **76**, 3257–68.

Pers, Wageningen.

O'Connor, M.L., Baldwin, R.S., Adams, R.S. & Hutchinson,

Dijkhuizen, A.A., Huirne, R.B.M., Jalvingh, A.W.

&

L.J. (1985) *An integrated approach to improving reproduc-*

Stelwagen, J. (1997) Economic impact of common health

tive performance. Journal of Dairy Science, 68, 2806–16.

and fertility problems. In Animal Health Economics –

O’Farrell, K.J. & Crilly, J. (1999) Trends in calving rates in Irish Principles and Applications (ed. by A.A Dijkhuizen & dairy herds 1991–1996. Cattle Practice, 7, 287–90.

Herd Fertility Management • 677

Peeler, E.J., Otte, M.J. & Esslemont, R.J. (1994a) Recurrence and Production Management in Dairy Practice (ed. by A.

odds ratios for periparturient disease and reproductive

Brand, J.P.T.M. Noordhuizen & Y.H. Schukken), pp. 313–32.

traits of dairy cows. British Veterinary Journal, 150, 481–8.

Wageningen Pers, Wageningen.

Peeler, E.J., Otte, M.J. & Esslemont, R.J. (1994b) Inter-

Wassell, T.R. & Esslemont, R.J. (1992) Survey of the

relationships of periparturient diseases in dairy cows.

operation of dairy herd health schemes by veterinary

Veterinary Record, 134, 129–32.

practices in the United Kingdom. Veterinary Record, 130, Peters, A.R. & Ball, P.J.H. (1995) Reproduction in Cattle, 2nd 260–3.

edn, pp. 47–61. Blackwell Science, Oxford.

Wathes, D.C., Robinson, R.S., Pushpakumara, A., Cheng, Z.

Royal, M.D., Darwash, A.O., Flint, A.P.F., Webb, R., Woolliams, & Abayasekara, D.R.E. (1998) Nutritional effects on J.A. & Lamming, G.E. (2000) Declining fertility in dairy reproductive performance in dairy cows. *Cattle Practice*, **6**, cattle: changes in traditional and endocrine parameters of 371–7.

fertility . *Animal Science*, **70**, 487–501.

Webb, R., Garnsworthy, P.C., Gong, J.G., Gutierrez, C.G., Scully, A., Hunter, M., Gregson, K., Lamming, G.E. & Logue, D., Crawshaw, W.M. & Robinson, J.J. (1997) Nutritional influences on subfertility in cattle. *Cattle Practice*, **5**, matic interpretation of milk progesterone data: an aid to 361–7.

dairy fertility management. *Cattle Practice*, **8**, 407–10.

Webb, R., Royal, M.D., Gong, J.G. & Garnsworthy, P.C. (1999) Varner, M. & Brand, A. (1996) Monitoring reproductive performance: decision making and follow-up. In *Herd Health* 227–34.

Chapter 42

Pharmacological Manipulation of Reproduction

J.G. Allcock and A.R. Peters

Introduction

678

(3)

By the simulation of corpus luteum function, by

Control of the oestrous cycle

678

administration of progesterone (or one of its syn-

Induction of luteolysis

678

thetic derivatives) for a number of days, followed

Trophic hormone and prostaglandin combinations

680

by abrupt withdrawal.

Use of progestagens

680

Factors affecting pregnancy rate after controlled

The three methods are discussed in greater detail below.

ovulation

682

Synchrony after prostaglandins

682

Synchrony after trophic hormone–prostaglandin

Induction of luteolysis

combination techniques

683

The most potent luteolytic agents available are deriva-

Synchrony after progestagens

683

tives of prostaglandin F_{2a} (PGF_{2a}). Injection of exoge-

Possible methods of overcoming problems of asynchrony 683

nous PGF

Field evaluation of oestrus synchronization techniques

684

2a or one of its analogues during the mid

luteal phase of the cycle results in premature luteolysis

Establishment of pregnancy

684

Pharmacological induction of parturition

686

and a consequential fall in peripheral progesterone

Methods of induction of parturition

687

concentrations. This is followed by a rise in the secre-

Delay of parturition

687

tion of pituitary gonadotrophins and eventual ovula-

tion. The fall in progesterone concentration is rapid,

Introduction

often reaching basal levels within 30 hours of injection.

There are now several commercial analogues of PGF₂a.

Examples of those currently marketed in the UK and

This chapter aims to cover the manipulation of repro-

elsewhere are shown in Table 42.1.

duction in cattle where this is not covered in the chap-

Prostaglandins have been used to control the

ters on general fertility. The discussion includes control

oestrous cycle in several different ways. Some possible

of the oestrous cycle, the establishment and mainte-

methods are given below:

nance of pregnancy and induction and postponement of

parturition.

(1)

Following rectal examination, only those cows with a corpus luteum are injected. These cows should

Control of the oestrous cycle

then show oestrus and ovulate three to five days later.

This method has the disadvantage that it is time-

The control of the oestrous cycle is dependent on consuming and that rectal palpation involves added manipulation of the hormonal events occurring during expense. The results also depend on the accuracy of the normal ovarian/oestrous cycle. The over-riding event detection of corpora lutea by rectal palpation.

controlling the development of an ovarian follicle

(2)

All cattle are observed for oestrus over a seven- to the point of ovulation in the cyclic cow is believed to day period, serving any that show oestrus. The rest are be the process of luteolysis or decrease in progesterone injected with prostaglandin on the following day and secretion occurring between days 17 and 18 of the may be inseminated either once or twice at fixed normal cycle (see Fig. 42.1). This fall in peripheral

times or at observed oestrus. The reason for the initial progesterone concentrations may be manipulated artificially in three ways:

of about seven days in the cycle (day 18 to day 0 and
(1)

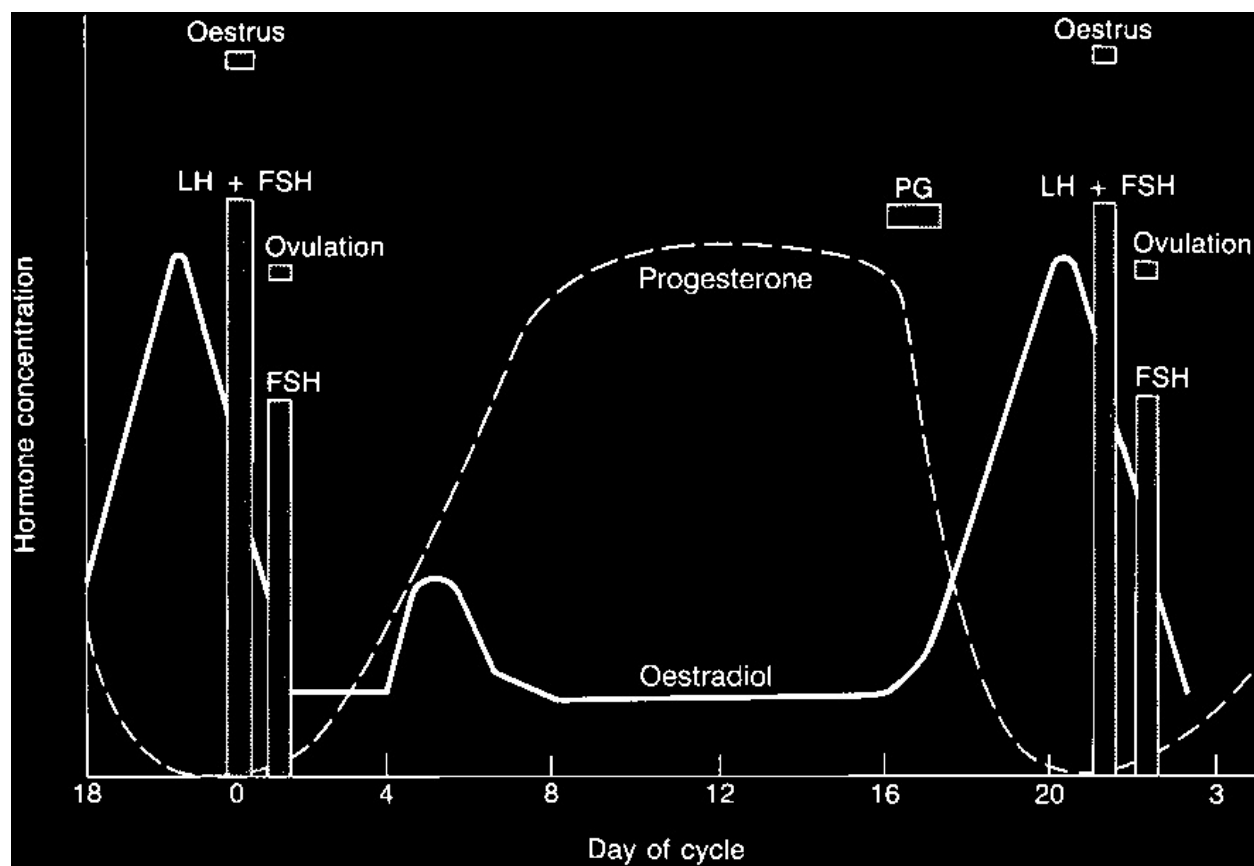
By the artificial induction of premature luteolysis day 1 to day 4) when the animal is unresponsive to using luteolytic agents, e.g. the prostaglandins.

prostaglandin, i.e. when no corpus luteum is present.
(2)

By attempting to modify the timing of normal

After seven days, those originally between days 18 and ovarian events through a combination of trophic 0 will have shown heat and been served, while those hormones and luteolytic agents.

that were between days 1 and 4 will now be between



Pharmacological Manipulation of Reproduction • 679

Fig. 42.1

Changes in blood plasma

concentrations during the bovine

oestrous cycle (schematic): — oestradiol;

--- progesterone.

Table 42.1

Examples of prostaglandin analogues available in the UK and elsewhere (2003).

Trade name

Active

Distributor

Route of

Dose rate

Insemination timing

component

injection

(mg)

recommendations (hours)

Double

Single

Estrumate

Cloprostenol

Schering-Plough

im

0.5

72, 96

72–84

Prosolvin

Luprostiol

Intervet

im

15.0

72, 96

72

Lutalyse

Dinoprost

Pharmacia &

im

25.0

72, 90

78

Upjohn

Enzaprost

Dinoprost

CEVA

im

25.0

72, 96

No recommendation

Noroprost

Dinoprost

Norbrook

im

25.0

72, 96

No recommendation

Prostavet

Etiproston

Bimeda

im

5.0

72, 96

No recommendation

tromethamine

*days 8 and 11, i.e. in the mid luteal phase, and therefore
sis in response to the injection and will ovulate some
responsive to prostaglandin.*

*four days or so later. At the time of the second injection
(10 or 11 days later) these cows will be on about*

(3)

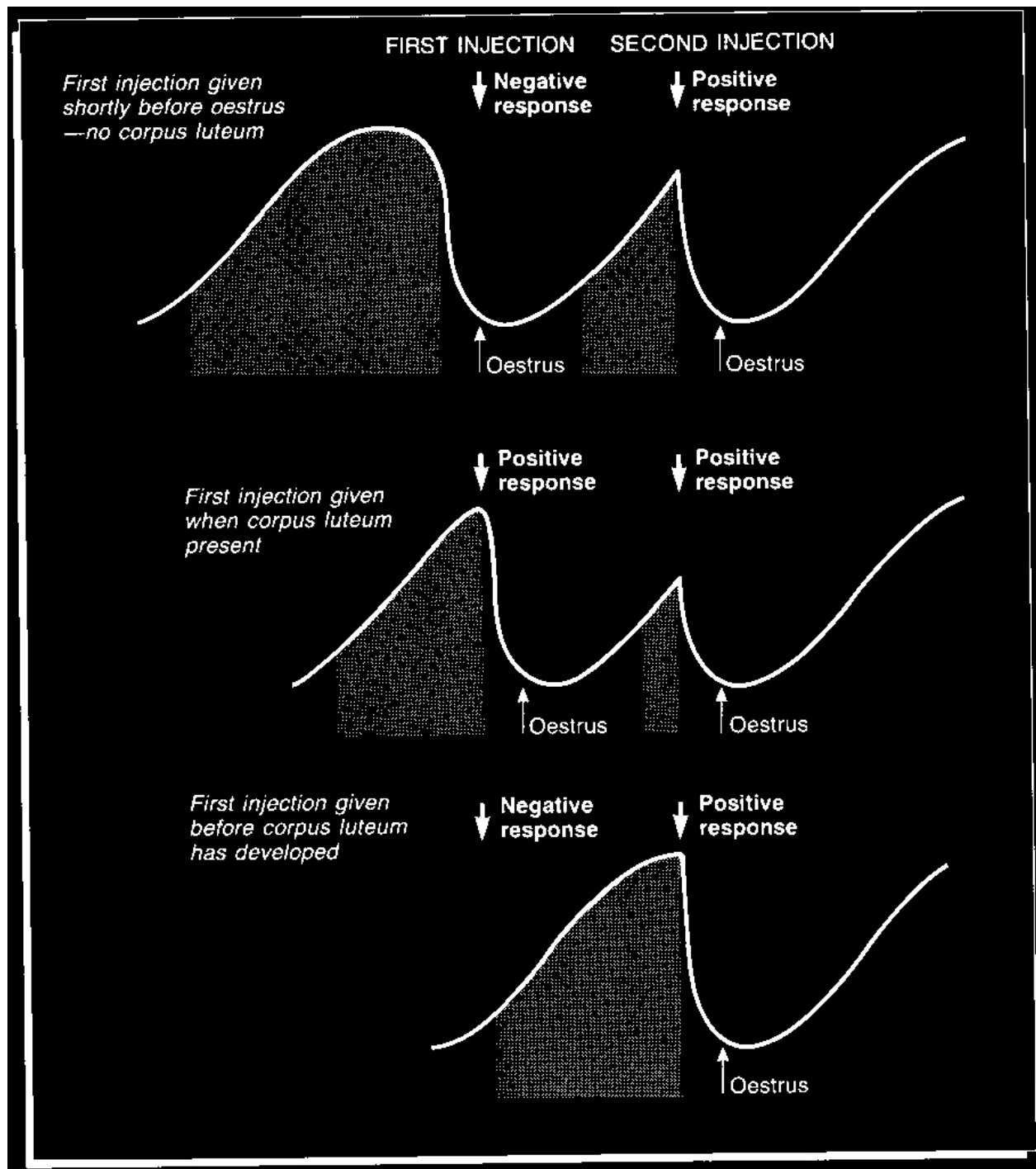
The two injections plus two inseminations

*days 8 and 15 of the next cycle (Fig. 42.2). The cows that
method. The so-called 'two plus two' technique was*

were not responsive to the first injection, i.e. those designed to synchronize groups of animals cycling at between days 18 and 4 of the cycle, would now be random without prior knowledge of their precise between days 8 and 15 at the time of the second injection. All cattle are injected on day 1 of treatment and the injection is repeated 10 or 11 days responsive mid luteal phase at the time of the second later. Artificial insemination (AI) is then carried out injection. The technique is popular and quite successful usually three and four days later (72 and 96 hours after in synchronizing cycles in heifers (Cooper, 1974) and the second prostaglandin injection).

has resulted in pregnancy rates equivalent to control The principle of this regimen is illustrated in animals. However, pregnancy rates achieved with this Fig. 42.2. At the time of the first injection some animals technique in adult cows have not always been will be responsive to the prostaglandin, i.e. between consistent and some of the reasons for this are discussed

days 5 and 17 of the cycle. These will undergo luteolysis later.



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Fig. 42.2

The effect on progesterone

concentrations of giving two injections of prostaglandin 11 days apart. (MAFF, 1984).

(4)

A further method of using prostaglandins that a dominant follicle being present at the time of induction has been quite popular is the so-called '11/2 method'. Cows are injected with prostaglandin and those that show oestrus are inseminated. Those that have not been 'priming' of the oestrous cycle have been tested in the UK (Mawhinney et al., 1996). In most cases a second injection and may be inseminated either at a fixed administration of GnRH has been used in an attempt time(s) or at observed oestrus. Although requiring further effort in terms of oestrous detection, this method tends to give better results than the 'two plus

hours after prostaglandin administration, with insemination being carried out 18 to 24 hours after the post main advantage, however, is the reduction in cost by the luteolytic GnRH (Roche, 1999).

reduction of both the number of treatments used and the number of inseminations per cow.

Use of progestagens

The third method of controlling the cycle is to simulate the function of the corpus luteum by the administration

Trophic hormone and

of progesterone or one of its derivatives. In this method,

prostaglandin combinations

gonadotrophin release, and hence ovarian follicular

Gonadotrophin releasing hormone (GnRH) or its

maturation, is suppressed until progesterone with-

analogues can be used to reprogram the developing

drawal. If progesterone is used to treat a group of cows

waves of ovarian follicles to maximize the possibility of

and then it is withdrawn from all cows simultaneously,



Pharmacological Manipulation of Reproduction • 681

this will theoretically synchronize ovulation in the whole group.

In order to synchronize a group of randomly cycling cows effectively, it is necessary to treat them with progesterone for a period at least equivalent to the length of the natural luteal phase, i.e. 16 days. This is because exogenous progesterone has little or no effect on the life span of the natural corpus luteum and therefore, in a small proportion of cases, the natural corpus luteum might outlive a short-term progesterone treatment, resulting in a failure of synchrony. However, it has been shown that longer-term progesterone treatments (18–21 days) result in poor pregnancy rates. It is

thought that this is due to adverse changes in the intra-uterine environment that may inhibit sperm transport.

Shorter-term progesterone treatments (7 to 12 days)

generally result in more acceptable pregnancy rates,

Unfortunately, short-term progesterone treatment does

Fig. 42.3

A progesterone-releasing intravaginal device (PRID).

not control the cycle adequately since, if treatment is

started early in the cycle, the natural corpus luteum

may outlast the progesterone treatment. Therefore, it is

often necessary to incorporate a luteolytic agent with

short-term progesterone treatments in order to elimi-

covered by a layer of grey inert silastic (Fig. 42.3) in

nate any existing corpus luteum.

which 1.55 mg progesterone is impregnated. A gelatin

Progestagens (progesterone-like compounds) can be

capsule containing 10 mg oestradiol benzoate is

administered in the feed, by injection or by implant.

attached to the inner surface of the coil. The oestradiol

Administration in feed requires that the compound

benzoate in the PRID is intended to act as a luteolytic

is 'orally active', i.e. it is absorbed into the systemic agent. The CIDR is a Y-shaped device consisting of a circulation unchanged. Progesterone itself is relatively inactive orally and thus synthetic analogues, for example medroxyprogesterone acetate (MPA) and melengestrol acetate (MGA), were developed for this purpose. However, this route of administration presents problems of controlled dosing and the possibility of tissue or milk residues, particularly in dairy cows. The progesterone is continuously released from the elastomer until removal of the device. Removal is effected by pulling on the string (PRID) or plastic 'tail'. Therefore, such techniques are not favoured in the UK (CIDR) which is left protruding from the vulva after insertion. Removal after 7 to 12 days causes peripheral oestrous control in cattle; however, they are used widely in the USA and other countries, particularly in

plasma progesterone concentrations to fall, thus simulating natural luteolysis. Consequently, the cow should

Progestagens can be given by injection, but as the show oestrus 48–72 hours later and fixed-time AI may half-life of progesterone and its analogues is short, then be used.

repeated treatments may be necessary. The rate of There has been some interest in the use of low doses absorption is often imprecise, so synchronized with- of oestrogens after the removal of the progesterone drawal of the compound can be unreliable. Implants delivery device. Doses of 1 mg have been used in an appear to be the most suitable method of administra- attempt to further synchronize the onset of oestrus tion of progestagens since withdrawal can then be pre- subsequent to the fall in plasma progesterone concen- cisely controlled by implant removal.

trations. Questions remain regarding the fertility consequences of this approach (Roche, 1999).

Intravaginal progestagen-containing device

(e.g. PRID or CIDR)

Norgestomet (Crestar)

The PRID (CEVA Animal Health) or CIDR (InterAg/

Norgestomet,

17 α -acetoxy-b-methyl-19-nor-preg-4-

ART) are specialized forms of implant that are inserted

ene, 20-dione (Crestar, Intervet), is marketed for

into, and held within, the cow's vagina for a period of

planned breeding of beef cows and dairy and beef

7 to 12 days. The PRID consists of a stainless steel coil

heifers. It is an example of a synthetic analogue of

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progesterone and consists of an impregnated silastic

Synchrony after prostaglandins

subcutaneous implant. The implant is inserted sub-

Three major circumstances in which asynchrony can

cutaneously behind the ear for a period of nine or ten

arise have been reported in the literature (Jackson

days, during which time the progesterone is absorbed

et al., 1979; Baishya et al., 1980; Peters et al., 1980).

into the circulation. The implant is removed by making

a small scalpel incision in the skin of the ear over the implant. At the time of the implantation an intramus-

Failure of complete luteolysis

cular injection of 5 mg oestradiol valerate is given as luteolysin, in combination with an initial injection of 3

This has occurred in 10 per cent or more of cows treated mg norgestomet.

with prostaglandins. It takes the form either of complete lack of effect on progesterone concentrations

Factors affecting pregnancy rate after

or, for example, a fall to half of pre-injection levels, fol-

controlled ovulation

lowed by luteal recovery, usually within 24–48 hours.

Causes of luteolytic failure are not clear but may be

It cannot be overemphasized that in order to maximize related to several factors including the following:

results obtained with pharmacological control of the oestrous cycle, nutritional status and general manage-

- *Non-responsiveness of some corpora lutea even in the appropriate phase of the cycle.*

ment must be of a high standard. The efficacy of the

pharmacological control of ovulation can be considered

- *Treatment too early in the luteal phase.*

as two components:

- *Incorrect injection site or technique: in the case of intramuscular injections, occasionally the material*

(1)

The degree of synchrony obtained following treatment may be injected accidentally into fat or ligamentous tissue. This may be defined as the proportion of

animals beginning to show oestrus or ovulating

- *Short half-life of the chosen exogenous prostaglandin in the animal.*

monal treatment. For the purposes of this discussion

The extent of these various problems is not known the target of synchrony is for the maximum and it must be admitted that all are to some extent number of animals to show oestrus within approximately speculative.

imately 48 hours of progesterone withdrawal or 72

hours after prostaglandin injection (or to ovulate approximately 24 hours later in each case).

Long follicular phases after injection

(2)

Reproductive performance,

which may be

expressed, for example, as the pregnancy rate

In up to 20 per cent of cows injected with prostaglandin, achieved after treatment.

although luteolysis appears to occur normally, progesterone concentrations remain low for an unusually

Obviously in some circumstances the pregnancy rate

long period. This may be associated with a delay in

may be highly dependent on the degree of synchrony,

the timing of ovulation relative to oestrus. However,

particularly if fixed-time AI is used. For example, if

extended follicular phases (longer than eight days)

there is a poor degree of synchrony there will be a wide

also occur in about 17 per cent of untreated dairy cows.

variation in the timing of ovulation between cows

Thus it is likely that this phenomenon is related to a

or, more correctly, a proportion of cows will ovulate natural aberration in the adult cow's ovarian cycle. The beyond the specific period anticipated. Therefore problem has not been reported in heifers and certainly insemination would not be accurately timed and the the cycles of adult cows would appear to be less uniform pregnancy rates following fixed-time AI may be than those of heifers. poor.

In view of the natural variation in timing of the behavioural and ovarian events around natural oestrus, Acyclic cows

it is perhaps not surprising that even after hormone treatments there is still considerable variation in the The ovary can only respond to prostaglandin if there is timing of responses between animals. Hence where a functional corpus luteum. Therefore, cows with little fixed-time AI is used, two inseminations are usually recommended in order to maximize the probability of con- The proportion of cows in this state will vary from herd

ception. In addition, other problems occur that appear to herd and the average stage after calving, but it is generally regarded as a more serious problem in suckling these are described below.

beef cows. For this reason some advisors recommend *Pharmacological Manipulation of Reproduction* • 683 that prostaglandins are not used before day 42 after without a luteolytic agent. Synchronization rates of 70 calving.

per cent have been reported in practice (e.g. Drew et al., 1979) Oestradiol is not always an effective luteolytic agent and, like prostaglandins, does not always prevent

Synchrony after trophic

formation of the corpus luteum when administered

hormone–prostaglandin

early in the cycle (Peters, 1984).

combination techniques

It has been shown that more than 90 per cent of cattle

Failure to maintain high blood concentration

treated with GnRH followed by prostaglandin and

of progesterone

a second GnRH (the so-called 'GPG' system) can be induced into a low progesterone phase at the time of It has been shown that in some circumstances proges-planned insemination. The proportion of cattle either terone concentrations in the cow may fall before with-failing to respond in this way or failing to show oestrus drawal of the progesterone source (Roche & Ireland, and ovulation as expected may be influenced by several 1981). Obviously this could result in oestrus and ovula-factors:

tion occurring before removal of the device. This premature fall has occurred particularly with the

- Some cows may fail to be in the luteal phase at intravaginal method of administration. It has been the time of prostaglandin administration. This is suggested that this is related to progesterone-induced thought to occur to a greater extent in heifer groups changes in absorption across the vaginal wall rather and may be a consequence of a difference in the than to exhaustion of progesterone in the device.*

frequency of follicular waves in immature cattle.

The GPG system is usually less effective in heifer groups.

Possible methods of overcoming

- *Failure to respond to prostaglandins as previously*

problems of asynchrony

discussed.

Failure to undergo complete luteolysis

- *Earlier than expected expression of oestrus. The normal recommendation is to serve on observed*

An alternative to the use of oestradiol as a luteolytic agent in combination with progestagens is to use a
oestrus should cattle be available for service earlier
than the time planned for insemination.

prostaglandin. Whilst problems of prostaglandin usage

- *Failure to exhibit behavioural oestrus due to slow or have been referred to above, they are clearly far more abnormal follicular development or true anoestrus*
potent luteolytic agents than oestradiol. Prostaglandin is usually injected on the day before progestagen withdrawal. Various studies have shown that such com-

Synchrony after progestagens

binations give well-synchronized oestrus and endocrine

There are two major circumstances in which synchrony responses (Beal, 1983; Peters, 1984). However, there may be incomplete following progestagen treatment.

have been few reports where fixed-time AI has been used (Smith et al., 1984). Unfortunately, such a treatment combination would obviously add further expense

Ineffectiveness of the luteolytic agent to a controlled breeding programme.

As discussed above, oestradiol is often used as a luteolytic agent, along with progestagen treatments. If, for

The use of low dose oestradiol example, a nine-day progestagen treatment is started, without a luteolytic agent, at day 9 of one cycle and

As detailed earlier, low (1 mg) doses of oestrogen have finishing before day 1 of the next, then these animals been used in the pro-oestrus period after removal of should synchronize adequately, since the end of treatment an intravaginal device. This is said to enhance the postment either coincides with, or occurs after, luteolysis (or

itive feedback that promotes the pre-ovulatory surge of the progesterone blocks ovulation). However, if treatment is started between days 2 and 8, then the corpus luteum can outlive the progestagen treatment. Hence, behavioural expression of oestrus.

it is for cows in the early stages of the cycle that the luteolysin is required. If it is assumed that the group of Prolonged follicular phases

cows is cycling at random, then one would expect about 30 per cent of them to be in this category and to respond. This phenomenon has been associated with a delay in poorly to oestrous synchronization using a progestagen oestrus and ovulation. Therefore a logical approach

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Table 42.2

Effect of cloprostenol treatment of cyclic cows that had not been observed in oestrus by day 50 after calving (from Ball, 1982).

Treatment

Time of insemination

No. of cows

Calving-to-conception

interval (days)

None until 90 days after calving

Observed oestrus

166

107.4

Cloprostenola

Observed oestrus

61

98.1

Cloprostenola

2 and 3 days after injection

75

104.0

a 0.5 mg cloprostenol (Estrumate, ICI) was injected intramuscularly 10–14 days after ovulation had occurred as judged from three times weekly milk progesterone measurements.

might be to attempt artificially to induce ovulation

formance. These will not be discussed in detail here but

at a standard time after prostaglandin treatment. A

they may be summarized as follow:

single injection of gonadotrophin-releasing hormone

- *In adult cows the calving rate of control groups*

(GnRH) administered approximately 60 hours

to single AI at observed oestrus is of the order of

after prostaglandin injection (Coulson et al., 1980) has 50 per cent. Most studies report equivalent rates

the effect of inducing a preovulatory-type LH surge

in treated cows.

normally associated with ovulation. This procedure has

- *Fertility results are often up to 20 per cent better in
been used following synchronization of follicle devel-
heifers than in cows.*

opment with GnRH as described earlier. There are also

- *In general, single fixed-time AI might be expected
many reports on using GnRH after observed oestrus to
to result in 10–15 per cent lower pregnancy rates
produce a predictable ovulation in order to enhance
than two fixed-time AIs.*

conception success. This approach is discussed later.

- *It is clear that the best reproductive performance is
achieved when oestrous cycle control is combined*

Acyclic cows

with insemination at observed oestrus. In that situation the reproductive performance of treated cows This problem may be due to ineffective management in may often be higher than that of controls. This may that either the cows are being treated too early after happen particularly in a herd where the efficiency calving or that nutritional management is, and has been, of oestrous detection is normally low. Following inadequate (see Chapter 9). Assuming these problems a synchronization treatment, it is to be expected are not present, then acyclicity may be overcome in that the vast majority of cows should show oestrus a proportion of cows by the use of progestagens as within 10 days after treatment. Therefore, the effect opposed to prostaglandins.

of treatment in this situation is to concentrate the occurrence of oestrous periods, so that detection Effect of the length of progestagen treatment on efficiency can be increased over a relatively pregnancy rate

short time. Results of studies on the use of the prostaglandin analogue cloprostenol with either The deleterious effects of long-term progestagen fixed-time AI or observed oestrus are shown in treatment on fertility have already been discussed. Table 42.2 and help illustrate the advantage of However, there is some evidence that even short-term insemination at observed oestrus. Following retro-treatments can cause reduced fertility, particularly spective analysis of data from 17 published trials where the treatment is started during the late luteal McIntosh et al. (1984) concluded that prostaglandin phase of the cycle. It is possible that this occurs because treatment combined with insemination at observed the animal has been exposed to an uninterrupted long-oestrus improved conception rates over controls by term progestagen treatment, albeit a combination of an average of 7 per cent.

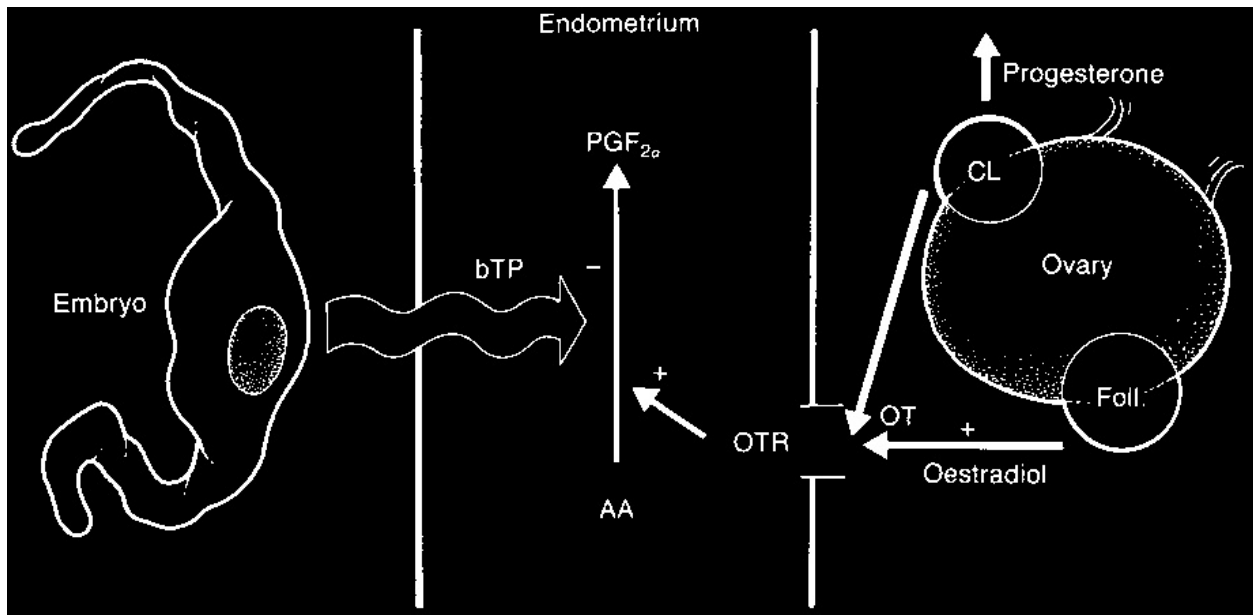
endogenous and exogenous sources.

Field evaluation of oestrous

Establishment of pregnancy

synchronization techniques

Many field trials have been carried out to assess the effect of the various treatments on reproductive performance. A low pregnancy rate to first



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Fig. 42.4

Possible endocrine relationship between the early embryo, endometrium and ovary. BTP, bovine trophoblast protein; AA, arachidonic acid; OT, oxytocin; OTR, oxytocin receptor; CL, corpus luteum; Foll,

follicle.

service is a major cause of poor reproductive perform-

length of the luteal phase in non-pregnant animals

ance. In the absence of specific infectious disease it

(Stewart et al., 1989; Thatcher et al., 1989). This work led has been shown that the major problem here, at least in

to the conclusion that some embryo mortality may be

cattle, is early embryonic death. Total embryonic and

related to a failure of some embryos to produce suffi-

fetal mortality is estimated to be around 38 per cent and

cient trophoblast protein.

it is thought that 70 to 80 per cent of that loss occurs

A number of methods have been used in the field to

between days 8 and 16 after insemination (Sreenan &

improve pregnancy rates in cattle. Diskin and Sreenan

Diskin, 1986).

(1986) reviewed the studies on comparisons of proges-

Pregnancy establishment involves active and passive

terone concentrations in pregnant and non-conceiving

communication between the embryo and the uterus.

cows before and after insemination and concluded

The exact cause(s) of embryonic death is unknown but that the data were conflicting and inconclusive in all it is circumstantially related to premature regression of respects. Progesterone supplementation during early the corpus luteum. In other words, the corpus luteum pregnancy has given equivocal results, but does seem to is normally maintained for the whole of gestation and be effective where control pregnancy rates are particularly early embryonic mortality is associated with its early larly low, i.e. 40 per cent or below. In summarizing the loss. This results in a decrease in progesterone concentrations of progesterone supplementation during early trations allowing the animal to return to oestrus, prob-pregnancy, Mann and Lamming (1999) conclude that ably at the normal time.

while progesterone concentrations during the luteal Before examining methods of reducing embryo phase are an important determinant of the outcome of mortality it might be useful to provide a brief review of pregnancy, they do not appear to be as important as the findings in relation to the establishment of pregnancy.

timing of the post ovulatory progesterone rise.

This is illustrated in Fig. 42.4. In the non-pregnant

More recently attention has been paid to the use of

animal PGF2a secreted by the endometrium causes

gonadotrophin or GnRH in enhancing early luteal

regression of the corpus luteum. There is evidence that

performance in support of the developing embryo.

oestradiol-17b from developing ovarian follicles stimu-

Peters (1996) found that 1500 iu of human chorionic

lates the synthesis of receptors for oxytocin on endome-

gonadotrophin on day 12 of the cycle resulted in a sus-

tained increase in progesterone concentration and an

(Wathes, 1984; Lamming & Mann, 1995), binds to these

increased diameter of the corpus luteum.

receptors, thereby stimulating the synthesis and secre-

It has been common veterinary practice for many

tion of PGF2a. In early pregnancy a protein of molecu-

years to inject cows at the time of service with a

lar weight of approximately 18 000 is secreted by the

‘holding’ injection using either human chorionic gona-

embryo. This protein appears to exert an anti-luteolytic effect and has been termed bovine or ovine trophoblast protein 1 (e.g. oTP-1). Stewart et al. (1989) showed that exact physiological rationale for this has not always been clear since such treatment could potentially have at least two effects. Firstly, there is a widely held belief that under some circumstances ovulation endometrium and intrauterine infusion of trophoblast proteins or recombinant interferon can extend the Administration of GnRH will result in preovulatory gonadotrophin release and subsequent ovulation.

Table 42.3

Pregnancy rate (per cent) to first insemination with
Secondly, LH is considered to be the major luteotrophic

10 mg buserelin injected between days 11 and 13 after insemination, at least during the first few days of pregnancy.
nation (from MacMillan et al., 1986).

Therefore, GnRH-induced LH release may facilitate

Trial

Treated

Control

the development and maintenance of the corpus luteum in the post ovulatory period. Numerous trials have been

A

75.4

62.2

carried out where either HCG (LH) or GnRH has

B

67.5

57.8

been given on the day of service. In a meta-analysis of

Overall

72.4a

60.9

many studies Valks (1996) illustrated that a small effect

was generally achievable (around 8 per cent) and that
a $P < 0.01$.

the magnitude of any effect was influenced by factors
including body condition score. Study results varied,
with some showing good responses and others showing
no differences from control animals. This could be for

Table 42.4

Pregnancy rates (per cent) to second insemination
one or more of the following reasons:

(from MacMillan et al., 1986).

(1)

Poor design of trials, involving a few animals in

Trial

Treated

Control

each study group. This is a common problem
where fertility rates are being studied. Due to the

A

78.6

69.0

fact that pregnancy rate is discrete variable and

B

94.7

70.8

that rates are very different between farms, very

Overall

85.1a

69.5

large numbers (several hundred per group) may

a $P < 0.05$.

be required to establish statistically significant

differences between groups.

(2)

The actual cause of pregnancy failure may differ

widely between farms and therefore one may

Thatcher et al. (1989) suggested that buserelin acts in be attempting to rectify many different primary

these situations by disrupting normal waves of ovarian

problems by the use of such a treatment. The

follicular growth and oestradiol secretion, resulting in a

primary cause is often impossible to diagnose, at

failure of the luteolytic mechanism (see Fig. 42.4).

least at that time.

Mann and Lamming (1995), however, maintain that

(3)

Thus it is probably a fair summary to state that

GnRH causes a short-lived reduction in oestradiol

the best results for the improvement of pregnancy

secretion which reduces the stimulus to the develop-

rates have been achieved where the control or

ment of the luteolytic mechanism.

background fertility of the herd is poor, although

there are some exceptions to this.

MacMillan et al. (1986), attempted to support the

corpus luteum when it became susceptible to the lute-

Pharmacological induction

olytic mechanism, i.e. approaching day 16 after oestrus in

of parturition

the cow. Treated cows (approximately 225) were given a

single injection of 10 mg buserelin (a synthetic analogue

Many attempts have been made to induce parturition

of GnRH) on day 11, 12 or 13 after AI. Treated and

artificially in the final days of gestation. These methods

control cows were palpated at six to nine weeks to determine pregnancy status, cows returning to service were re-anisms involved in the normal parturition process.

inseminated. Pregnancy rates at six to nine weeks were

These have included the use of corticosteroids, oestro-

72.5 per cent and 60.9 per cent for the treated and control

gens and prostaglandins since these are all involved in

cows respectively (see Table 42.3). Of those cows return-

the endocrine pathway (see Fig. 42.5).

ing to service the pregnancy rates to second service were

These are several indications for the induction of

85.1 per cent and 69.5 per cent for treated and control

parturition in cows. Firstly, in countries such as New

cows, respectively (see Table 42.4).

Zealand, where a tight seasonal calving pattern is often

In the UK a similar approach was adopted, with 1619

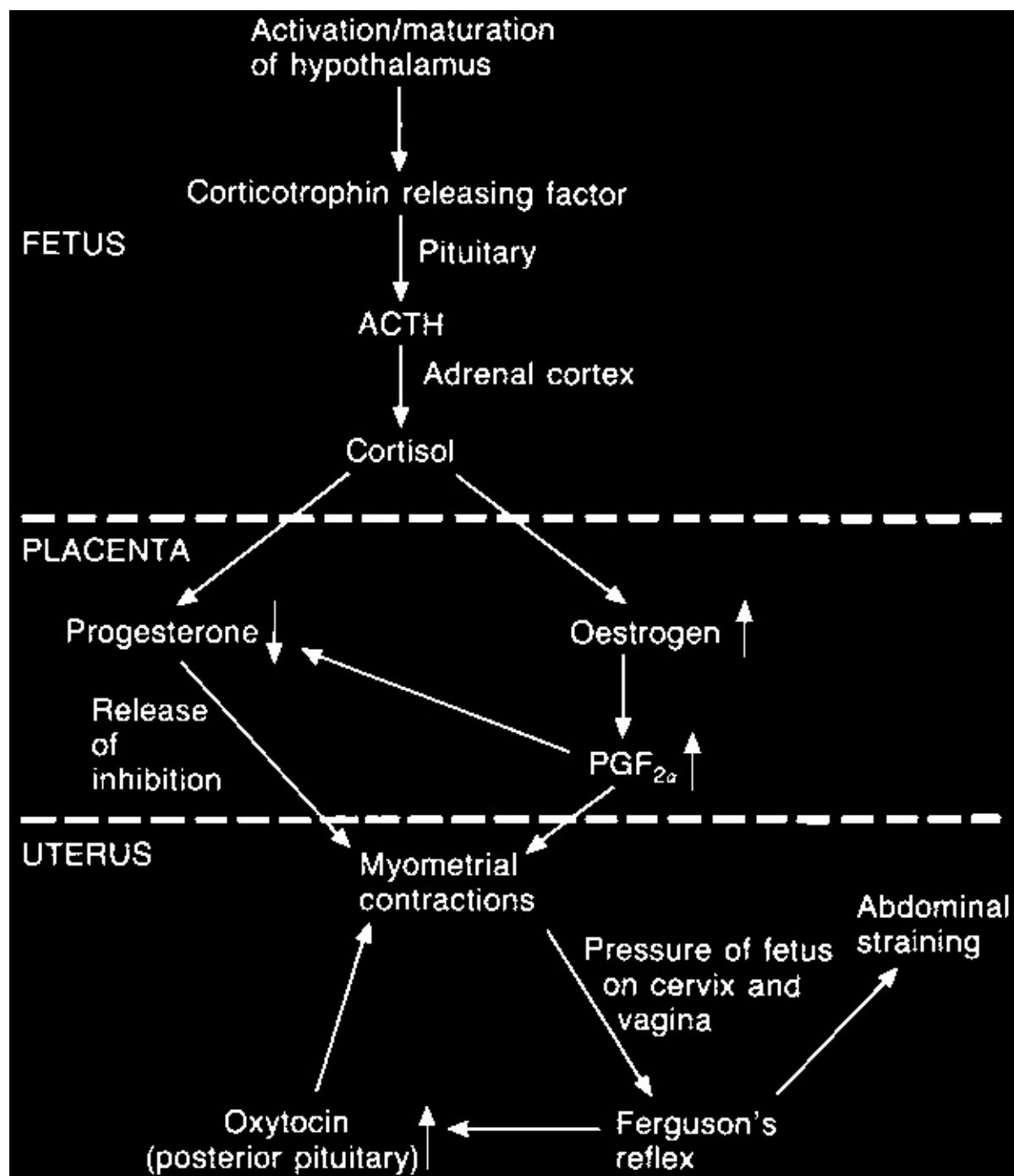
required, this is considered to be an important aid to

cows studied (Sheldon, 1993). When GnRH was used at

optimum management and utilization of feed resources.

11 days after insemination first service pregnancy rates

*Secondly, cows can be induced to calve at a time when
were enhanced by 9.4 per cent when compared with
supervision is most readily available. Thirdly, if it is
untreated controls. This benefit rose to 30 per cent in
suspected that a high calf birth weight might result in
cows treated at third and subsequent services.
dystokia, early induction may alleviate the problem.*



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Oestrogens

There is no clear evidence that exogenous oestrogen is effective in inducing parturition in cows, although in

one trial cited by First (1979) treatment with oestradiol-17 β before corticosteroid treatment shortened the time to delivery and reduced the variation between cows.

Prostaglandins

Prostaglandins, both PGF₂ α and synthetic analogues, may be used to induce parturition in cows, although treatment before day 270 of gestation is not recommended. Parturition usually occurs between one and eight days after injection, but at an average of three days. A higher incidence of retained placenta compared to non-treated cows may be expected.

A study under UK conditions (Murray et al., 1984) used a treatment regimen whereby cows were injected with 20 mg dexamethasone and those that had not calved 10 days later received an injection of 0.5 mg cloprostenol (an analogue of PGF₂ α). Although there was a high incidence of retained placenta, this did not

Fig. 42.5

The endocrine pathways controlling the induction of affect subsequent reproductive performance. It was parturition.

concluded that, provided management was organized adequately to supervise parturition and to take care of the newborn calves, then this procedure could be

Methods of induction of parturition

carried out to advantage.

A characteristic of early studies on the pharmaco-

Corticosteroids

logical induction of parturition was the high rate of

Parturition can be induced quite reliably from about

calf mortality and post calving problems, particularly

day 255 of pregnancy onwards by a single injection of

retained placenta. An important determinant of the

a synthetic glucocorticoid, such as dexamethasone,

incidence of retained placenta appears to be the oestro-

betamethasone or flumethasone. It is assumed that such

gen status of the cow at the time of induction. As dis-

therapy simulates the effect of the fetal adrenal cortex

cussed above, oestrogen concentrations rise during late

at term.

pregnancy, hence the oestrogen status may simply be

Induction of parturition using corticosteroids is an

a reflection of the proximity of term or 'readiness to important part of management in many New Zealand calve'. From an exhaustive review of the available dairy herds and considerable experience of the tech-literature, First (1979) concluded that if induction nique has been gained (Welch et al., 1979; Verkerk et al. is carried out when oestrogen levels are elevated, 1997). Both short- and long-acting formulations have both glucocorticoids and prostaglandins are effective. been used.

However, glucocorticoids were the most appropriate Short-acting formulations, generally in the form of a treatment if induction was to be attempted earlier. The soluble ester of the steroid, usually result in parturition earlier that interference is attempted, the higher the two to three days later. Although the calves are usually probability of calf mortality, retained placenta and viable, this method has been associated with a high rate other related problems.

of retention of the fetal membranes. Verkerk et al. (1997) used 21 mg dexamethasone isonicotinate fol-

Delay of parturition

lowed by a short acting formulation of dexamethasone sodium phosphate 10 to 14 days later. Using this regime It is also possible to delay parturition for several hours 35 per cent of cows calved before the second corticosteroid had been administered. This was more common supervision for calving can be more conveniently and if the cow was in the last 30 days of gestation. A 14 readily available. Injection of the potent adrenergic per cent incidence of retained fetal membranes was drug clenbuterol inhibits myometrial contractions, thus recorded.

slowing down the first stage of labour. However, if treat-

688 • Chapter 42

ment is started after second stage labour has already

esterone during early pregnancy in cattle. Reproduction in commenced, it would have little effect. Clenbuterol is

Domestic Animals, 34, 269–74.

not currently indicated for obstetric use.

Mawhinney, I., Drew, B. & Peters, A.R. (1996) The use of a GnRH and prostaglandin regime for planned breeding of

groups of dairy cows. *Cattle Practice*, **4**, 285–8.

Murray, R.D., Nutter, W.T., Wilman, S. & Harker, D.B. (1984) **References**

Induction of parturition using dexamethasone and cloprostenol. Economic performance and disease incidence

Baishya, N., Ball, P.J.H Leaver, J.D. & St Pope, O.S. (1980) *after treatment. Veterinary Record*, **115**, 296–330.

Fertility of lactating dairy cows inseminated after treat-

Peters, A.R., (1984) *Plasma progesterone and gonadotrophin*

ment with cloprostenol. British Veterinary Journal, **136**, concentrations following Norgestomet treatment with and

227–39.

without cloprostenol in beef cows. Veterinary Record, **115**, Ball, P.J.FH. (1982) *Milk progesterone profiles in relation to* 164–6.

dairy fertility. British Veterinary Journal, **138**, 546–51.

Peters,

A.R.

(1996) *The use of human chorionic*

Beal, W.E. (1983) *A note on synchronisation of oestrus*

gonadotrophin as an antiluteolytic agent in cattle post

in post-partum cows with prostaglandin PGF_{2a} and a

oestrus on luteal performance. Cattle Practice, **4**, 307–11.

progesterone-releasing device. Animal Production, **37**, Peters, A.R., Riley, G.M., Rahim, S.E.A & Lowman, B.G.

305–8.

(1980) Milk progesterone profiles and the double injection

Cooper, M.J. (1974) Control of oestrous cycles of heifers with of cloprostenol in post-partum beef cows. *Veterinary*

a synthetic prostaglandin analogue. *Veterinary Record*, **95**, Record, **107**, 74–7.

200–3.

Roche, J.F. (1999) Reproductive efficiency in post-partum

Coulson, A., Noakes, D.E., Hamer, J. & Cockrill, T. (1980) cows. *Cattle Practice*, **7**, 27–43.

Effect of gonadotrophin releasing hormone on levels of

Roche, J.F. & Ireland, J.J. (1981) *Effect of exogenous proges-luteinising hormone in cattle synchronised with dinoprost.*

terone on time of occurrence of the LH surge in heifers.

Veterinary Record, **107**, 108–9.

Journal of Animal Science, **52**, 580–6.

Diskin, M.G. & Sreenan J.M. (1986) *Progesterone arid*

Sheldon, M. (1983) *GnRH on day 11 after insemination to*

embryo survival in the cow. In Embryonic Mortality in Farm increase pregnancy rates in cattle at first second and subse-Animals (ed. by J.M. Sreenan & M.G. Diskin), pp. 142–55.

quent services. Cattle Practice, **1**, 93–6.

Martinus Nijhoff, Dordrecht.

Smith, R.D., Pomerantz, A.J., Beal, W.E., McCann, J.P.,

Drew, S.B., Wishart, D.R. & Young, I.M. (1979) Fertility of Pilbeam, T.E. & Hansel, W. (1984) Insemination of Holstein Norgestomet treated suckler cows. *Veterinary Record*, **104**, heifers at a preset time after oestrous cycle synchronisation

523–5.

using progesterone and prostaglandin. *Journal of Animal*

First, N.D. (1979) Mechanisms controlling parturition in farm
Science, **58**, 792–800.

animals. In *Animal Reproduction* (ed. by H.W. Hawk).

Sreenan, J.M. & Diskin, M.G. (1986) The extent and timing

Beltsville Symposia in Agricultural Research No. 3, pp.

of embryonic mortality. In *Embryonic Mortality in Farm*
215–57. John Wiley & Sons, New York.

Animals (ed. by J.M. Sreenan & M.G. Diskin), pp. 1–11.

Jackson, P.S., Johnson, C.T., Bulman, D.C. & Holdsworth, R.J.

Martinus Nijhoff, Dordrecht.

(1979) A study of cloprostenol-induced oestrus and sponta-

Stewart, H.J., Flint, A.P.F., Lamming, G.E., McCann, S.H.E. & neous oestrus by
means of the milk progesterone assay.

Parkinson, T.J. (1989) Antiluteolytic effects of blastocyst

British Veterinary Journal, **135**, 378–90.

secreted interferon investigated in-vitro and in-vivo in the

Lamming, G.E. & Mann, G.E. (1995) Control of endometrial

sheep. *Journal of Reproduction and Fertility*, **37**, (Suppl) oxytocin receptors and prostaglandin F2a responses to oxy-127–38.

tocin in ovariectomised cows by progesterone and estradiol.

Thatcher, W.W., MacMillan, K.L., Hansen, P.J. & Drost, M.

Journal of Reproduction and Fertility, **103**, 69–73.

(1989) Concepts for regulation of corpus luteum function

McIntosh, D.A.D., Lewis, J.A. & Hammond, D. (1984)

by the conceptus and ovarian follicles to improve fertility.

Conception rates in dairy cattle treated with cloprostenol.

Theriogenology, **31**, 149–64.

Veterinary Record, **115**, 129–30.

Valks, M. (1996) Factors influencing the benefit of GnRH on

MacMillan, K.L., Taufa, V.K. & Day, A.M. (1986) Effects of an the day of insemination. *Cattle Practice* **4**, 311–17.

agonist of gonadotrophin releasing hormone in cattle: III.

Verkerk, G.A., Goble, G., McDougall, S., Woods, M. & Clark, Pregnancy rates after a post-insemination injection during

B. (1997) Induction of parturition in dairy cows: a survey

metoestrus or dioestrus. *Animal Reproduction Science*, **11**, from spring 1996 in the Waikato region. *Proceedings of*

1–10.

the New Zealand Society of Animal Production, **57**, MAFF (1984) Dairy Herd Fertility. Reference Book 259, p. 46.

231–3.

HMSO, London.

Wathes, D.C. (1984) Possible actions of gonadal oxytocin and

Mann, G.E. & Lamming, G.E. (1995) Effects of treatment with vasopressin. *Journal of Reproduction and Fertility*, **71**, buserelin on plasma concentrations of oestradiol and prog-315–45.

esterone and cycle length in the cow. *British Veterinary*

Welch, R.A.S., Day, A.M., Duganzich, DM. & Featherstone, P.

Journal, **151**, 427–32.

(1979) Induced calving; a comparison of treatment regimes.

Mann, G.E. & Lamming, G.E. (1999) The influence of prog-

New Zealand Veterinary Journal, **27**, 190–4.

Adult Cattle

Major Infectious Diseases

Chapter 43

Viral Diseases

(a) Bluetongue

691

Not all the BTV types are found throughout this

(b) Enzootic bovine leukosis

693

enzootic region, and the distribution of the different

(c) Foot-and-mouth disease

700

types can vary between years. BTV types found within

(d) Rinderpest

707

a region tend to have greater genome sequence homol-

(e) Vesicular stomatitis

710

ogy, in genome segments other than those which encode

(f) Bovine immunodeficiency virus (BIV)

713

*the serotype-specific outer capsid proteins, than their
designated serotype number would suggest. The type is
defined by the outer capsid proteins alone, and while*

(a) Bluetongue

*these are found to differ between different serotypes
found in a particular region, the remaining genome*

R.P. Kitching

*segments show high levels of sequence homology. The
BTV types have therefore been additionally grouped
into Australian types and African types, with the*

Bluetongue (BT) is an infectious, non-contagious North American types being more closely related to disease of ruminants characterized by congestion, the African. The Australian BTV type 1 has more in oedema and haemorrhage. The disease is caused by common with other Australian types than with the strains of orbivirus, within the family Reoviridae. African type 1, although sharing with the African type The genus orbivirus, Reoviridae also contains Ibaraki its antigenic determinants. This diversity may explain disease virus of cattle, epizootic haemorrhagic disease the marked difference in pathogenicity of the strains of virus of deer (EHD), African horse sickness virus and BTV which does not appear to be related to a specific Colorado tick fever virus. The outer shell of bluetongue type designation; it also makes epidemiological studies virus (BTV) has a diameter of 65 nm, within which is an based on serotype determinations of doubtful sig- inner shell of 32 ring-shaped capsomers. The genome nificance. Recombination can occur between different consists of ten segments of double-stranded RNA

strains of BTV which adds further to the potential for which code for the structural and non-structural viral diversity of BTV isolates.

proteins and which can be separated according to their

The closely related EHD group of viruses has been

relative sizes by polyacrylamide or agarose gel

isolated in the USA, Canada, Nigeria and Australia,

electrophoresis. Two of these segments (numbers two

whereas Ibaraki disease virus is restricted to South

and five) code for the outer structural proteins (VP2

Korea, Japan, Philippines and Indonesia.

and VP5) which determine the serotype of the virus.

Twenty-four immunologically distinct BTV serotypes

History

have so far been identified by virus neutralization tests;

however, it is probable that more types will be identi-

Bluetongue was first diagnosed in South Africa in sheep

fied in the future. BTV is sensitive to low pH and

at the beginning of the twentieth century. It was first

storage at -20°C; it is partially resistant to lipid solvents.

seen outside Africa in 1943 in Cyprus, although it had

possibly been present in Cyprus as early as 1924.

Subsequently BT was diagnosed in Israel in 1951, in

Distribution

Pakistan in 1959 and in India in 1963. A disease at

The distribution of BTV is approximately defined by

first identified as sore muzzle of sheep in Texas in 1948

the latitudes 40°N and 35°S, which includes most of

and California in 1952 was the following year diagnosed

Africa, the Middle and Far East, Northern Australia,

as BT. Between 1956 and 1960, BTV caused a major

United States of America, Central America and South

epizootic in sheep in Portugal and southern Spain,

America north from Southern Brazil, Paraguay and

which reportedly resulted in the loss of 180 000 animals,

Bolivia.

but the virus then disappeared from the region.

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Epidemiology and transmission

isolated on less than three occasions and those which

shed virus over a two to three week period. BTV could

The distribution of BTV between 40°N and 35°S only be isolated from the semen when there was a reflects the distribution of its main biological vectors, concurrent viraemia. Six out of nine susceptible certain tropical and subtropical species of Culicoides heifers inseminated with the BTV-contaminated semen midges, in particular C. imicola in Africa and the Middle became pregnant and three of the nine became East, C. variipennis in North America and C. brevitarsis-viraemic. None of the calves born at term showed any sis, C. fulvus and C. wadai in North Australia. clinical abnormality. Considerable importance has been

The adult female Culicoides lays her eggs in damp attached to reports of a bull which was persistently muddy areas containing decaying vegetable material infected but seronegative from birth and intermittently or in cattle dung, two to six days after a blood meal. shed virus in semen over an 11-year period (Luedke

Depending on the temperature these eggs may hatch in et al., 1982). Attempts to duplicate the conditions which two to three days into larva. The larval stage lasts 12–16 produce persistently BTV-infected, seronegative calves

days, followed by pupation and, two to three days later, have been unsuccessful.

the emergence of the adult *Culicoides*. In the subsequent 24 hours the adult females take a blood

Host range

meal and mate, and they will continue to take a blood meal every three to four days until the end of their life, Sheep, goats, cattle, water buffalo, camels and many which may last for 70 days, but probably rarely exceeds wild ruminants are susceptible to infection with BTV.

10. Optimum conditions are between 13°C and 35°C.

BT is predominantly a disease of sheep and has only Larvae of temperate species can remain dormant over been reported as a disease of cattle in the USA, South winter and pupate the following spring. Seven to ten Africa, Israel and Portugal.

days after taking a BTV infected blood meal, vector species of *Culicoides* midge are able to transmit virus.

Pathogenesis

Culicoides usually feed at dusk, during the night or at dawn, and are subject to being transported, sometimes

Infection follows the bite of an infected Culicoides over considerable distances, by strong wind currents. midge. The virus is carried to the local lymph node. The passive movement of infected Culicoides may where primary replication occurs before dissemination be responsible for the introduction of BT into areas of virus throughout the body. Viral replication then usually outside the enzootic region, such as Western Turkey and Cyprus. This introduction of BTV into an lymph glands. In sheep, BTV also replicates in the area may be associated with abnormal wind currents endothelial cells of the blood vessels and, unlike in or may be a regular occurrence. The winds of the cattle, has been clearly shown to cross the placenta and Intertropical Convergence Zone annually reintroduce can replicate in the developing fetus causing fetal BTV-infected Culicoides to South Africa from Central resorption, abortion or developmental abnormalities. Africa. The movement of BTV into Sudan from Central The peak viraemia occurs two to three weeks after

Africa is also associated with a prevailing wind from the infection, its duration and severity depending on the South. However, BTV may also become enzootic in strain of BTV.

new regions as climatic changes allow the main vectors to extend their breeding sites or, alternatively, virulent Clinical signs

strains of new serotypes of BTV may be introduced into an area already infected with mild or avirulent strains.

BT in cattle is seen as a transient fever followed by

Within BT enzootic regions the prevalence of sero-

hyperaemia and erosions of the buccal and lingual

positive animals may be very localized around areas

mucosa and nose and, rarely, the teats. Affected cattle

particularly suitable for the breeding and survival of

salivate excessively and may walk with a stiff gait.

Culicoides, so called 'hot spots'. The possibility also The skin of the nose appears mottled and dark and has

exists for new species of Culicoides to take on the role been described as 'burnt muzzle', and may completely

of BTV vectors; it has recently been shown that some

slough. Fewer than 1 per cent of cattle in the USA

British species of Culicoides can biologically transmit infected with BTV show signs, and the lesions may be

BTV under experimental conditions.

due to a delayed type hypersensitivity reaction. There

Bulls may shed BTV in their semen intermittently

is considerable controversy over whether BTV can

during the viraemia following infection. Bowen et al.

cross the placenta of the pregnant cow. If the virus is

(1985) classified bulls into three categories, those from

able to cross the placenta it may only do so in associa-

which virus could not be isolated from the semen (the

tion with other placental pathogens, or be restricted to

majority), those from which only low titres of virus were

only certain strains of BTV.

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Diagnosis

erologous protection against a third serotype (Jeggo,

1986). There would still be the difficulty of predicting the

It is not possible to make a diagnosis of BT on solely

probable challenge BTV serotypes. The distribution of

clinical signs. Cattle infected with BTV develop precipi-

the serotypes of BTV tends to be dynamic, with some tating antibody detectable on an agar gel precipitation serotypes being present one year to be replaced the fol- test (AGPT) which is not BTV serotype specific. Type- lowing year by other serotypes.

specific neutralizing antibodies can be titrated in a virus

There is no evidence to indicate that BT can be

neutralization test using BHK21 cells in a microplate

transmitted during embryo transfer using standard

against each of the BTV types present or suspected

techniques. BTV can be transmitted in the semen col-

present in the area. Some cross-reaction is seen on the

lected from viraemic bulls, but reports of a persistently

AGPT with antibodies to EHD virus, however a group-

infected, seronegative animal (Luedke et al., 1982) have specific competition ELISA using monoclonal antibody

not been confirmed by further research.

which does not cross-react with EHD antibody is now

available. The viraemia associated with BTV infection

can persist up to 120 days, in the presence of neutraliz-

Economic significance

ing antibodies. The virus is attached to the red blood
The cost of BTV infection in a 1400 dairy cow herd in
cells and appears to be protected from the developing
California, in terms of reduced fertility was \$23 000 over
immune response. Intravenous inoculation of sonicated
a 52-week study period (Osborn et al., 1986). Of parti-blood into eight to ten-
day old embryonated chicken
cular interest in this study was that the dairyman
eggs is a very sensitive laboratory method of isolating
responsible for the herd was unaware of the passage of
virus from blood, whereas the inoculation of sus-
BTV through the herd and the associated increase in
pect material directly into sheep and the examination
return to service of the affected animals. There is, there-
of sequential serum samples for evidence of serocon-
fore, the possibility that BT has a greater economic sig-
version is the most sensitive test available. Virus can
nificance in cattle than previously thought and should
also be grown in the yolk sac of six-day old eggs kept
not be considered solely in terms of isolated epizootics.
between 33.5°C and 35°C, lamb kidney cells, hamster

Animals and semen from BTV enzootic areas are lung cells, BHK21 cells, some mosquito cell lines and subject to movement and export restriction, the costs of following intracerebral inoculation of day old mice.

which are of greater significance than the direct effects

Chick embryos which die from BTV infection have a of disease. Many of these restrictions have been characteristic haemorrhagic appearance.

formulated on unconfirmed experiments, but they reflect the cautious attitude of the veterinary authorities of importing countries.

Control

Bluetongue is a non-contagious disease and can only spread by the bite of infected Culicoides or the direct transfer of blood or semen from an infected to a

(b) Enzootic bovine leukosis

susceptible animal. Bluetongue may be controlled by eliminating the vector or vaccinating susceptible

C. Venables and M.H. Lucas

animals against the serotypes prevalent in the area.

Control of insects has usually been directed towards

those insects which carry human disease, and experi-

Two forms of bovine leukosis have been recognized, experience gained in these programmes could undoubtedly be enzootic bovine leukosis (EBL), which occurs in adult of value in BT control. However, there would be little cattle and is associated with bovine leukaemia virus economic justification for attempting to control Culi- (BLV) infection, and, less commonly worldwide, spocoides solely to prevent BT in cattle

radic bovine leukosis (SBL), which affects calves and Cattle can be protected against BTV infection by vaccination (p. 1018) with live attenuated vaccine, although young cattle and is of unknown aetiology.

it would be extremely unlikely for it to be considered

Enzootic bovine leukosis

worthwhile. The practice of mixing together vaccines against each of the prevalent serotypes may result in the Enzootic bovine leukosis was first described over 100 failure of one or more of the serotypes present in the years ago in Europe. Clinical disease, in the form of vaccine to replicate (Jeggo, 1986). However, vaccination

multiple cases of lymphosarcoma, occurs most frequently against one serotype, followed one month later with a second serotype can provide protection not only against Tumours may develop in peripheral lymph nodes, and the two serotypes in the vaccines, but can provide an heterogeneous are therefore easily detectable, or they may be confined

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to internal organs, producing rather ill-defined signs. In significant animal health problems, EBL can also represent some areas of the world appreciable economic losses

resent a major cause of economic loss.

occur through the tumorous form of the disease. Early workers recognized that the disease could spread slowly

Transmission

from known foci to adjoining regions, and because lymphosarcoma often occurred in familial aggregations it Virus is present in colostrum and milk, in tracheal and was interpreted as evidence that the disease was hereditary bronchial secretions and sometimes in nasal secretions itable. It has since been established that EBL is caused

and saliva of infected animals. Virus is also found in the by bovine leukosis virus (BLV). This virus was first cellular fraction of blood, but not in plasma or serum isolated from mitogen-stimulated, peripheral blood unless haemolysis has occurred. However, it has not mononuclear cells by Miller et al. (1969) in the USA. been found in faeces or urine and is probably absent from the semen of most infected bulls. Very small numbers of infected blood cells are capable of trans-

The virus

mitting BLV. As little as 0.1 ml of whole blood from an Bovine leukosis virus is an RNA virus belonging to the infected cow can be infectious when given intra-Retroviridae family. The family includes tumour and dermally to cattle.

non-tumour inducing viruses of many species including man. The virus particle is spherical, 80–100 nm in Experimental transmission: Cattle can be infected diameter and enveloped with surface glycoprotein produced by the intratracheal route, although not as reliably as by injections. The virus envelope glycoprotein, gp51, is be-

the subcutaneous route. Infection can be transmitted
by interaction with a cell surface receptor, initiating
by instillation of infected lymphocytes into the nose and
receptor-mediated endocytosis, leading to entry into the
by aerosol exposure to cell-free BLV. A calf can be
host cell. Within the particle are three non-glycosylated
infected orally when newborn, but is probably resistant
protein structures, the matrix, the nucleocapsid and the
to infection by this route by three weeks of age. Adult
core. Three enzymes, reverse transcriptase, integrase
cattle are not susceptible to infection by the oral
and a protease, are also packaged within the virion. The
route. Adult cows can be infected by the instillation
retrovirus genome is composed of two identical copies
of infected lymphocytes into the reproductive tract.
of positive sense, single-stranded RNA. During replica-
Semen mixed with the inoculum may, however, have an
tion, viral RNA is converted to DNA by means of the
inhibitory effect on transmission, and susceptibility of
enzyme reverse transcriptase. This enables retroviruses
the genital tract of cows may decrease at the time of

to integrate into the host DNA, establishing infection
oestrus. The virus has been transmitted experimentally
that persists for the life of the animal. Bovine leukosis
to sheep and cattle by rectal inoculation of whole blood
virus is present in a subpopulation of circulating B-
from infected cattle.

lymphocytes where its genetic information may be found
Sheep can readily be infected experimentally by par-
integrated at multiple sites in the cellular DNA. Lym-
enteral routes, but not consistently by the oral route.

phocytes of BLV-induced tumours appear to be of the
Between 10³ and 10⁶ lymphocytes from infected cattle,
B-cell lineage. Tumour cells are monoclonal or oligo-
given intravenously, are sufficient to infect sheep.

clonal for the site of BLV integration. No evidence has
Tumours arise at a higher frequency and after a much
been observed so far for a common integration site for
shorter time than in cattle and may develop after 10
BLV provirus in different tumours.

months to three years. As in cattle, various lymph nodes
BLV probably does not remain viable for long

and visceral organs including heart, abomasum, uterus, outside the host environment. It is readily inactivated kidney and urinary tract are commonly affected. Per- by exposure to ultraviolet light, heating at 56°C for 30 sistent lymphocytosis is not usual in sheep; once the minutes and pasteurization. The virus can, however, number of circulating lymphocytes rises it invariably remain viable in blood stored at 4°C for at least two indicates the onset of tumour development. In contrast weeks.

to cattle, contact transmission does not occur when infected sheep are kept in close contact with uninfected sheep. Goats inoculated with BLV orally and parenter-

Distribution

ally become infected and develop antibody but do BLV is distributed worldwide, but with marked regional not usually develop persistent lymphocytosis or lym- differences in prevalence. It is most common in North phosarcoma. The incubation period in sheep and goats and South America, Australia and some regions of is variable, ranging from about two to 16 or more weeks.

Africa. In Western Europe BLV has largely been eradicated. Experimental infection with BLV as indicated by persistent antibody production has been reported for chimpanzees, macaques, pigs, domestic rabbits, cats, dogs, and deer and rats. There is no evidence for the production of BLV antibodies following BLV inoculation in the mouse, chipmunk, ground squirrel, Japanese quail and chicken. It was concluded that it was safe to transfer embryos from infected cows providing that the embryos were washed before transfer. Virus transmission to dam or to progeny is not associated with the use of semen from infected bulls for artificial insemination (AI). Naturally

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Natural transmission: Transmission of virus by contact occurring lymphosarcoma in sheep is rare. A retrovirus is one of the most important means of spread of BLV has been isolated from a diseased sheep and was found in a herd. The rate of spread of virus is influenced by

to be identical to BLV. Where BLV is found in sheep a management and husbandry practices that determine bovine origin must be suspected. The virus has also the degree of contact between animals. Because the been found in capybaras and water buffaloes. Human virus probably does not survive for long in the environment and because infectivity is associated with the BLV antibodies.

cellular fraction of secretions it is therefore assumed that transmission takes place by direct exchange of
Signs

infected lymphocytes in nasal, saliva and tracheo-
Following infection of an animal with BLV disease
bronchial fluids and possibly vaginal discharges. The
progression depends on genetic, environmental and
role of milk in transmission under natural conditions
unknown factors. Over 60 per cent of infected cattle are
does not appear to be very great. The presence of spe-
asymptomatic, however almost all develop detectable
cific antibody in these secretions inhibits virus trans-

antibodies. Between 30 and 70 per cent of infected animals show persistent lymphocytosis, but less than 10 per cent develop lymphosarcoma. Persistent lymphocytosis is seen in 28–85 per cent of tumour cases. In BLV-infected cattle with leukaemia the increase in leukocyte count is due to an increase in B-lymphocytes. The percentage of B-lymphocytes in the blood can rise to 80 per cent, compared with normal values of 15–20 per cent. In clinically normal BLV-infected cattle without neoplastic lesions there can still be an increase in B-lymphocytes to 40–50 per cent.

be important in tropical and subtropical climates, but Clinical signs (Figs 43.1, 43.2) in animals that develop statistical evidence of seasonal trends is inconclusive. tumours depend on the particular organ or organs Iatrogenic transmission is probably one of the main involved. One or more superficial lymph nodes may be reasons for the high prevalence of infection in some enlarged and these can be felt as lumps beneath the herds. Infected blood transferred from one animal to skin, especially in the neck and hind flank areas. another on a hypodermic needle can transmit bovine However, when the internal lymph nodes are the only leukosis virus. The use of multidose syringes has been ones affected diagnosis may be more difficult. Tumours incriminated in virus spread, however there is no can occur in the abomasum, right side of the heart, published evidence to associate intradermal tuberculin spine, uterus, lymph nodes, central nervous system (Fig. testing with an increased incidence of enzootic bovine 43.3) and the retrobulbar aspect of the orbit. Clinical leukosis. Dehorning and ear tattooing have been iden-

signs may include depression, indigestion, chronic bloat, tified as possible methods of transmission. It has been displaced abomasum, lameness or paralysis. Abdominal suggested that the technique of rectal palpation to tumours are sometimes detected by rectal palpation examine the reproductive tracts of cows could be a during pregnancy examination. Infection with BLV means of transmission if separate clean gloves were not does not appear to be associated with lower milk produced for each animal.

duction, impaired reproductive capacity in either sex, or Transfer of embryos from BLV-infected cows into with mastitis, lesser longevity or increased susceptibility to other diseases. Bovine leukosis virus does not virus. Virus has nevertheless been found in uterine flush appear to cause significant immunosuppression in the fluid, but this may be due to contamination with blood fetus or adult animal.

cells. Virus has not been found in eggs or embryos from infected cattle. Ova, morulae and blastocysts have been

Diagnosis

exposed in vitro to BLV, but after washing no virus could be detected. Embryos similarly exposed to virus

Haematology: Herds with a high incidence of lymphosarcoma often contain many clinically normal cattle which did not subsequently develop antibodies to BLV. with persistent lymphocytosis. The development of





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Fig. 43.2

Adult cow with enzootic bovine leukosis showing loss of condition, with wasting of lumbar muscle, brisket and sub-mandibular swelling. (Courtesy of J. Miller, USA.)

Fig. 43.1

A cow with enzootic bovine leukosis showing enlarge-

ment of the mammary lymph nodes. (Courtesy of Dr J. Miller, USDA National Animal Diseases Center, Ames, Iowa, USA and editors and publishers of Modern Veterinary Production.) lymphosarcoma is often preceded by persistent lymphocytosis in the absence of any clinical signs. Haema-

tological methods were the main diagnostic tools for a number of years and various 'keys' were developed

Fig. 43.3

Brain from a cow with neoplasm between cerebellum relating lymphocyte counts and age, presenting and medulla. (Courtesy of J. Miller, USA.)

maximal values above which an animal was declared to have persistent lymphocytosis. The percentage of B-lymphocytes in normal cattle varies from 18 to 28 per cent. In BLV-infected cattle with persistent lymphocytosis the percentage of B-lymphocytes can increase to the detection of infected animals. The test is simple and as high as 70 per cent. In clinically normal BLV-infected practical and has been very widely used. The glycoprotein antigen employed in the test is prepared from the

increased to 40–50 per cent.

supernatant fluid of a cell line persistently infected with BLV.

Serology: The agar gel immunodiffusion test (AGIDT)

Using BLV-infected cell monolayers, sera can also be used to detect specific antibody to viral antigens tested for specific antibodies by indirect immunofluorescence or immunoperoxidase techniques. Various

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neutralization tests have also been described, based on and politics may all be significant. Increasingly, driven the ability of antibodies to inhibit the effects of BLV in by trade advantages, EU Member States and other cell cultures. These include a virus neutralization and European countries have set in place surveillance and syncytia inhibition tests. The complement fixation test eradication strategies. Many of these have been using viral antigens from a cell line infected with BLV successful and the countries are now officially EBL- has been used, but seems to be less sensitive than the

free. The types of actions used, linked with serological agar gel immunodiffusion test for antibodies to the gp monitoring, to produce BLV-free herds are detailed antigen. A reverse transcriptase inhibition test, based below:

on the fact that serum of some leukaemic cattle inhibits

- BLV-positive animals are kept physically separated the activity of the reverse transcriptase of BLV, has from BLV-negative animals. Check testing of negative animals is carried out at regular intervals.

but has the disadvantage that radiolabelled reagents

- Calves born to negative cows are kept apart from and special equipment are required. infected animals.

Enzyme-linked immunosorbent assay (ELISA) is the

- Calves born from infected cows are reared only on currently preferred method for the detection of BLV colostrum and milk from negative cows and are antibodies. The test can be used with milk or tissue kept isolated. If serologically negative at seven

fluids as well as serum samples. ELISA is rapid, sensitive and suited to the testing of large numbers of herd.

months of age these calves join the negative

samples. Commercially available ELISA kits, sufficiently sensitive to permit the examination of pooled

- Replacement animals should be introduced from sources known to be virus-free, and should be sera and milk for screening and surveillance, have logically screened. Any originating from herds of formed the basis of successful EBL eradication programmes in several European countries.

at six-month intervals before joining the main herd.

- Embryo transfer and AI are sometimes used as part of a control programme so that new genetic stock virus particles can be demonstrated by electron microscopy in short-term cultures of lymphocytes from BLV.

BLV-infected animals. Virus can also be recovered from infected cattle by cocultivation of peripheral blood

Where the prevalence of infected cattle has been low, mononuclear cells with a susceptible indicator cell, e.g. as has been the case in most Western European countries now free of EBL, serological screening followed by slaughter of reactors has been successfully used.

Giemsa-stained monolayers. Infected cells may be

However, increased prevalence of infection rapidly specifically visualized, often in advance of syncytium formation, using immunofluorescence or immunoperoxidase techniques. However, as with other retro-

viruses, neither electron microscopy nor virus isolation

formation, using immunofluorescence or immunoperoxidase techniques. However, as with other retro-

viruses, neither electron microscopy nor virus isolation

Vaccination

is a particularly sensitive method for demonstrating

Preliminary experiments using inactivated BLV, perinfection.

sistently infected cell lines or purified gp51 indicated

Nowadays, techniques including assaying for reverse that high antibody titres to gp51 could produce some transcriptase activity or demonstrating the presence of short-term protection to BLV infection. However, no specific BLV nucleic acid sequences are frequently successful vaccine is currently available.

used. Polymerase chain reaction (PCR) assays, generally developed to detect BLV proviral DNA and offering exquisite sensitivity and specificity, are rapidly

Public health

gaining the advantage over cultural methods in the research, diagnostic and surveillance fields.

The structural similarities between BLV and the human T-cell leukaemia viruses, HTLV I and II, have led to speculation about the zoonotic potential of BLV. Sero-logical reactions to BLV p24 in two small studies of Control

people with these diseases and multiple sclerosis have Control measures appropriate to any particular situa-also been reported, but current opinion indicates sero-tion are largely dictated by local factors. Prevalence

*logical cross-reactions with HTLV proteins to be the
of infection is probably the primary consideration,
cause. This is supported by repeated failure to demon-
but economics, husbandry practices, cattle movements
strate BLV proviral DNA in other studies on human*



Sporadic bovine leukosis in calf with parotid lymph node enlargement. (Courtesy of J. Miller, USA.)

Fig. 43.5

Sporadic bovine leukosis in calf with submandibular, parotid and retropharyngeal lymph nodes on the head and prescapular lymph node enlargement. (Courtesy of J. Miller, USA.)

leukaemia patients and the failure of epidemiological marrow (Figs 43.4–43.7). During the early stages of studies to demonstrate a link between BLV and human disease, marked enlargement of superficial lymph disease.

nodes may be the only clinical sign. But as the disease progresses internal organs such as the heart and liver may also become affected, leading to the

Sporadic bovine leukosis

death of the animal.

Lymphosarcoma may be found in young animals in the

- *The thymic form is seen in animals 6–30-months old. There is massive tumour formation in the thymus and tumorous changes are also seen in the lymph nodes of the neck and thorax (Fig. 43.8). The condition is fatal.*

- *The juvenile form in calves under six months of age involves lymph nodes, liver, spleen and bone in animals 18 months to three years of age, in*





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Fig. 43.6

Same calf as in Fig. 43.5

showing gross distension of the head

lymph nodes and the prefemoral lymph

nodes. (Courtesy of J. Miller, USA.)

Fig. 43.7

Liver from a calf with spo-

radic bovine leukosis showing the

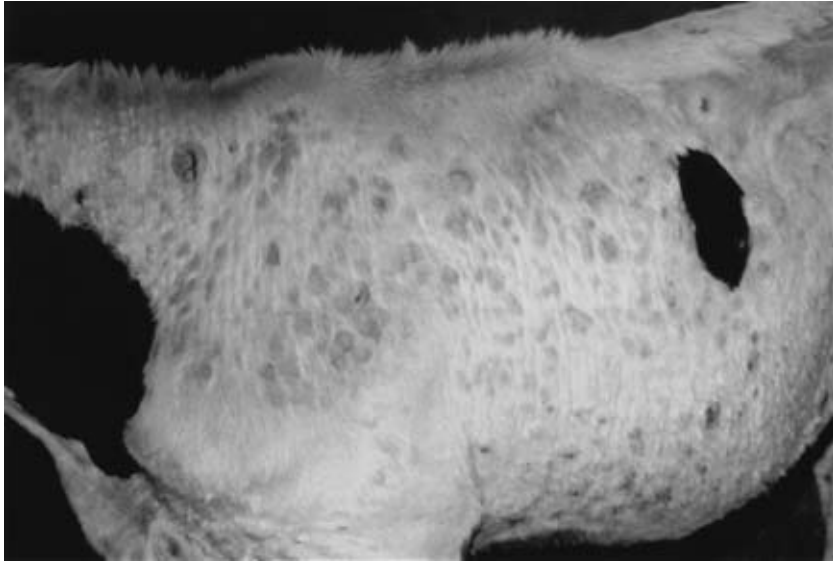
surface and cut surface with focal neoplastic areas. (Courtesy of J. Miller, USA.)

which nodular lymphocytic neoplasia is seen in the skin. The first signs are urticaria-like nodules (1–2 cm in diameter) in the skin, especially round the neck, back and thighs (Fig. 43.9). These become encrusted with thick scabs, and alopecia and hyperkeratosis follow. Apparent recovery may take place over several weeks (Fig. 43.10). However, the remission is temporary, and lesions reappear together with general lymph node involvement, leading to the death of the animal.

The cause or causes of all forms of sporadic bovine

Fig. 43.8

Bullock showing thymic neoplasm. (Courtesy of J. leukosis remains unknown. Miller, USA.)



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Fig. 43.9

A heifer with acute cutaneous leukosis. (Courtesy of J. Miller, USA.)

Fig. 43.10

Same heifer as in Fig. 43.9

with cutaneous leukosis lesions subsequently resolving. (Courtesy of J. Miller, USA.)

(c) Foot-and-mouth disease

naviridae. There are seven antigenically distinct types of FMD virus, identified as types A, O, C, SAT (South R.P. Kitching

African Territories) 1, SAT 2, SAT 3 and Asia 1. Within each of these seven types there are a large number of strains which form an antigenic spectrum, from closely Foot-and-mouth disease (FMD) is a highly contagious related strains to strains so antigenically different as to disease of domesticated and wild ungulates characterized by vesicles in the mouth and on the feet. Hedge- almost justify the establishment of additional types.

Attempts to classify the strains into subtypes within the hogs and very rarely man may also become infected. types founded on the ever increasing number of strains which fulfilled the criteria for creating a new subtype. New isolates of FMD virus are now referred

Aetiology

to by the World Reference Laboratory for FMD by
Foot-and-mouth disease is caused by infection with a
their type, country of origin, a sequential number relat-
virus of the genus aphthovirus, in the family Picor-
ing to the number of isolates received in that year from
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the same country and the final two numbers of the year
Epidemiology

in which they were received, e.g. O India 53/79, Asia 1
India 8/79. The cause of recent infections in Europe,
Foot-and-mouth disease is an extremely contagious
South Africa, Asia, the Far East and the Middle East
disease, with as few as ten infectious units being able to
(Pan Asia strain) was first isolated from an outbreak
initiate disease in a bovine by the respiratory route. The
of FMD in northern India in 1990 (Knowles et al.,
virus can survive in dry faecal material for 14 days in
2001). Classical subtyping is now only of historical
summer, in slurry up to six months in winter, in urine
interest.

for 39 days and on the soil between three days in summer and 28 days in winter. FMD virus is, however, diameter of 24 nm. The outer capsid consists of 32 capsomeres and surrounds a single-stranded molecule of RNA of approximately 8000 bases and molecular weight 2.8×10^6 daltons. This RNA codes for a single large polyprotein which is cleaved into eight non-structural proteins (L, 2A, 2B, 2C, 3A, 3B, 3C and 3D) and the four structural proteins (VP1, VP2, VP3 and VP4), 60 copies of which make up the outer capsid. The virus is optimal when the relative humidity is above 60 per cent. Natural ultraviolet light in sunlight has little

reflected in variations in the amino acid sequence of the direct effect on the FMD virus.

proteins for which it codes. The structural characteris-

Like many diseases, FMD is most commonly spread

tics of the outer capsid proteins of the virus stimulate

by the movement of infected animals; of particular sig-

an immune response in the infected host animal. Thus

nificance are sheep, goats and wild ungulates, because

mutations in the genome which change the structure of

disease in them can be mild, and pigs because of the

these proteins can reduce the ability of a vaccinated

amounts of virus they can excrete. An infected pig

or previously infected animal to resist challenge with

excretes up to 400 million infectious units per day, 3000

mutated virus.

times more than an infected bovine, sheep or goat. In

infected cattle, milk products and semen many contain

FMD virus up to four days before the appearance of

clinical signs and can also be responsible for the spread

Distribution

of disease. Pigs can carry virus for ten days before

Foot-and-mouth disease is endemic throughout sub-Saharan Africa as far south as Tanzania, and also may also be contaminated with virus from infected carcases, although the reduced pH of the carcase following rigor mortis is sufficient to inactivate the virus in the and Far East. Canada, Central and North America, Australia, New Zealand, Japan, Argentina, Chile and South Korea are free of FMD. Most of Europe is also free of FMD, but suffers occasional outbreaks of disease in temperate countries where the climate is conducive to the survival of the virus. There is evidence to indicate however, it can be severe, as in the European and in particular the British outbreak commencing February sea and up to 60 km over land. The spread of disease by

2001. From 1992 routine vaccination against FMD the wind is dependent on the amount of virus generated ceased in all countries of the European Union and has by infected animals, the weather conditions, the topography since also stopped in Eastern Europe, other than parts of Russia. In Southern Africa FMD virus is usually the animals contacting the airborne virus. A plume of restricted to wildlife in the game parks, although it virus will be subjected to vertical and horizontal dispersion, which is related to wind speed and turbulence, Types O and A of FMD virus are the most wide- the vertical air temperature gradient and ground topography; the survival of the airborne virus will depend on Asia; types SAT 1, SAT 2 and SAT 3 are generally relative humidity. Cattle have a large respiratory tidal restricted to Africa, although they have periodically volume compared with other FMD susceptible stock spread into the Middle East; Asia 1 occurs in the Far

and can be infected following inhalation of relatively East and India, although it also has spread into the low quantities of virus and thus are most at danger to Middle East. Type C only rarely causes outbreaks in infection from the airborne virus. Windborne spread of Asia and has all but disappeared (Kitching, 1998). FMD virus is believed to have occurred in 1981 when



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Fig. 43.11

A recently ruptured vesicle in the mouth of a cow with foot-and-mouth disease, showing epithelial separation.

In tracing the movement of FMD between countries, and identifying specific strains of FMD virus, molecular epidemiology has proved very valuable (Samuel et al. , 1997). By precisely characterizing the RNA of an outbreak strain by its nucleotide sequence, it is possible to

show its relationship to strains previously isolated in other countries or in use in vaccines. For instance, in Europe between 1970 and 1990 there were a number of FMD outbreaks attributed to the use of improperly inactivated vaccines and escape of virus from establishments producing vaccine. In several instances biochemical analysis of the outbreak strains showed a clear identity with a vaccine strain in contemporary use.

Monoclonal antibodies, which specifically identify

Fig. 43.12

Recently ruptured vesicles on the tongue and some of the individual antigenic determinants on the dental pad of a cow with foot-and-mouth disease. (Courtesy of FMD virus, are becoming important in FMD virus

A.I. Donaldson.)

strain characterization. These determinants may change as the virus mutates through the course of an outbreak. Cattle recovered from FMD and vaccinated cattle in infected pigs in Brittany, France, spread disease to cattle contact with FMD virus may retain virus in their pharyngeal region for many months. This is the carrier predominantly over sea. Computer models now exist state. Vaccinated cattle which have had contact with which can predict the likely windborne spread of virus disease may also develop a pharyngeal infection from an infected herd. The maximum daily excretion of without showing any clinical signs. The significance of virus can be calculated by establishing the number of these carrier animals is not clear but, although it has clinically infected animals and the species infected, and proven difficult to show transmission from a carrier to estimating the duration and quantity of airborne virus a susceptible animal under experimental conditions, excreted. The local meteorological office provides

there is considerable circumstantial evidence supported information about wind speed and direction, relative by sequencing of carrier and outbreak strains suggest-humidity and precipitation. When combined with local ing that carriers may have initiated outbreaks.

topographical information and local distribution of live-stock holdings, the computer model can give an indication of which herds are most at risk from secondary

Transmission and pathogenesis (Figs 43.11–43.13)

windborne spread of FMD. Manpower resources for surveillance activity can then be concentrated on the Cattle are most susceptible to FMD by inoculation of herds adjudged to be at greatest risk.

the virus intradermally into the tongue and this is a



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An infected bovine can excrete up to $10^{5.1}$ infectious doses/day by the respiratory route and can provide a potent source of FMD virus to the remaining uninfected cattle in the herd. This may be sufficient to overcome a waning vaccinal immunity.

The incubation period for FMD can be up to 14 days with low infecting doses and with strains of virus of low virulence. However, as the quantity of virus in the environment of an FMD outbreak increases, the incubation period in cattle decreases. For susceptible cattle in

contact with an infected animal it is frequently between two and four days.

Clinical signs

The incubation period is between two and 14 days, depending on the route of infection, the dose, the strain of virus and the susceptibility of the host. Following an initial pyrexia in the region of 40°C (104°F), lasting one or two days, a variable number of vesicles develop on the tongue, hard palate, dental pad, lips, muzzle, coronary band and interdigital space. Vesicles may also be seen on the teats, particularly of lactating cows. Young calves may die before the development of vesicles because of a predilection by the virus to invade and destroy the cells of the developing heart muscle.

The vesicles in the mouth quickly rupture, usually

Fig. 43.13

Vesicular lesions of the interdigital skin of the foot within one to two days of their formation, leaving a shallow ulcer surrounded by shreds of epithelium. The vesicles on the tongue frequently coalesce and a large

proportion of the dorsal epithelium of the tongue may commonly used method of challenging cattle during be displaced. The vesicles on the feet may remain for vaccine trials. However, natural infection is most frequently by inhalation of droplets containing FMD virus terrain or floor surface of the cattle accommodation. or by ingestion of FMD virus contaminated material. Healing of the mouth lesions is usually rapid; the ulcers One infectious unit of FMD virus is sufficient to infect a bovine by intradermolingual inoculation, while fill with fibrin and by day 10 after vesicle formation they appear as areas of pink fibrous tissue, still, however, between 10 and 100 infectious units can initiate disease without normal tongue papillae. Healing of the lesions in a bovine following inhalation. Many thousand infection on the feet is more protracted and the ulcers are susceptible to secondary bacterial infection. The horn of ingestion, although less will be required by a calf for the heels may become under-run, as a consequence

lowing insufflation of infected milk.

of both the initial vesicle and secondary bacterial

The primary site of replication of inhaled virus is in infection.

the pharynx and lymphoid tissue of the upper respira-

Acutely infected cattle salivate profusely and

tory tract. FMD virus then enters the blood stream, is

develop a nasal discharge, at first mucoid and then

distributed around the body and following secondary

mucopurulent, which covers the muzzle. They stamp

replication in other glandular tissues appears in the

their feet as they try to relieve the pressure on first one

body fluids such as milk, urine, respiratory secretions

foot and then another. They may prefer to lie down and

and semen, before the appearance of frank clinical signs

resist attempts to raise them. Lactating cattle with teat

of FMD. However, it is during the early vesicular stage

lesions are difficult to milk and the lesions frequently

of the disease that the majority of virus is excreted into

become infected, predisposing to secondary mastitis.

the environment. Milk may contain $10^{6.7}$ infectious

Affected cattle quickly lose condition; the drop in milk yields 50/ml, semen $\log_{10} 6.2$ infectious doses/50/ml, urine $\log_{10} 4.9$ infectious doses/50/ml and faeces $\log_{10} 5.0$ infectious doses/50/g.

completely regain their previous condition, due to the
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development of lesions in the thyroid gland – ‘hairy when diagnosis is performed within a country, it is recommended that duplicate samples also be sent to the

An outbreak of FMD can be economically devastating. WRL or regional laboratory for confirmation of diagnosis in an intensively farmed region. However, in the extensive husbandry systems of South America and submission of samples are described by Kitching and Africa, where expectations of cattle productivity are low, FMD may seem insignificant compared with the

apply to sending pathological specimens by air.

prevalent clostridial, haemoparasitic and deficiency

Foot-and-mouth disease virus is very sensitive to pH

diseases. This attitude frustrates programmes to com-

values away from neutrality and is, for example, quickly

pletely control FMD and attempts to introduce

inactivated below pH 6. Virus can also be isolated with

intensive farming or a dairy industry.

heparin from vesicular fluid or from whole blood col-

lected from the viraemic animal (up to four days after

the initial appearance of vesicles). Although high titres

Pathology

of virus can be recovered from milk and internal

The epithelial cells of the stratum spinosum of the skin

body organs such as lymph nodes and muscle, these

undergo ballooning degeneration. As the cells disrupt

specimens should only be sent in addition to, and not

and oedema fluid accumulates, vesicles develop which

instead of, epithelium samples. Negative tests on these

coalesce to form the aphthae and bullae that charac-

tissues could be misleading and cause a false sense of

terize FMD. The cells of the squamous epithelium of security.

the rumen, reticulum and omasum may also become

On receipt at the WRL, epithelial samples are pre-involved. In young animals the virus invades the cells pared as 10 per cent suspensions for the enzyme linked of the myocardium and macroscopic grey lesions may immuno-sorbent assay (ELISA). This test identifies be seen particularly in the wall of the left ventri-virus antigen within the sample and can distinguish cle, giving it a striped appearance (tiger heart). Cells between the seven FMD virus types; it has now of the skeletal muscles may also undergo hyaline replaced the classical complement fixation test for degeneration.

FMD diagnosis. Results from this test are available within three hours of arrival. At the same time as the ELISA test is being prepared, samples of the epithelial
Diagnosis

suspension are inoculated onto primary bovine thyroid

Initial diagnosis is usually on the basis of clinical signs,

cells and pig kidney cells (IB-RS-2 cells). If a clear positive result is not obtained from the ELISA test, virus growth and an infected animal or reports of FMD in the vicinity on either or both of these two cell systems would provide sufficient antigen after 24 or 48 hours for a frequently severe and pathognomonic. However, in second ELISA test. In the absence of virus growth on endemic regions in herds which have a partial natural thyroid or pig kidney cells after 48 hours (first passage) or vaccinal immunity, clinical signs may be mild and the cells are inoculated onto fresh thyroid and pig may be missed. All vesicular lesions in cattle should be kidney cells (second passage). Samples are considered investigated as potential FMD (OIE, 1996).

negative following negative ELISA and failure of virus growth after 48 hours on second tissue culture passage, The success of the laboratory confirmation of a presumptive diagnosis of vesicular virus infection depends i.e. after a minimum of 96 hours following arrival at the

on the submission of adequate material, sent under suitable conditions. A minimum of 2 square cm of epithelium from a ruptured vesicle in a 50/50 mixture of diagnostic samples.

glycerine and 0.04 molar buffered phosphate (pH 7.4–7.6) should be sent to a laboratory designated for there is a requirement to relate the outbreak strain to handling FMD virus and equipped with the reagents existing vaccine strains. This can be shown using a two-way microneutralization test. Mixtures of field virus

Diagnosis of FMD is usually controlled by a government department. Where laboratory diagnosis cannot be adequately carried out within a country, samples should be sent by the relevant government department

Diagnosis of FMD is usually controlled by a government department. Where laboratory diagnosis cannot be adequately carried out within a country, samples should be sent by the relevant government department

microtitre plate. The plates are placed in a 37°C incubator to the regional FMD laboratory or to the World Reference Laboratory (WRL) for Foot-and-Mouth Disease, Institute for Animal Health, Pirbright Laboratory, Ash which neutralizes 100 tissue culture infective dose (TCID₅₀) of virus is calculated and compared with the Viral Diseases • 705

titre of the same serum which neutralizes 100 TCID₅₀ Control

of the vaccine virus. The ratio of titre of serum against field virus to titre of serum against vaccine virus is the

The control of FMD depends on prevention of the

r 1 value, and gives an indication of the antigenic relationship between the field and vaccine strains and the prevention of spread of virus from infected

therefore the probable usefulness of that vaccine in animals. How this is achieved by individual coun-

controlling the outbreak of FMD.

tries depends on a variety of economic and practical

A similar r_1 value can be derived using the ELISA considerations.

(Kitching et al., 1988).

Exotic FMD virus may enter an area or country

The serum antibody titre against FMD virus of cattle

in infected animals and this can include certain zoo

vaccinated against FMD can also be measured using the

animals. Acutely infected animals are usually recog-

virus neutralization (VN) or ELISA test. Immunity in

nized, but sheep and goats frequently only develop very

cattle to FMD can be correlated with the level of serum

mild clinical signs. Vaccinated cattle can also develop

antibody at 30 days after primary vaccination, although

only local lesions of FMD, particularly in the mouth,

the relationship is not absolute, being dependent on the

and may carry infection if imported to FMD-free

challenge dose of virus and the closeness of its antigenic

areas. The importance of cattle and buffalo that are car-

relatedness to the vaccine strain. The VN and ELISA

rying FMD virus in the pharynx is not proven, although are FMD virus type-specific, so that when used to determine whether an animal has had contact with FMD the tests must be performed separately against each of the introduction of FMD in this manner, FMD-free countries may refuse entry of any ungulate from FMD contact. In some parts of the world, e.g. Africa, this endemic areas or may insist that any ungulate entering could be any one of up to six different FMD serotypes. the country has no serum antibody to FMD virus

Such testing can be time-consuming, expensive and and that oesophageal-pharyngeal scrapings taken by present results difficult to interpret. A non-type-specific probang are negative for the presence of FMD virus. screening test has been developed which estimates the Any animal vaccinated against FMD is, therefore, presence of antibody to the non-structural proteins of prohibited.

FMD virus; these are formed in an infected animal as FMD virus could also enter in the carcass or product the virus replicates and the non-structural proteins are products of an animal infected before slaughter. In skeletal muscle the virus is inactivated as the pH of the meat falls as the carcass 'sets', but virus in the bone and lymph glands is not subject to this increased acidity and will escape inactivation. Regulations in FMD-free countries require that meat imported from endemic areas has had the bones and lymph glands removed and recovered animals, have been obtained by regular vaccination and certification of the absence of

expressed. The antigenic characteristics of these proteins are conserved between serotypes. Traditionally the 3D non-structural protein or virus infection associated antigen (VIA antigen or polymerase) was used, but better results, particularly in distinguishing between vaccinated and recovered animals, have been obtained by detecting antibody to the polyprotein 3ABC (Mackay et al., 1998) together with antibody to the 2C protein FMD from the farm of

origin of the meat, the slaugh-

(Lubroth & Brown, 1995). Both can be produced as

ter house and the surrounding areas. If infected meat

pure reagents for an ELISA test by expression in a

should still enter in spite of these restrictions, an addi-

plasmid vector system. Because animals vaccinated

tional safeguard is the prohibition of feeding uncooked

with the dead FMD vaccine produce relatively little

meat or other swill to pigs. There are many examples of

antibody to the non-structural proteins (some may be

pigs being the first animals to be infected in an FMD

produced against the 3D protein), the tests will also dis-

outbreak. The Office International des Epizooties

tinguish animals which have been vaccinated – these

(OIE) provides guidelines for trade between countries

are positives only for the antibodies to the major

of different FMD status (OIE, 2001)

structural proteins (see above) – from recovered

The early detection of an FMD outbreak requires the

animals which have antibodies to both structural and

existence of an efficient veterinary service and a rapid

non-structural proteins. This would therefore also identify potential virus carrier animals. The problem still remains of how to detect the vaccinated animal which although by the time samples are received from abroad has had contact with live virus and thus become a carrier, but because of its protected status therefore an FMD outbreak could be well established. Nevertheless, the service can provide valuable additional support remains negative for antibodies to the non-structural and identify the most suitable vaccine for use to control proteins.

an outbreak.

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Following suspicion of an FMD outbreak the movement of all infected animals must be prevented and again at six months of age. Subsequently cattle should be vaccinated twice yearly, or even three times yearly local markets and abattoirs closed. The slaughter of all in areas where FMD is prevalent.

cattle, sheep, goats and pigs on infected premises is

The method used by different countries to control

practised by countries that do not routinely vaccinate

FMD is dependent on a number of factors. Countries

against FMD, and by many countries that do vaccinate,

free of FMD and protected on their borders by natural

as this will eliminate the source of virus emission and

barriers such as desert, sea or mountain ranges can

prevent the establishment of a nucleus of potential

maintain their status by strict import controls and can

carrier animals. Cattle, sheep and goats on surrounding

avoid the recurring cost of vaccination or the possibil-

farms that have already been vaccinated should be

ity of initiating an outbreak through the use of improp-

revaccinated with a vaccine antigenically related to

erly inactivated vaccine or escape of FMD virus from a

the outbreak strain. Pigs are generally also included

vaccine production plant. In addition, should FMD

in emergency vaccination programmes. All previous

virus enter a non-vaccinating country it can usually be

movement of animals, animal products and other

immediately identified clinically because of the comparatively long incubation period of the virus, whereas FMD virus is highly contagious and can be identified by the appearance of the first clinically infected animal. This will not only indicate the source of infection but also indicate which other farms may develop secondary outbreaks of infection. Finally, countries free of FMD which do not have a privileged international trading status. Subsequent movement on and off the infected premises must then be kept to a minimum and adequate facilities for cleaning and disinfection provided. Such consequences. Controllable precautions will identify and considerably reduce the chance of secondary spread. Windborne spread can be controlled by mass vaccination. The movement of nomadic people

spread of FMD virus prior to the slaughter of the with their animals, and of wild animals across international borders in Africa, the Middle and Far East, makes models developed to predict this windborne spread can disease regulations impossible to enforce. The airborne be used to indicate those farms most at risk and have spread of FMD virus cannot be controlled by legislation. Barrier vaccination can reduce the danger of FMD FMD in Europe.

entering a country or area and has been successful Vaccination against FMD is an effective method for in preventing exotic strains of FMD entering Europe protecting livestock against disease (p. 1016). Only through Turkey and Greece, and in allowing South inactivated vaccines are used and they are assessed by Africa and Zimbabwe to restrict vaccination to their their antigen content and the results of potency trials, borders and around game reserves. Countries which do ideally carried out in fully susceptible cattle (OIE,

not vaccinate or vaccinate against two or three strains (2000). An FMD vaccine is most effective against infection may use emergency vaccination to control an outbreak with the homologous strain from which the vaccine was prepared. However, a good vaccine must also protect animals and ring vaccinating around the infected area against closely related strains of FMD virus, with a monovalent vaccine specifically against the strain although its effectiveness will be reduced the more the exotic strain.

antigenic characteristics of the outbreak strain differ from the vaccine strain. It is therefore necessary to

Economic importance

identify the types and strains of FMD virus that pose the most significant threat. Nowhere has it been necessary to use more than a quadrivalent vaccine, although accurately. The direct costs of vaccination, slaughter of infected animals, movement restrictions and closure

include more than one strain of the same serotype. In of markets can be measured. The indirect local and addition, the characteristic variability of FMD virus has national costs, e.g. loss of potential export markets, may rendered some vaccine strains no longer effective, and be the most significant and yet most uncertain cost. new ones must be introduced.

Assuming that a country wishes to prevent FMD Maternal antibody can interfere with the develop- remaining or becoming endemic, two options are avail- ment of active immunity in young animals. It is there- able. Either all cattle (and possibly sheep, goats and fore recommended that if calves of immune dams are pigs) are routinely vaccinated or no vaccination is vaccinated in their first three months of life they should carried out and outbreaks are controlled by slaughter be vaccinated twice more at four and five and possibly as they occur. Which policy is the most economic can

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be assessed by critical point analysis or by estimating occurred. Rinderpest has however continued to persist

the critical point at which the cost of one policy equals in Asia and the Indian subcontinent and is still present the cost of the other policy. Costs that must be considered are the cost of vaccine and its administration or its outbreaks occur in the Near and Middle East. Rinder-storage as a strategic reserve. If it is also assumed that pest was introduced into Africa in the early 1800s and neither policy will eliminate the possibility of FMD was responsible for a massive pandemic between 1889 outbreaks, the cost of controlling an outbreak must be and 1897. Africa has not been free of rinderpest since, assessed and multiplied by the estimated total number although it has largely been confined to countries north of outbreaks.

of the Tropic of Capricorn. An international vaccination The cost of an FMD outbreak must include the cost programme carried out between 1962 and 1975 almost of controlling the outbreak, including ring vaccination, eradicated the disease (Scott, 1985). A second interna-the cost of slaughtered animals, the loss of production

tional vaccination programme, commencing in the mid- and the interruption of domestic and international 1980s, has reduced the incidence in West Africa to a trade. The international trading status of a country that level where eradication may be achieved (Rossiter, vaccinates already will be considerably less affected 1996). Extensive outbreaks have, however, continued in than that of a country that does not vaccinate. Similarly, eastern Africa where vaccination coverage has been the status of a country that does not use routine FMD hampered by civil unrest. The disease is still endemic in vaccine but vaccinates in order to control an FMD out- the horn of Africa and southern Sudan.

break may be affected by lengthy trade restrictions with other non-vaccinating countries following the cessation

Aetiology

of vaccination (OIE, 2001).

The difficulty in assessing these uncertain costs may

The causal agent of rinderpest is a paramyxovirus of the be illustrated by an analysis of the cost of annual vac- genus morbillivirus. The other members of the morbil-

cination (policy A) and the cost of a stamping out with
livirus genus are measles, canine distemper, phocine dis-
ring vaccination (policy B) carried out in the Federal
temper (Osterhaus & Vedder 1988; Mahy et al., 1988; Republic of Germany
(Lorenz, 1987). The average
Kennedy et al., 1988) and peste des petit ruminants
annual cost of policy A was estimated to be between 52
(PPR) to which rinderpest virus is antigenically related.
and 286 million DM, while the average annual cost of
Recently morbilliviruses have been isolated from dis-
policy B was between 2.5 and 321 or more million DM.
eased marine mammals (Barrett et al., 1993) and a
The wide range reflected the number of assumed FMD
morbillivirus-like agent caused fatal respiratory disease
outbreaks that could occur under each regime. The
in man and horses in Australia. The morbilliviruses are
estimates considered most likely, however, were
pleomorphic, enveloped, helical particles of between
between 183 and 227 million DM for policy A and
150–300 nm diameter and contain a non-segmented
between 47 and 61 million DM for policy B.

negative-strand RNA genome. This codes for six structural proteins and possibly one non-structural protein. Any scenario for assessing the cost of FMD control must ultimately assume the existence of an efficient veterinary service, capable of diagnosing the disease protein (N), which surrounds the genomic RNA, a large polymerase protein (L) and a small polymerase-associated protein (P), a matrix protein (M) associated with the virus envelope and two envelope glycoproteins, the haemagglutinin (H) and fusion protein (F). The P gene shows significant homology between members of the morbillivirus group (Barrett & Underwood, 1985).

(d) Rinderpest

There is only one serotype of rinderpest virus and rinderpest virus can be distinguished from PPR virus in reciprocal cross-neutralization tests. Alternatively, the

two viruses may be distinguished by comparing their protein patterns in polyacrylamide gels as the N pro-

The State Veterinary Service in the UK was brought

teins have been shown to have markedly different

into being specifically to deal with rinderpest or cattle

molecular weights (Diallo et al., 1987). They can also be plague. In the eighteenth and nineteenth century dev-distinguished by nucleic acid hybridization using cDNA

astating outbreaks of rinderpest were responsible for

probes to the H gene (Diallo et al., 1989) and by the

millions of deaths in cattle in Europe. By 1930, with the

reverse transcription-polymerase chain reaction (PT-

exception of parts of Turkey, Europe was free of the

PCR) (Forsyth & Barrett, 1995). It is now also possible

disease and since that time only small outbreaks have

to distinguish the two diseases in recovered animals by

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means of a competitive enzyme-linked immunosorbent

from two to five days. The virus is shed in all secretions

assay (ELISA) based on the use of monoclonal anti-

and excretions, the peak of virus production being

bodies (Mabs) to the haemagglutinin of each virus

during the prodromal fever but continuing after the
(Anderson et al., 1991).

appearance of erosive lesions. Virus levels fall as anti-
The virus is sensitive to lipid solvents, is relatively
body begins to be produced with viraemia ceasing
heat sensitive and is unstable at low pH. It is also labile
before the disappearance of the virus from the tissues,
when exposed to light and survives best at low or high
about 14 days after the onset of fever. Viraemia lasts on
relative humidities but is rapidly destroyed when the
the average about six days but there is considerable
relative humidity is between 40–60 per cent. Infectivity
variation between strains of virus. Viraemia can occur
is lost when it is suspended in glycerol or water but it
following exposure to some strains that lasts four to six
is stable in 0.86 per cent sodium chloride at low tem-
days without the development of lesions.

peratures with the loss in infectivity rising exponentially
with temperature. The use of molar concentrations of

Clinical signs

magnesium sulphate improves the thermostability of

the virus.

The clinical signs of rinderpest in cattle and other natural hosts are essentially the same but show wide variations in severity depending on the strain involved,

Species susceptible

and the resistance of the animal, natural or acquired.

Rinderpest is potentially infective for all members of

The disease may be hyperacute, acute, subacute or the order Artiodactyla (Scott, 1964) but in particular chronic (Plowright, 1968). The typical acute disease can infects members of the families Bovidae, Suidae be divided into four phases: incubation, prodromal, and Cervidae (Plowright, 1968). Of these cattle, water mucosal and convalescent.

buffalo, Cape buffalo (Syncerus caffer) and yak are most susceptible. The disease occurs in sheep and goats

Incubation phase

and, in India, also in pigs. It also affects camels. It has been recorded in a large number of wildlife species in

Africa including eland (Taurotragus oryx), lesser kudu Prodromal phase: This is characterized by a sudden

(*Tragelaphus imberis*), giraffe (*Giraffa camelopardis*) onset in fever reaching a peak on the second or third

and warthog (*Phacochoerus aethiopicus*). In 1994 the day after onset. It is accompanied by depression or rest-disease caused significant mortality in buffalo, lesser lessness, loss in appetite and a fall in milk yield in cows. kudu and eland in the Tsavo National Park in Kenya The visible mucous membranes are congested, the (Barrett et al., 1998).

muzzle is dry and there may be the beginning of a serous discharge from the eyes and nostrils. There is tachycardia and accelerated respirations, ruminal stasis

Transmission

and constipation. This phase lasts about three days with Rinderpest is spread by direct contact between infected lesions appearing between two to five days following and susceptible animals, by the inhalation of virus-the onset of pyrexia.

containing aerosols or by ingestion of infected secretions and excretions. On rare occasions it has spread through

Mucosal phase: The first lesions comprise small foci of indirect contact with contaminated fodder and water.

necrosis, superficial erosion and capillary haemorrhage

Pigs have been infected through eating meat from carcasses in the mucosae of the mouth cavity, which are particularly noticeable on the lower gum and tips of the buccal papillae. They extend to involve the lips, upper gum, hard palate and ventral surface of the tongue. Similar lesions occur in the nasal, vulval and preputial mucosae

Pathogenesis

where they may occur earlier than in the oral cavity.

Infection is through the upper respiratory tract with

The lesions extend and fuse to produce extensive areas of necrotic erosion with a characteristic fetid smell.

lymph nodes (Plowright, 1968). The virus is disseminated in the blood, where it is closely associated with

There may be excessive salivation and at this stage the lacrimal and nasal secretions become profuse and the mononuclear leucocytes, to all lymphoid tissue and purulent.

the mucosae of the alimentary and respiratory tracts.

Animals at this stage of the disease are very

The incubation period lasts from two to nine days

depressed and respirations are laboured, but pneumo-

and viraemia can be detected one to two days before

nia is rare. Diarrhoea appears usually between four and

the onset of pyrexia. The prodromal phase usually lasts

seven days of pyrexia and one to two days after the

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appearance of lesions. It is at first watery but later

virus. Retrospective diagnosis is obtained by the detec-

dysentery develops and it may contain pieces of intes-

tion of specific antibody (Scott et al., 1986).

tinal mucosa. Dehydration is rapid resulting in weak-

ness, prostration and death between six and twelve days

Antigen detection: Virus is present in the secretions and after the onset of pyrexia. The mortality rate is over 90

excretions within two days of the onset of fever. Spe-

per cent when susceptible animals are infected with

cific antigen can be detected from this time particularly

virulent strains.

in the lacrimal secretions but also later in swabs or

Mild forms of the disease in partially immune

material from lesions in the mouth, vagina or prepuce.

animals or following infection with less virulent strains

Lymph node biopsies may also be taken.

give rise to reduced general signs and less extensive

Early diagnosis is desirable and a rapid chromato-

mucosal lesions. However, these may be completely

graphic strip test based on Mab-labelled latex particles

absent with the only sign a transient diarrhoea. Even

is now available for pen-side diagnosis (Buning et al., this may be absent, and some strains of virus result in a

1999). The most sensitive method is, however, RT-PCR

complete spectrum of signs from the classical acute

and this can also be used with lacrimal secretions as well

febrile disease with extensive lesions and eventual

as material from mouth lesions and lymph node biop-

death to a form in which there is fever but no lesions

sies. This method also has the advantage of generating

although antigen can be detected in lacrimal secretions

DNA fragments whose sequence may help in defining

(Anderson et al., 1990; Forsyth et al., 2003).

the origin of the outbreak or differentiating the disease from PPR (Forsyth & Barrett, 1995). Specific antigen

Convalescent phase: Visible mouth lesions may heal

can also be detected by the less sensitive immunodiffu-

within as little as two to three days beginning from the

sion or counterimmunoelectrophoresis (CIEOP) tests

third to the fifth day after their appearance. Diarrhoea

in field laboratories. The ELISA is a very sensitive test

may persist for longer. Complete recovery from the

used in suitably equipped laboratories and is prefera-

acute form takes about four weeks depending on the

ble to the complement fixation test (Libeau et al., 1994).

environment and plane of nutrition.

Histochemical methods using fluorescein or enzyme-

conjugated antiserum may be used on smears, biopsy

material or tissue sections (Scott et al., 1986; Saliki et al., Pathology

1994).

The gross pathology has been described by Maurer et

Virus isolation: Virus may be isolated in tissue cultures al. (1956). The mucosae of the upper alimentary and

of primary or secondary calf or sheep kidney cells or respiratory tract are eroded and necrotic often being Vero cells. It may be isolated from swabs of lesions or coated with a mucopurulent exudate. Erosions, ulcers secretions, from the leucocyte fraction of blood col- and oedema occur in the abomasum, which may also be lected in EDTA or from lymphoid tissue collected at congested. Peyer's patches in the small intestine are post mortem. Specimens should be transported on ice haemorrhagic, oedematous and necrotic. The mucosal but glycerol should not be used as a transport medium, surface of the caecum, colon and rectum frequently has as it inactivates the virus.

characteristic haemorrhagic stripes due to the congestion of the capillaries. Erosion and ulcers also occur in

Antibody detection: The detection of a rising antibody the urogenital tract. The virus has a predilection for

titre in paired serum samples or in disease surveys may lymphoid tissues in which there is extensive necrosis be done using the virus neutralization test. This is suit- of the lymphocytes of the germinal centres, and the

able for small numbers of diagnostic samples but for appearance of multinucleated giant cells about eight days after infection. The appearance of cytoplasmic and numbers of samples the ELISA is the test of choice.

intranuclear inclusions has been described (Plowright, 1968). Other tests that are used include indirect immunofluorescence (CIEOP).

of the upper part of the alimentary tract shows syncytium formation and degenerative changes, which are followed by necrosis and detachment to form erosions

Differential diagnosis
and ulcers.

In cattle the one disease that cannot be distinguished from rinderpest without laboratory tests is bovine virus diarrhoea. In sheep and goats peste des petits ruminants is identical to rinderpest. Otherwise there are few

Diagnosis

Laboratory confirmation of rinderpest is based on the conditions that should be confused with the acute form

*detection of specific antigens and the isolation of the
of rinderpest. The mild forms in particular, where*

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*diarrhoea is the only clinical sign, are indistinguishable
Enzootic VS has a limited geographical location, within
from other enteric conditions.*

*which the appearance of disease is cyclical, apparently
related to season or rainfall. The majority of infections*

Control

*are inapparent and can occur in animals isolated in
cages or otherwise separated from other susceptible
Rinderpest spreads slowly in endemic countries where
species. Epizootic spread of VS is associated with simul-
it affects mainly immature animals. In these countries
taneous outbreaks over a wide area, although some
control is by vaccination annually of all immatures. In
herds within this area may remain unaffected. Of the
such countries attempting eradication, vaccination of
domesticated species, cattle are the most commonly
the entire cattle population, as well as sheep and goats
affected, followed by horses and then pigs; sheep and*

in those countries where the disease occurs in these goats never show clinical signs of VS. Few cases are species, for three to five years is practised. reported in young cattle, most cases appearing in In high risk countries adjacent to endemic regions or milking cows. Mouth lesions are most frequently those importing livestock from endemic countries quar- reported, although in some outbreaks only teat lesions antine and vaccination are combined (see p. 1013). are seen. Insects have been strongly implicated in the All rinderpest vaccines in use are live attenuated transmission of VS, but this has not yet been conclu- vaccines. Most countries use a tissue culture vaccine sively demonstrated. The cyclical appearance of VS sug- (Plowright, 1968). New lyophilization techniques have gests the existence of interepizootic reservoir hosts, but improved the thermostability of these vaccines (House these have also resisted identification. & Mariner, 1995). Goat adapted vaccine and lapinized A number of theories have been put forward to vaccine are still used in some countries.

explain these observations. It has been suggested that VSV circulates in feral pigs, elk, mule deer and antelope and possibly also in water birds and rodents such as

(e) Vesicular stomatitis

wood rats and deer mice. In support of this theory antibody against VSV has been found in all these species

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and, in some areas, prior to the onset of a VS epizootic in domesticated animals. It has also been suggested,

without much supporting evidence, that VSV could

Vesicular stomatitis (VS) is a vesicular disease of cattle,

persist in the soil and be circulated by arthropods or

horses and pigs which can also infect a large range of

even that VSV is primarily a plant virus.

wild animal species (see p. 366). The virus belongs to the

Studies using T1 oligonucleotide mapping of VSV-NJ

genus vesiculovirus, within the family Rhabdoviridae.

(Nicol, 1987) and nucleotide sequencing of VSV-NJ and

There are two serologically distinct types of VS virus

VSV-IND (Rodriguez, 1999) have helped to identify

(VSV): New Jersey (NJ) and Indiana (IND). The IND

the origin of VS epizootics in the USA. By precisely type can be further subdivided into Indiana 1 (Indiana characterizing the outbreak strains it has been shown strain), Indiana 2 (Cocal and Argentina strains) and that a VS epizootic is not caused by the simultaneous Indiana 3 (Alagoas and Brazil strains). In common with eruption of many strains of VSV within the United all other rhabdoviruses VSV is a single-stranded RNA States but the rapid spread of a single strain north from virus, the RNA being arranged in an enveloped helical the enzootic region of Mexico. A correlation has been nucleocapsid. The intact virus is bullet shaped, measuring 180 nm by 75 nm, and is covered with 10 nm spikes. wind direction from VS infected areas, indicating a possible involvement of insect vectors carried by the pre-primary cells and continuous cell lines, and laboratory vailing wind.

animals such as mice, rats, ferrets, guinea pigs, hamsters VSV-IND appears to have different epidemiological

and chick embryos. Humans are susceptible to VS, the characteristics from VSV-NJ, and its spread is less associated with clinical disease and has consequently vomiting, headaches and occasionally vesicles on the attracted less attention. It is also apparent that the mucosa of the mouth and throat. No deaths have been behaviour of VSV need not remain consistent; its mode reported, and the disease rarely lasts more than a week. of transmission, for instance, can vary even during epizootics.

Epidemiology

Distribution

The behaviour of VS in the field has been well documented but in spite of detailed observations there are VSV is restricted to North, Central and South America many aspects of its epidemiology which are still unclear. and the Caribbean islands. The disease was transported

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with horses from America to South Africa in 1884 and tinuation of the North American 1982/83 epizootic

1887, and to France in 1915, but did not persist in either through the winter.

country. The first report of VS in N. America was in

Transmission of VS can also occur by direct contact

horses during the American Civil War, and there were

between infected and susceptible cattle, this being fre-

further reports in 1889, 1904 and 1907. In South

quently associated with subclinical infections. An asso-

America VS was first diagnosed in Argentina in 1939,

ciation has been made between the feeding of abrasive

in Venezuela in 1941 and in Colombia in 1943.

feeds, which compromise the integrity of the buccal

Indiana 1 and New Jersey strains of VSV are enzootic

mucous membranes, and the spread of VS.

in southern Mexico and Central America, but a char-

acteristic of both is their periodic movement out of the

enzootic areas to cause epizootics in the southern

Clinical signs

United States and northern South America. VSV-IND

Vesicular stomatitis in cattle may be an inapparent, mild

has spread as far north as the United States–Canadian

or severe disease, animals over nine months of age border. These seasonal epizootics occur typically at the being most commonly affected. Following an incubation period of two to three days, a usually mild fever rainy season, and usually finish in the temperate regions develops accompanied by depression, lameness and at the onset of the frosts. The 1982 VSV-NJ epizootic excessive salivation. The fever reduces as vesicles did not, however, follow this cycle and persisted develop on the coronary bands of the feet, or in the through the winter. Epizootics which spread into the mouth or on the teats; rarely are vesicles seen on more mid-western and western states of North America tend than one of these sites. In severe cases over 50 per cent to occur at intervals of approximately five to ten years, of the tongue epithelium may be affected and the while major epizootics have been occurring every 30 resultant difficulty in eating can cause dramatic weight years. The predominant serotype causing vesicular loss. Milk yield is depressed. True vesicles may fail to

disease in the United States is VSV-NJ, and it is endemic develop, lesions appearing as crusts or ulcers. Recovery in at least one site in the USA – Ossabaw Island, is usually rapid, although milk yield frequently fails to Georgia. Vesicular stomatitis has not been reported in recover during the remaining lactation and secondary the New England area, eastern Canada or Alaska.

mastitis may be a problem. Some animals fail to recover fully and remain in poor condition. The lesions pro-

Transmission

duced by vesicular stomatitis virus in cattle are clinically indistinguishable from lesions of foot-and-mouth

The epidemiological characteristics of VS suggest that disease.

the disease is predominantly spread by insects. *Culex*

and *Aedes* species of mosquitoes, *Phlebotomine* sandflies, *Culicoides*, *Simulium* blackflies, *Musca* species, *Pathogenesis*

Hippelates eye gnats and *Anthomyidae* have all been implicated in the transmission of VS. Transovarial trans-

Vesicular stomatitis virus enters the animal through a mission of VSV has been shown in the sandfly *Lut-*

skin or mucosal abrasion or it may be inoculated by
zomyia trapidoi and biological transmission of VSV-NJ
an infected insect bite. Aerosol infection has been
by Simulium sp. (Mead & Maré, 1999). However, the reported in humans and
may also occur in cattle. A low
very low or possibly absent viraemia associated with VS
titre viraemia has been detected in some experimen-
rules out traditional concepts of vector transmission.
tally infected animals between 11 and 56 hours after
The vesicular lesions of clinical VS are rich in virus and
infection, but even this must be considered a rare event.
could provide a potent source for insect infection, but
Replication of virus occurs at the site of infection in the
this fails to explain the transmission of VS between sub-
prickle cells of the Malpighian layer, but there is no
clinically infected animals. Nevertheless, the climatic
information on subsequent sites of virus replication. As
conditions which predispose to the spread of VS and the
the cells degenerate and transudate from the blood
more frequent appearance of VS in animals at pasture
stream accumulates, vesicles develop and the animal

strongly implicate the involvement of insects. In addition, the characteristic termination of a VS epizootic becomes febrile. It is not clear why lesions are rarely generalized, but are usually restricted to the teats, with the onset of subzero night time temperatures is mouth or feet. Immunosuppressed or overcrowded typical of many vectorborne diseases. Experimentally animals are more likely to develop lesions. VSV does inoculated sandflies and *Aedes aegypt* have been shown not cross the placenta and there are no reports of calves to transmit VSV to vertebrate hosts (Letchworth et al., from infected dams being viraemic or having pre-1999). The virus does not appear to be totally dependent on insect transmission, as evidenced by the once been recovered from an aborted fetus.

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Diagnosis

ity that animals can remain carriers of VSV. It has been observed that recovered cattle have appeared to spread
Diagnosis of VS is by clinical examination of affected VSV to susceptible animals, and that some of these animals and the demonstration of the presence of VSV

recovered cattle have again developed vesicles, usually, or of a rising antibody titre to VSV. Laboratory confirmation of VS is essential in order to distinguish the area of infection. These recurrent lesions have usually disease from foot-and-mouth disease.

occurred within 48 hours of moving the recovered VSV has a characteristic appearance under the electron microscope. The virus will also grow on a wide range of primary and continuous cell lines, on the stress. Although it has not been possible to isolate virus from clinically normal animals recovered from VS, the fertile eggs and in many laboratory mammals. The persistence of high levels of neutralizing antibodies in Vero-M (green monkey) cell line is used extensively for these animals does suggest the presence of a continuous antigenic stimulation.

virus neutralization test. The complement fixation test

*An additional problem is the high proportion of sub-
and fluorescent antibody test are also used in the diag-
clinical cases during epizootics and the possibility that
nosis of VS, although indirect sandwich ELISA is now
VS could circulate in an area unobserved.*

considered the most economic and rapid (OIE, 2000).

Measures designed to eliminate virus shed in the

*Details given for the submission of samples for FMD
saliva of infected cattle do reduce transmission; 10 per
diagnosis (see p. 704) apply also to the submission of
cent household bleach used on utensils and dairy equip-
samples for VS diagnosis, although VSV is much less
ment, and for washing hands, has proved useful. In addi-
susceptible to pH outside the range of 7.2 to 8.0. As a
tion, replacement of hard or abrasive feed with a softer
differential diagnosis is usually required between FMD
alternative reduces mouth lesions and thereby intro-
and VS, the more stringent requirements of FMD virus
duction of the virus.*

should be followed.

Within the USA, animals infected with VSV are
Virus neutralization and liquid phase blocking
quarantined for at least 30 days after all signs of VS
ELISA antibodies may be detected 96 hours after
have disappeared. Countries free of VS are usually con-
experimental inoculation, and they reach a peak by day
siderably more stringent and will not import any animal
12. These antibodies persist for many years and are
with virus neutralizing antibodies to VS. This precau-
valuable in showing evidence of previous infection.
tion is intended to exclude the possibility of importing
Their persistence has led to the suggestion that the virus
a persistently infected animal.
itself may persist in an animal long after recovery from
Living and formalin-inactivated vaccines have been
the disease, although it may remain in a defective, non-
prepared against VSV-NJ (see p. 1018). The dead
infectious form. Complement fixing antibodies recede
vaccine reduces the incidence of overt disease, while the
three to six months after infection. A four-fold or
live vaccine, given by intramuscular inoculation, gives a

greater increase in VSV neutralizing antibodies
better protection and does not spread to in-contact sus-
between early infection and convalescent serum is diag-
ceptible animals. A recombinant vaccine has also been
nostic of infection. The presence of neutralizing anti-
developed by inserting the gene for the VSV glycopro-
bodies does not appear always to prevent reinfection or
tein into the genome of vaccinia virus. This vector
the development of clinical signs.

vaccine against VS is reported to be highly effective, but
Prior to the establishment of suitable laboratory
has not been used in the field due to its potential danger
tests, VS was diagnosed by scarifying infective material
to humans (Letchworth et al., 1999).

into the snout of swine and the tongues of cattle and
horses, and by intramuscular inoculation of cattle. VSV
produced lesions on pigs, cattle and horses, but not in
Economic importance

cattle when given by intramuscular injection. FMD
It is difficult to assess the full economic importance of
virus produced disease in swine and cattle, but not

VS because of the effect the presence of disease has on horses, and vesicular exanthema virus affected only export markets. Direct losses due to disease can be swine, although occasionally lesions were also produced severe, particularly in high yielding dairy herds, and on the tongues of horses.

have been calculated to have been between \$97 and \$253 per clinical case during the 1982 epizootic in the United States (quoted by Monath et al., 1986). Losses

Control

due to reduced growth rates in beef cattle in South Measures designed to control VS reflect the poor America are also reported to be significant. Consequence- understanding of its epidemiology, notably the possible-quent movement restrictions and the closure of local

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markets can also cause considerable loss. Nevertheless, natural infection is probably confined to cattle. Laboratory workers exposed to infection have not seroconverted-epizootic areas from countries free of VS that causes

verted. The virus is heat labile and is killed by milk
the most significant economic losses.

pasteurization.

The disease appears to be expressed in few clinical
signs, although in some herds in the USA and possibly
in Europe disease can be infrequently expressed. This

(f) Bovine immunodeficiency

lack of ability to identify the organism or detect it con-
sistently serologically means that its economic impor-

virus (BIV)

tance cannot be properly assessed. In many instances

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infection appear to be concomitant with another viral
disease, such as enzootic bovine leukosis (EBL) (see
p. 693).

In infected animals the infection follows a progres-

The virus

sion similar to that seen with HIV. Thus the animals

The bovine immunodeficiency virus (BIV) is a

have a persistent and chronic lifelong infection. The

lentivirus first isolated as strain R29 in the USA from a

period of development for natural infection is not
cow with lymphoproliferative lesions. The organism has
known, although following experimental infection
also been known as bovine immunodeficiency-like
cattle become seropositive in two to four weeks. Trans-
virus, bovine visna virus and bovine lentivirus. The virus
mission is thought to be horizontal, although proviral
is difficult to isolate, thus there have only been two sub-
DNA has been detected in the blood and semen of bulls
sequent isolates in the USA and also two in Japan. The
experimentally infected (Gradil et al., 1999). However, virus has a similar
morphology to other lentiviruses

transplacental BIV infection has been demonstrated
with a viral envelope of structural gene products and a
between seropositive dams and their calves (Scholl
core of viral nucleic acid, p26 capsid, p16 nucleocapsid,
et al., 2000).

reverse transcriptase and integrase proteins. The RNA
genome is 1842 nucleotides in length. While the BIV
Clinical signs
contains the same non-structural accessory genes as

HIV, it lacks the addition gene *nef* found in primate
There is much debate as to the severity of the disease
lentiviruses. A genetically related virus to BIV,
in cattle. It does appear that in many instances the virus
Jembrana disease virus, has been isolated but is
causes no detectable signs, although in a few herds it
restricted to Indonesia (see p. 766) and causes a severe
does appear to cause major problems. It has been diffi-
disease in Banteng cattle (*Bos javanicus*) but a milder cult to define whether the
virus caused problems on its
problem in *Bos taurus*.

own or whether infection predisposes animals to oppor-
tunistic infections by other cattle disease pathogens.

There is still argument as to whether or not BIV causes

Distribution

true immunodeficiency and this is why it is often

Although the virus has been rarely isolated there is
referred to as bovine immunodeficiency-like virus.

serological evidence to suggest that it is distributed

The main signs in severely affected animals include
worldwide. While it is said to be more prevalent in

loss of weight and poor condition, unthriftiness leading to emaciation in some animals. There is a variable degree of lymphadenopathy, which may be localized or generalized. Lameness or stiffness may be generalized or result in one or more legs showing lameness. It is also reported to be present serologically in New Zealand, Australia, Canada, China, Korea and Pakistan. There are nervous signs including changes in behaviour, nervousness, hyperaesthesia and also aggression in some animals, but other cases exhibit depression, dullness and lethargy. Some animals become ataxic and will fall over. Milk yield is depressed or even absent. A few animals may show prolapse of the third

and 5.0 per cent in beef cattle (Scobie et al., 2001). While eyelid. sheep, goats and rabbits have all been infected experimentally, there is no evidence to suggest that this problems in calves and older animals such as diarrhoea, happens naturally. There is nothing to suggest that BIV pneumonia and abortion will result in particularly is of any significant risk to man. It would appear that severe disease in some individuals and not in others.

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Pathology

Jeggo, M.H. (1986) A review of the immune response to bluetongue virus *Revue Scientifique et Technique d'Office International des Epizooties*, 5, 357–62.

and severity. In some animals there is lymph node

Luedke, A.J., Jochim, M.M. & Barber, T.L. (1982) Serologic hyperplasia and mild perivascular cuffing in the brain.

and virologic responses of a Hereford bull persistently

The most common finding at post mortem examination

infected with bluetongue virus for eleven years. Proceedings has involved body lymph nodes which become or

of the American Association of Veterinary Laboratory
contain large spheroidal haemal nodes, black in colour.

Diagnostics, **25**, 115–34.

These haemal nodes vary considerably between

Mertens, P.P.C., Burroughs, J.N. & Anderson, J. (1987) Purified infected animals
in size, distribution and number

cation and properties of virus particles, infectious subviral
particles, and cores of bluetongue virus serotypes 1 and 4.

(Munro et al., 1998).

Virology, **157**, 375–86.

Osborn, B.I., Huffman, E.M., Sawyer, I.N. & Hird, D. (1986) Diagnosis

Economics of bluetongue in the United States. In *Arbovirus Research in
Australia, Proceedings 4th Symposium, Brisbane, Diagnosis is difficult because
the virus is not easily*

May 1986, pp. 245–7.

isolated. Thus virus isolation in tissue culture is of

Verwoerd, D.W., Els, H.J., DeVilliers, E.M. & Huisman, H.

limited use as it is of low sensitivity and is also usually

(1972) The structure of the bluetongue virus capsid. Journal complicated by the
presence of other viral agents.

of *Virology*, **10**, 783–94.

However, polymerase chain reaction (PCR) techniques

have been successfully used to detect the presence of

proviral DNA. Very variable results have also been

Enzootic bovine leukosis

obtained by serological methods. The unreliability of

Burny, A., Brock, C., Chantrenne, H., Cleuter, Y., Dekegel, D., some assay methods has meant that there may have

Ghysdael, J., Kettman, R., Leclercq, M., Leunen, J.,

been false positive results during testing in the past. At

Mammerickx, M. & Portetelle, D. (1980) Bovine leukemia

present, most diagnostic methods involving serology

virus. In *Molecular Biology and Epidemiology in Viral*

use indirect immunofluorescent antibody (IFA),

Oncology (ed. by G. Klein), pp. 231–89. Raven Press, New enzyme linked immunosorbent assay (ELISA) or

York.

western blot. The tests most likely to be of use in screen-

Burny, A., Bruck, C., Cleuter, Y., Couez, D., Deschamps, J.,

ing large numbers of cattle would appear to be based

Ghysdael, J., Gregoire, D., Kettmann, R., Mammerickx, M.,

on ELISA and one currently in use is based on the TM

Marbaix, G. & Portetelle, D. (1985) Bovine leukaemia virus: region of BIV.

a tantalizing story. In *Viruses and Cancer* (ed. by P.W.J.

Rigby & N.M. Wilkie), pp. 197–216. Cambridge University

Press, Cambridge.

Treatment and control

Burny, A., Bruck, C., Cleuter, Y., Couez, D., Deschamps, J.,

Gregoire, D., Ghysdael, J., Kettman, R., Mammerickx, M.,

There is no available treatment for BIV infection. The

Marbaix, G. & Portetelle, D. (1985) Bovine leukaemia virus best that can be done at present is to ensure full and

and enzootic bovine leukosis. Onderstepoort Journal of

adequate treatment of all secondary infections. In some

Veterinary Research, 52, 133–44.

herds where clinical signs have been present it has been

Burny, A., Bruck, C., Cleuter, Y., Couez, D., Gregoire, D.,

the result of management deficiencies such as poor

Kettmann, R., Mammerickx, M., Marbaix, G., Portetelle, D.

nutrition or poor housing. In some herds where those

& Willems, L. (1986) Bovine leukemia virus as an inducer

deficiencies have been improved, the clinical signs have

of bovine leukemia. In Animal Models of Retrovirus Infec-

been reduced or become latent.

tion and their Relationship to AIDS (ed. by L.A. Saizman), pp. 107–19. Academic Press, New York.

Callebaut, I., Burny, A., Krchnak, V., Grasse-Masse, H.,

References and further reading

Wathet, B. & Portetelle, D. (1991) Use of synthetic peptides to map sequential epitopes recognised by monoclonal

Bluetongue

antibodies to bovine leukaemia virus external glycoprotein.

Virology, **185**, 48–55.

Bowen, R.A., Howard, T.H. & Pickett, B.W. (1985) Seminal

Callebaut, I., Portetelle, D., Burny, A. & Mornon, J.-P. (1994) shedding of bluetongue virus in experimentally infected

Identification of functional sites on bovine leukaemia virus

bulls. In *Bluetongue and Related Orbiviruses* (ed. by T.L.

glycoproteins using structural and immunological data.

Barber & M.M. Jochim), *Progress in Clinical and Biologi-*

European Journal of Biochemistry, **222**, 405–14.

cal Research, Vol. 178, pp. 91–6. Alan R. Liss Inc., New

Evermann, J.F. (1983) *Bovine leukemia virus infection*.

York.

Modern Veterinary Practice, **64**, 103–105.

Gorman, B.M., Taylor, J. & Walker, P.J. (1983) Orbiviruses. In Ferrer, J.F. (1980) *Bovine Lymphosarcoma. Advances in The Reoviridae* (ed. by W.K. Joklik), pp. 287–357. Plenum, *Veterinary Science and Comparative Medicine*, Vol. 24.

New York.

Academic Press, New York.

Viral Diseases • 715

Ghysdael, J., Bruck, C., Kettman, R. & Burny, A. (1984)

Rinderpest

Bovine leukemia virus. In Current Topics in Microbiology and Immunology (ed. by M. Cooper et al.), Vol. 112, pp.

Anderson, E.C., Hassan, A., Burrett, T. & Anderson, J. (1990) 1–19, Springer-Verlag, Berlin.

Observations on the pathogenicity for sheep and goats and

Miller, J.M., Miller, L.D., Olson, C. & Gillette, K.G. (1969) the transmissibility of the strains of virus isolated during the Virus-like particles in phytohaemagglutinin-stimulated lym-rinderpest outbreak in Sri Lanka in 1987. Veterinary Micro-phocyte cultures with reference to bovine lymphosarcoma.

biology, 21, 309–18.

Journal of the National Cancer Institute, 43, 1297–305.

Anderson, J., MaKay, J.A. & Butcher, R.N. (1991) The use of Parodi, A.L. (1986) Enzootic bovine leukosis. Its aetiology,

monoclonal antibodies in competitive ELISA for the detec-

epidemiology and principles of control. Pro Veterinario, i, tion of antibodies to rinderpest and peste des petits

1–4.

ruminants viruses. In The Sero-monitoring of Rinderpest

Schultz, R.D., Manning, T.O., Rhyan, J.C., Buxton, B.A.,

Throughout Africa, Phase One. Proceedings of the Final Panangala, V.S., Bause,

I.M. & Yang, W.C. (1986) Immuno-Research Co-ordination Meeting of the IAEA Rinderpest

logic and virologic studies on bovine leukosis. In Animal Control Projects, Ivory Coast, November, 1990. IAEA publication TECDOC-623.

AIDS (ed. by L.A. Saizman), pp. 301–23. Academic Press, Barrett, T. & Underwood, B. (1985) Comparison of messenger-RNAs induced in cells infected with each member of

the Morbillivirus group. Virology, 145, 195–9.

Foot-and-mouth disease

Barrett, T., Visser, I.K.G., Maemaev, L., Goatley, L., van Bresse, M.-F. & Osterhaus, A.D.M.E. (1993) Dolphin Kitching, R.P. (1998) A recent history of foot-and-mouth and porpoise morbilliviruses are genetically distinct from disease. Journal of Comparative Pathology, 118, 89–108.

phocine distemper virus. Virology, 193, 1010–12.

Kitching, R.P. & Donaldson, A.I. (1987) Collection and transportation of specimens for vesicular virus investigation.

Wambua, J., Mwanjia, J. & Rossiter, P. (1998) Rediscovery Revue Scientifique et Techniques d'Office International des of the second African lineage of rinderpest virus: its epi-

Epizootics, **6**, 263–72.

demioological significance. *Veterinary Record*, **142**, 669–71.

Kitching, R.P., Knowles, N.J., Samuel, A.R. & Donaldson, A.I.

Buning, A., Bellamy, K., Talbot, D. & Anderson, J. (1999) A (1989) Development of foot-and-mouth disease virus strain

rapid chromatographic strip test for pen-side diagnosis of

characterisation – a review. *Tropical Animal Health and*

rinderpest virus. *Journal of Virological Methods*, **81**, 143–54.

Production, **21**, 153–66.

Diallo, A., Barrett, T., Lefevre, P.C. & Taylor, W.P. (1987) Com-Kitching, R.P., Rendel, R. & Ferris, N.P. (1988) Rapid correla-parison of proteins induced in cells infected with rinderpest

tion between field isolates and vaccine strains of foot-and-

and peste des petits ruminants viruses. *Journal of General mouth disease virus. Vaccine*, **6**, 403–408.

Virology, **68**, 2033–8.

Knowles, N.J., Samuel, A.R., Davies, P.R., Kitching, R.P. & Diallo, A., Barrett, T., Barbron, M., Subbarao, S.M. & Taylor, Donaldson, A.I. (2001) Outbreak of foot-and-mouth

W.D. (1989) Differentiation of rinderpest and peste de

disease virus serotype O in the UK caused by a pandemic

petits ruminants viruses using specific cDNA clones. *Journal strain. Veterinary Record*, **148**, 258–9.

of Virological Methods, **23**, 127–436.

Lorenz, R.J. (1987) Guide to the economic evaluation of FMD

Forsyth, M.A. & Barrett, T. (1995) Evaluation of polymerase vaccination programmes. Report of the 27th Session of the

chain reaction for the detection and characterisation of

European Commission for the control of FMD. Appendix

rinderpest and peste des petits ruminants viruses for epi-

12, pp. 121–36. Rome, 21–24 April 1987.

demiological studies. Virus Research, 39, 151–63.

Lubroth, J. & Brown, F. (1995). Identification of native foot-Forsyth, M.A., Parida, S., Alexandersen, S., Belsham, G.T. & and-mouth disease virus non-structural protein 2C as a

Barrett, T. (2003) Rinderpest virus lineage differentiation

serological indication to differentiate infected from

using RT-PCR and SNAP-ELISA. Journal of Virological

vaccinated livestock. Research in Veterinary Science, 59, Methods, 107, 29–36.

70–8.

House, J.A. & Mariner, J.C. (1995) Stabilisation of rinderpest Mackay, D.K.J., Forsyth, M.A., Davies, P.R., Berlinzani, A.,

vaccine by modification of the lyophilisation process. New

Belsham, G.J., Flint, M. & Ryan M.D. (1998) Differentiat-

approaches to stabilisation of vaccine potency, WHO Head-

ing infection from vaccination in foot-and-mouth disease

quarters, Geneva, 29–31 May 1995 (ed. by F. Brown).

using a panel of recombinant, non-structural proteins in

Karger, Basel. Developments in Biological Standardisation

ELISA. Vaccine, **16**, 446–59.

(1996) **87**, 235–44.

OIE (2000) *OIE Manual of Standards for Diagnostic Tests and* Kennedy, S.,
Smyth, J.A., McCullough, S.J., Allan, G.M.,

Vaccines, 4th ed., pp. 77–92. Office International des McNeilly, F. & McQuaid,
S. (1988) *Confirmation of cause of Épizooties*, Paris.

recent seal deaths. *Nature*, **335**, 404.

OIE (2001) *International Animal Health Code*, pp. 63–75.

Libeau, G., Diallo, A., Colas, F. & Guerre, L. (1994) *Rapid dif-Office*
International des Épizooties, Paris.

ferential diagnosis of rinderpest and PPR using immuno-

Samuel, A.R., Knowles, N.J., Kitching, R.P. & Hafez, S.M.

capture ELISA. Veterinary Record, **134**, 300–304.

(1997) *Molecular analysis of foot-and-mouth disease type*

Mahy, B.W.J., Barrett, T., Evans, S., Anderson, E.C. & Bostock, O viruses
isolated in Saudi Arabia between 1983 and 1995.

C.J. (1988) *Characterization of a seal morbillivirus. Nature, Epidemiology and*
Infection, **119**, 381–9.

336, 115.

716 • Chapter 43

Maurer, F.D., Jones, T.C., Easterday, B. & Detray, D.E. (1956) Monath, T.P.,
Webb, P.A., Francy, O.B. & Walton, T.E. (1986) *Pathology of rinderpest. Journal*

of the American Veterinary The epidemiology of vesicular stomatitis – new data, old

Medical Association, **127**, 512–14.

puzzles. In *Proceedings of the 4th Symposium Arbovirus*

Osterhaus, A.D.M.E. & Vedder, E.J. (1988) Identification of Research in Australia, CSIRO pp. 193–8. Brisbane, May 1986.

virus causing recent seal deaths. *Nature*, **335**, 20.

Nicol, S.T. (1987) *Molecular epizootiology and evolution of*

Plowright, W. (1968) Rinderpest Virus, *Virology Monographs vesicular stomatitis Journal of Virology*, **61**, 1029–36.

No. 3. Springer-Verlag, Berlin.

OIE (2000) *OIE Manual of Standards for Diagnostic Tests and Rossiter, P. (1996) Epidemiological and clinical features of*

Vaccines, 4th edn. Office International des Epizooties, Paris.

rinderpest in the 1990s. *FAO Animal Production and Health*

Rodriguez, L.L. (1999) *Genetic and epidemiological evidence*

Paper, **129**, 67–9.

indicates that vesicular stomatitis virus are not endemic in

Saliki, J.T., Brown, C.C., House, J.A. & Dubovi, E.J. (1994) Dif-northern Mexico and south western USA. *Annals of the*

ferential immunohistochemical staining of peste des petits

New York Academy of Science **916**.

ruminants and rinderpest antigens in formalin-fixed,

paraffin-embedded tissues using monoclonal and polyclonal

Bovine immunodeficiency virus

antibodies. *Journal of Veterinary Diagnostic Investigation*, **6**, 96–8.

Gradil, C.M., Watson, R.E., Renshaw, R.W., Gilbert, R.O. & Scott, G.R. (1964) Rinderpest. *Advances in Veterinary Science*, Dubovi, E.J. (1999) Detection of bovine immunodeficiency-9, 113–224.

like virus DNA in the blood and semen of experimentally

Scott, G.R. (1985) Rinderpest in the 1980s. *Progress in Veteri-infected bulls. Veterinary Microbiobogy*, **70**, 21–31.

nary *Microbiology and Immunology*, **1**, 145–74.

Munro, R., Lysons, R., Venables, C., Horigan, M., Jeffrey, M. & Scott, G.R., Taylor, W.P. & Rossiter, P.B. (1986) *Manual on the Dawson, M. (1998) Lymphadenopathy and non-suppurative*

Diagnosis of Rinderpest. Food and Agriculture Organiza-meningo-encephalitis in calves experimentally infected with

tion of the United Nations, Rome.

bovine immunodeficiency-like virus (FL112). *Journal of*

Comparative Pathology, **119**, 121–34.

Vesicular stomatitis

Scholl, D.T., Truax, R.E., Baptista, J.M., Ingawa, K., Orr, K.A., O'Reilly, K.L. & Jenny, B.F. (2000) Natural transplacental Letchworth, G.J., Rodriguez, L.C. & Barrera, J. del C

infection of dairy calves with bovine immunodeficiency

(1999) *Vesicular stomatitis. Veterinary Journal*, **157**, 239–

virus and estimation of effect on neonatal health. Preven-

60.

tive Veterinary Medicine, **43**, 239–52.

Mead, D.G. & Maré, C.J. (1999) *Vector competence of wild*

Scobie, L., Venables, C., Sayers, A.R., Weightman, S. & Jarrett, and colonized black flies to the Indiana and New Jersey

O. (2001) *Prevalence of bovine immunodeficiency virus*

serotypes of vesicular stomatitis virus. Annals of the New infection in cattle in Great Britain. Veterinary Record, **149**, York Academy of Science **916**.

pp. 459–60.

Chapter 44

Bacterial Conditions

A.H. Andrews and the late B.M. Williams

Anthrax

717

vegetative bacilli are not very resistant to environmen-

Bacillary haemoglobinuria

719

tal conditions or to physical and chemical agents,

Botulism

721

and are rapidly destroyed by putrefactive processes in

Clostridial myositis: blackleg and malignant oedema

723

unopened carcasses (Sterne, 1959). However, sporula-

Blackleg

723

tion occurs when carcasses are opened or when dis-

Malignant oedema

724

charges containing bacilli are exposed to air. Mature

Contagious bovine pyelonephritis

725

spores are extremely resistant to environmental condi-

Endocarditis

726

Haemorrhagic septicaemia

728

tions and certain disinfectants, and in soil they remain

Infectious necrotic hepatitis (black disease)

729

viable for many years.

Pericarditis

731

Anthrax has been reported from most if not all cattle

Tetanus

733

rearing countries of the world, but the incidence of

*Leptospirosis (other than *Leptospira hardjo* infection) 734*

disease is dependent on a number of factors, including

**Leptospira hardjo* infection (flabby bag)*

735

climate, soil, animal husbandry and disease control

**Haemophilus somnus* infection*

737

methods. Serious outbreaks of disease are more com-

Pyæmia

737

monly encountered in tropical and subtropical coun-

tries. In such areas, infection persists in the soil – spores

Anthrax

can survive for decades – and is a major source of infec-

tion. The disposal of animal carcasses also presents a

**Bacillus anthracis* infection in cattle causes a per-*

serious problem unless whole carcasses are removed.

acute or acute disease, which is characterized by a
Otherwise cattle and other animals readily come into
septicaemia and sudden or rapid death. The disease is
contact with the tissues, particularly bones, of animals
a zoonosis and is subject to official control measures in
that die from anthrax. This method of infection is very
a number of countries. Infection in man usually results
important in phosphorus deficient areas where cattle
in the cutaneous form of the disease. Inhalation
develop pica and chew bones in an attempt to remedy
producing 'wool sorter's disease' is very severe but
the deficiency.
uncommon.

In temperate countries sporadic outbreaks occur
involving single or a small number of animals, arising
from the ingestion of contaminated feedingstuffs.

Aetiology

Campbell (1969) reported that the vast majority of
Bacillus anthracis is a Gram-positive capsulated
anthrax outbreaks in cattle in England and Wales were
bacillus and its morphology, together with the staining

associated with compound feedingstuffs containing reaction of the capsula, is of diagnostic importance. meat and bone meal derived from imported materials When stained by Giemsa's, Wright's or similar stains, from Asia and South America, but he also emphasized the capsule stains a reddish mauve colour, is square that often vegetable protein became contaminated with ended and its outline is rather ragged or 'shaggy' (Plate anthrax organisms in the holds of ships that were being 44.1). A similar reaction is produced by methylene blue, or had been used for the transport of meat and bone but because of the variability in the content of its oximeal. Hugh-Jones & Hussaini (1975) confirmed that in dized products, azur A and B, which have an affinity for Great Britain, contaminated feedingstuffs were the the capsule, the staining reaction is less consistent and major source of infection, although infection was some- the staining of the capsule is a less intense mauve times derived from tannery effluent and soil at sites colour. The organism produces a lethal toxin that causes where the carcasses of animals had been buried some

death through shock and acute renal failure.

years previously.

The organism is a spore-forming bacillus, but mature

Infection is acquired through the ingestion of soil or

spores are not formed in the animal before death. The

effluent-contaminated fodder or contaminated com-

717

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pound feedingstuffs. Schlingham et al. (1956) have

carcass must be carried out. If anthrax cannot be elim-

demonstrated that clinical disease in cattle can be reg-

inated, then a blood sample should be taken from a

ularly produced by the oral administration of organisms

superficial blood vessel and a stained smear examined

in feed pellets. The spores penetrate the intact mucosa,

microscopically. It is more difficult to identify anthrax

or through small abrasions in the mucosa of the mouth

bacilli if animals have been treated with antibiotics. The

and pharynx, and are then transported to the local

procedures for dealing with suspected anthrax cases

lymph nodes where germination and multiplication

vary from country to country and those currently in occurs, followed by passage via the lymphatics into the force in Great Britain are discussed below.

bloodstream, leading to a septicaemia with an explosive Rapid decomposition of the carcass sets in soon after invasion of all body tissues.

death and in most instances rigor mortis is absent and dark tarry blood exudes from all the body orifices.

There is bloodstained fluid in all body cavities and

Signs

there are widespread haemorrhages throughout the

All ages of cattle are susceptible to infection. The

carcass, particularly on the parietal pleura and peri-

incubation period is thought to be one to two weeks,

toneum. Unclothed or poorly clotted blood oozes from

although in some incidents it would appear to be

the cut blood vessels and there is an intense inflamma- three to five days.

tion of the mucosa of the abomasum and both small and

At the beginning of an outbreak, the peracute form

large intestine. The spleen is almost invariably greatly

of the disease is more common, animals usually being enlarged with sometimes a rupture of the capsule, and found dead within a few hours of being seen in normal a dark semifluid pulp. The lesions in animals that have health. On the rare occasions that animals are seen been treated with antibiotics before death are similar ailing, fever, muscle tremors, dyspnoea, collapse and but much less spectacular.

terminal convulsions are the predominant signs, with death occurring in 1–4 hours. In the acute form, which

Diagnosis

runs a course of 24–48 hours, fever, depression, rapid and laboured respirations, diarrhoea or dysentery,

The diagnosis of anthrax is based on the demonstration haemorrhagic congestion of the visible mucous membranes of capsulated bacilli in peripheral blood and subsequent branes and in dairy cattle a sudden drop in milk yield confirmation by laboratory isolation and identification. are the main signs. Pregnant animals may abort.

In Great Britain and many other countries, anthrax is a Atypical signs have been recorded in calves receiv-

notifiable disease and the State Veterinary Service is
ing prophylactic levels of oxytetracycline or chlortetra-
responsible for confirmation.

cycline for the control of salmonellosis. In such animals,

There are numerous causes of sudden death in cattle,
a mild fever, bleeding from the nose and eyes and
including clostridial infections, hypomagnesaemia, lead
melaena were the only signs observed before death,
poisoning and lightning strike, all of which can be con-
which occurred 48–72 hours after onset (B.M. Williams,
firmed by a post-mortem examination and appropriate
unpublished data).

laboratory examinations. As emphasized earlier, such
procedures should not be undertaken until anthrax has
been eliminated.

Pathology

Because of the peracute/acute nature of the disease

Treatment and prevention

*in cattle, there is little opportunity for ante-mortem
laboratory examinations. It may be possible to detect*

Bacillus anthracis is sensitive to a number of antibiotics organisms in

appropriately stained smears of peripheral and treatment of animals during the early stages of blood, but sufficient numbers of organisms are only the disease is likely to be successful, although severely likely to be present during the later stages of the ailing animals are unlikely to recover. Greenough disease. A haematological examination may reveal a (1965) successfully treated animals with 5 megaunits leucocytosis and a shift to the left, but because of of penicillin alone or with streptomycin. The recommended dosage of penicillin is 10 000 units/kg body are not marked.

weight administered twice daily for at least three to five days. Streptomycin, in 4–5 g doses, should also be has died suddenly or after a very short illness, it is essential that anthrax be eliminated, to prevent contamination of the environment and ensure proper disposal of

treated with a combination of large doses of penicillin the carcass. Thus a careful evaluation of the circumstances and streptomycin twice daily, although oxytetracycline and chlortetracycline are the next antibiotics of choice.

Bacterial Conditions • 719

Bailey (1953) also recommends tetracycline for the where MBM can be legally used, it is essential to use treatment of anthrax. Anthrax antiserum administered sterilized meal and minerals in their finished feeds. intravenously is effective but the high cost and large volumes required makes its routine use impractical.

When anthrax has been confirmed in a herd, all

Bacillary haemoglobinuria

animals should be carefully observed at frequent intervals and any that are showing signs of ill health Bacillary haemoglobinuria or infectious ictero-haemo-isolated, their temperatures taken and, if elevated, globinuria of cattle was described by Roberts (1959) as immediately treated with antibiotic.

a rapidly fatal infectious disease, manifested clinically

In Great Britain and elsewhere, anthrax is a notifiable disease under the provisions of the Anthrax Order 1938 for the protection of both animal and human health. An owner or veterinary surgeon must report any suspicion of disease to a police constable, who informs the Local Authority, which then immediately imposes

Aetiology

The causal organism is Clostridium novyi type D, previously designated Cl. haemolyticum. Like other clostridia it is a soil-borne anaerobe, the spores of which must be detained and the skin must not be incised other

are resistant to environmental factors and may survive than for the removal of a blood sample for diagnostic in soil for weeks if not months. The disease is con-

purposes by the owner's veterinary surgeon or a veterinary officer appointed by the Minister, usually the

swampy land, which favours survival of the organism

Divisional Veterinary Manager or a veterinarian acting

and is likely to harbour Lymnaea spp., the host snail on his/her behalf.

of the liver fluke. The principal toxin produced by

This State Veterinary Service is responsible for the

the organism is beta, which is haemolytic, necrotizing

disease investigation and a stained smear is made from

and lethal.

a superficial blood vessel and examined microscopi-

After ingestion the organism is transported from the

cally. If the Veterinary Inspector is satisfied that disease

alimentary tract to various organs. Smith (1957) recov-

does not exist, the Local Authority is informed and the

ered the organism from the liver, kidneys and bone

movement restrictions are withdrawn. If the inspector

marrow of normal cattle, where it remains as a latent

is not satisfied, an unfixed blood smear and a sample of

infection. It is thought that, as in infectious necrotic

blood is submitted to the Central Veterinary Labora-

hepatitis, the latent spore infection is activated by

tory for cultural and biological examination and the

liver damage, especially by migrating immature liver

Local Authority informed accordingly: On receipt of

fluke. The disease has been produced experimentally by

this information the Local Authority is responsible for

infecting calves orally with the spores of Cl. novyi type disposal of the carcass, usually by burning, and carrying

D and carrying out a liver biopsy, and by implanting the

out disinfection of the premises as prescribed in the

organism in the liver suspended in calcium chloride

Order. The disinfectants of choice are 5 per cent lysol,

solution (Blood et al., 1983). It has also been postulated 5 per cent formalin or 5–10 per cent caustic soda. When

that telangiectasis may also be a precipitating factor.

these procedures have been completed, movement

When conditions are favourable for the activation of

restrictions are withdrawn.

the latent infection, the damaged liver tissue provides

Confirmation of the disease is dependent on the

a focus for initial multiplication of the bacteria. An

results of the examinations at the Central Veterinary

organized thrombus develops in a subterminal branch

Laboratory, which may take seven or more days. On

of the portal vein resulting in a large anaemic infarct in rare occasions disease is not confirmed; even so the which further rapid bacterial multiplication and toxin existing procedures are considered effective in view of production occurs. The toxin produces a haemolytic the serious nature of the disease and the human health anaemia, and later a bacteraemia develops. The dura-implications.

tion of illness may vary from about 18 hours to four Where enzootic disease exists, then an avirulent days. Bacillary haemoglobinuria has been observed in spore vaccine is available, and all cattle should be North and South America, Australia and New Zealand. vaccinated on an annual basis. Vaccination is seldom Few incidents have been reported in Great Britain. necessary in Great Britain (see p. 1005).

The number of confirmed incidents in Great Britain
Signs

is now very low. This is largely due to the efforts of feedingstuffs compounders, who no longer use meal Cattle at pasture that are inspected at infrequent inter-

and bonemeal (MBM) in their feeds. In other countries, vals may be found dead. The onset of disease is usually



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Fig. 44.1

Bacillary haemoglobinuria: a large infarct in the bovine liver caused by Cl. novyi type D.

sudden, with cessation of feeding, rumination and defecation. Cows in lactation suffer a sudden and dramatic drop in milk yield. Animals are disinclined to move and hyperaemia (Fig. 44.1). The kidney cortex is petechial the temperature is elevated to 39–41°C (102–106°F).

ated and deep-red coloured urine may be present in

The mucous membranes are jaundiced and there may

the kidney pelvis and bladder. Evidence of liver fluke

be oedema of the brisket, submaxillary region and con-
damage is not a constant feature.

conjunctiva. Small amounts of bloodstained faeces may be

passed in the early stages, but later there may be a frank

dysentery. Not all these signs may be seen in individual

Diagnosis

cases and the variation may be due, in part, to different

strains of organisms.

Bacillary haemoglobinuria must be differentiated from

other conditions in which haemoglobinuria is one of

*the clinical signs. Acute leptospirosis, due to *Leptospira* Pathology*

interrogans serovar pomona (p. 734), is one of these and One of the obvious
features of the disease is the

to confirm a diagnosis in the live animal serological tests

profound anaemia that develops before death. Blood

and cultural examination of the urine is necessary,

samples from ailing animals show a depressed erythro-

although there should be little difficulty in differentiat-

cyte count, which may be as low as $106/\text{mm}^3$ and haemoglobin values are in the range of 40–80 g/l. Leucocyte and anaplasmosis (p. 761) can be differentiated by the counts are normally elevated to around $20\,000/\text{mm}^3$. demonstration of the organisms in blood smears.

The organism may be recovered from blood cultures

Post-parturient haemoglobinuria is accompanied by a taken during the acute phase. There is a very obvious hypophosphataemia (see p. 792), whilst blood and liver haemoglobinuria in a proportion of cases, but there are copper levels are elevated in chronic copper poisoning no free red cells in the urine.

(p. 948). Both these conditions and haemoglobinuria

In the typical case, the necropsy picture is considered due to the consumption of cruciferous plants (p. 941), to be pathognomonic, the main features being generalized jaundice often with anaemia; subcutaneous

such as rape and kale, are afebrile.

Confirmation of diagnosis of bacillary haemoglobin-

oedema especially over the brisket; accumulation of

uria is based on the clinical signs, necropsy findings and slightly bloodstained fluid in the pericardial sac, pleural demonstration of the causal organism in the liver lesion and peritoneal cavities; widespread haemorrhages in and other sites by fluorescent antibody techniques. It the subcutaneous tissues, over the pleural and peritoneal serosa, and endocardium; haemorrhagic abortion and demonstrate toxins in the liver infarct, but both mastitis and enteritis. The liver is usually mahogany procedures are time-consuming and laborious.

Bacterial Conditions • 721

Treatment and prevention

ference with the secretion of acetylcholine, which is the chemical mediator of nerve impulse transmission.

Bacillary haemoglobinuria can be successfully treated

Two forms or types of botulism are recognized in by the administration of wide-spectrum antibiotics in cattle. The first is the form that develops after the consumption of, or contact with, carcasses or skeletons of

ment with large doses of penicillin is also effective
dead animals containing botulinum toxins. The second
(Williams, 1964). Supportive treatment by the adminis-
form of the disease follows the consumption of con-
centration of electrolyte solutions orally and parenterally
served fodder that is contaminated by the toxins.

and the provision of mineral supplements containing

The first type of botulism has been widely reported
iron, copper and cobalt is also necessary, as well as
in South Africa (lamsieket), Australia (bulbar paralysis)
careful nursing.

and in the USA (loin disease). In these countries it

The disease can be successfully prevented by
has been associated with low phosphorus levels in soil,
vaccination with an aluminium hydroxide adsorbed,
poor pastures and drought. Under such circumstances
formalinized whole culture and it is also claimed
animals including the local fauna, which carry Cl.
that infectious necrotic hepatitis vaccines also confer
botulinum in their intestines, die and the organisms
immunity. Annual vaccination is necessary in enzootic

invade the carcass tissues and produce large amounts of toxin. Muller (1961) has demonstrated that the levels of toxin may reach 10⁵–10⁶ mouse lethal units/g of tissue. Animals that consume tissue from such carcasses,

of toxin. Muller (1961) has demonstrated that the levels of toxin may reach 10⁵–10⁶ mouse lethal units/g of tissue. Animals that consume tissue from such carcasses,

Botulism

because of a phosphorus deficiency and a subsequent pica, or feed shortage, ingest both toxin and Cl. botu-

Botulism may be defined as a lethal type of food

linum spores and will subsequently become a source of poisoning in man and several species of animals,

toxin and spores for other animals. In Europe, similar

caused by the ingestion of Clostridium botulinum

circumstances may arise after the spreading of litter

toxins, which have been produced by the organism in

from poultry houses on to cattle pastures. Investiga-

decaying plant or animal tissue.

tions into such outbreaks reveal the presence of poultry

carcasses in the litter, which are the source of the toxins

(Appleyard & Mollison, 1985; Clegg et al., 1985).

Aetiology

The second type of botulism, forage poisoning, occurs Smith (1977) defines Cl. botulinum 'not as a single species, but as a conglomerate of several distinct culture baled hay and silage, is fed to cattle. The source of toxin groups, alike in that they are clostridia and produce has usually been identified as the carcasses of small animals (mice, rats, rabbits and birds) accidentally killed are a number of distinct serological types (A, B, C, D, E, F and G) and although the toxins are similarly grass or cereal crop. Prevot & Sillioc (1955) recorded designated, the serological specificity of the toxin that more than half of the cattle botulism in France was produced by any strain may not be entirely related to associated with the presence of cat carcasses in the feed. its serological classification.

Fjolstad & Kluna (1969) also reported an outbreak in Like other clostridia, Cl. botulinum is a spore- which the source of toxin was a cat carcass and demon-

forming organism, which under certain conditions
strated 500 000 mouse lethal doses/g of the carcass. A
thrives in putrefying animal tissue or decaying plant
hedgehog, discovered in a hay rack, was the source of
material. The organism has a world-wide occurrence
toxin in an outbreak in cattle with 20 000 mouse lethal
and is found in the intestinal tracts of herbivores and in
doses in its subcutaneous tissue (Ektvedt & Hanssen,
soil. It would appear that different soil types favour
1974). Recently, however, it has been demonstrated that
different types of the organism. Smith (1997) reported
proteolytic strains of *Cl. botulinum* may produce toxin that in the USA, type A
strains were prevalent in
in silage under certain conditions and without the pres-
the alkaline soils of the south-west, types B and E in the
ence of animal tissue (Notermans et al., 1979a, b). In damp soils of most areas,
type C in the acid soils of the
would appear that a low pH prevents toxin formation
Gulf Coast and type D in the alkaline soils of the west.
in silage. The incorporation of poultry manure and
The toxins of *Cl. botulinum* are neurotoxins, causing
poultry waste in cattle feed can also lead to outbreaks

motor paralysis without the development of any gross of botulism (Egyed et al., 1978) and brewers grains or histological lesions in the nervous system. Although contaminated with Cl. botulinum has also been the mode of action is still debated, it appears that the incriminated (Breuknik et al., 1978).

site of action of the toxins is at the synapses of efferent Botulism in any species of animal tends to be associ-parasympathetic and somatic motor nerves, by inter-ated with certain types of the organism. Ruminants are

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susceptible to types C and D, although in The are non-specific and include haemorrhages on the Netherlands disease has been associated with type endocardium and epicardium and congestion of B (Haagsma & Laak, 1977).

the parenchymatous organs and intestinal mucosa.

However, it may be possible to demonstrate toxin in the intestinal contents or liver by mouse inoculation tests.

Signs

Whilst the presence of the organism in the intestine is

Clinical signs usually appear within two to fourteen

of little diagnostic value, its isolation from the liver is of days of ingestion of the toxin, although in peracute

significance. In many cases investigation of outbreaks is

cases the incubation period may be only a few hours.

unrewarding.

Illness is afebrile.

In peracute cases the onset of disease is sudden, char-

acterized by a rapid paralysis and death within 12–18

Diagnosis

hours. In less acute cases the onset is more gradual,

It must be emphasized that in many instances it is not

affected animals showing a progressive muscular paral-

possible to confirm all suspected cases of botulism by

ysis of the head, neck and limbs, leading to recumbency,

detection of toxin either in the sera of affected animals

often with the head and neck outstretched or deviated

or in the intestinal contents or liver at necropsy. As Cl.

towards the flank.

botulinum is not normally found in the liver of cattle, The majority of cases, however, appear to be of the

isolation from the liver is regarded as significant.

subacute type. The first signs are periodic periods of Attempts may be made to demonstrate toxin in suspected feed, but such an approach has its limitations. and an apparent difficulty in chewing and swallowing. Suspected feed may be fed to experimental animals of These signs progress to ataxia, difficulty or inability to the same species, or an infusion of the feed may be rise, recumbency and in some an obvious paralysis administered to experimental animals. However, toxins of the tongue, which protrudes from the mouth. There in feedingstuffs are not distributed evenly, rather in is usually partial or complete anorexia and adypsia. pockets, and samples of feed for testing must therefore Animals may survive for up to seven days after become carefully selected, e.g. from near carcasses or areas ing recumbent, but during the terminal stages respiration of contamination. It is also possible that all the botulism becomes laboured and of the abdominal type due linum-contaminated feed will have been consumed to paralysis of the thoracic muscles. An animal often

before the onset of clinical signs.

appears to rest its chin on the ground and will lift it

Postparturient paresis/hypocalcaemia (p. 781) can be

when stimulated. A foreleg may be extended. As the

differentiated from the disease by the examination of

condition progresses nominal movements reduce. Later

blood samples and the response to calcium therapy. In

stages often involve lateral recumbency with extended

some cases of listeriosis (p. 904) there is a paralysis of

legs which can be flexed. The animal is conscious.

the tongue, but it is accompanied by fever and other clin-

Some animals may recover after showing relatively

ical signs such as unilateral facial paralysis and panoph-

mild clinical signs over a period of three to four weeks

themia. Bovine spongiform encephalopathy (p. 911) in

(Clegg & Evans, 1974; Davies et al., 1974). Clegg & its later stages might be confused but there is no paral-Evans reported that surviving animals developed a pro-

ysis and the animal will still eat.

nounced respiratory roaring sound, which persisted for

three months after recovery.

Treatment and prevention

Pathology

There is little value in the administration of antitoxin,

*A number of authors have reported on the value of
except possibly in the very early stages, and there are
certain biochemical tests on blood and urine from
conflicting views on the merits of purgatives to remove
affected animals as aids to diagnosis, but in cattle such
the toxins from the intestine. However, Breuknik et al.
tests are of little help in the live animal. However, it is
(1978) reported that symptomatic treatment for dehy-
possible in some cases to demonstrate circulating toxins
dration and acidosis seemed to assist recovery. As a
in the serum of clinically affected animals by mouse
general rule, treatment of subacute cases only should be
inoculation tests (Clegg et al., 1985), but such cases are undertaken, as these are
the ones most likely to recover.*

of the peracute or acute type.

Vaccination with toxoid is only necessary in enzootic

*As already indicated, the toxins of Cl. botulinum do
areas and should be given every two years (p. 1005).
not produce any specific or detectable lesions in the*

Where botulism is associated with the disposal of central nervous system, nor do they produce specific poultry litter on to pasture, every effort should be made changes in the carcass. Those changes that are observed to remove any poultry carcasses in it before application.

Bacterial Conditions • 723

All poultry waste should ideally be heat treated animals are related to the site of lesion. Limb involve- before incorporation into compound feeds for direct ment is manifested as a lameness with a swelling of the feeding to livestock.

upper part which, at first, is hot and painful, but later becomes cold and emphysematous. A lesion of the tongue results in a tongue and throat swelling, with the

Clostridial myositis: blackleg and

tongue protruding from the mouth and marked respi- ***malignant oedema***

ratory distress. Stiffness and a reluctance to move is apparent when the sublumbar muscles are involved.

In addition to these clinical signs, there is a marked

Two forms of clostridial myositis are recognized in

depression, anorexia, rapid pulse rate and high temperature, usually in excess of 40°C (104°F). Later there is *chauveoi*, and malignant oedema, which is caused by *dyspnoea*, recumbency and coma leading to death within a number of clostridial species and is nearly always in 12–24 hours.

associated with wound infection. It is often difficult or impossible to differentiate between the two conditions at a clinical or post-mortem examination (Williams, Pathology 1977).

The disease usually runs an acute course so that there is little opportunity for collecting specimens for

Blackleg

laboratory examination before death. After death the carcass becomes bloated and putrefaction occurs. Blackleg or blackquarter is defined as a gangrenous rapidly. Bloodstained froth exudes from the body orifices. It may be possible to palpate the lesion, if it is in

veoi spore infection (Jubb et al., 1983). These authors a superficial muscle group, but this is usually difficult

also refer to a 'false or pseudo-blackleg' caused by the because of the rapid onset of putrefaction.

activation of Cl. novyi and Cl. septicum spore infection, Animals dying from blackleg are in good body condi-but this condition is more appropriately designated

tion. The body cavities contain bloodstained fluid and malignant oedema.

the parenchymatous organs show evidence of degeneration and post-mortem decomposition. All the skeletal

Aetiology

muscles must be carefully examined by palpation and incision for lesions, which may not be extensive. The

Clostridium chauveoi is a Gram-positive, spore-bearing lesion produced by Cl. chauveoi has a characteristic

anaerobic bacillus, the spores of which are highly resistant appearance (Williams, 1977) (Plate 44.2). The muscle is

ant to environmental conditions and therefore re-

blackened, dry and crepitant with a spongy appearance

main viable for many years. It is, like other clostridia,

and a rancid odour. Pale yellow serous fluid surrounds

regarded as a soil organism and following ingestion by

the affected muscle, but this becomes progressively more cattle, sheep and other animals, the spores localize in bloodstained as post-mortem decomposition proceeds. the spleen, liver and muscles (Kerry, 1964). The vegetative form of the organism produces a number of toxins, which are capable of inducing local muscle necrosis and

Diagnosis

toxaemia. The trigger mechanisms responsible for the

A diagnosis can be reached on the basis of clinical signs activation of the endogenous latent spore infection and necropsy findings, but it is essential that when no are unknown, but it is assumed that a lowered oxygen clinical signs have been observed, anthrax (p. 717) is tension and a degree of muscle damage are necessary. eliminated before a necropsy is carried out. Clostridium

After activation, rapid bacterial multiplication and chauveoi can be identified by the staining of lesion toxin formation produce the typical muscle gangrenous impression smears by the specific fluorescent antiglob- lesion and systemic toxaemia.

ulins that are now commercially available. Cultural

Most cases occur in animals between six months and examination is likely to be unrewarding unless fresh two years of age at pasture, although incidents may tissue is available and special techniques used. occur in housed animals.

Blackleg may be confused with other conditions especially when death is sudden. Lead (p. 944) and other chemical poisonings (pp. 941–3) require labora-

Signs

tory examination for confirmation, but the typical Often when stock are infrequently inspected animals lesions of blackleg are absent. Black disease (p. 729) may be found dead without signs of illness having been and bacillary haemoglobinuria (p. 719) may also have observed. The clinical signs that are seen in ailing to be considered in a differential diagnosis, but the

724 • Chapter 44

characteristic liver infarcts are a diagnostic feature of nificance. There is still some uncertainty about the role these two diseases.

of Cl. septicum in bovine malignant oedema.

*Deep puncture wounds,
accidentally inflicted,
provide ideal conditions for the multiplication of anaer-*

Treatment and prevention

*obes and development of malignant oedema. It may
Antibiotic treatment of affected animals is likely to
also develop after surgical operations, parturition, intra-
be effective only if commenced early. Large doses of
muscular administration of non-antibiotic preparations
penicillin (10 000 units/kg body weight) should be
such as prostaglandins (Harwood, 1994), anthelmin-
administered intravenously, followed by longer acting
tic and vitamin preparations, and vaccination. The
preparations, some of which should be given into
clostridia are soil organisms that persist in the animal
the affected tissue (Radostits et al., 2000). However, environment and therefore
readily gain entry to
because of the extensive tissue involvement, even if
wounds. The tissue damage and low oxygen tension
the infection is eliminated, the subsequent muscle loss
allow rapid multiplication and toxin production so*

is so great that recovered animals are of little economic value. Treatment of animals with tongue infection Occasionally, malignant oedema may affect a group of animals that had previously been housed or penned for a short period of time and the absence of any form of early slaughter on humane grounds should be considered. Carcasses should be burned or undergo deep bruising, may have activated a latent spore infection. should not be attempted, because even if successful the animals that had previously been housed or penned for whole tongue or most of it will be subsequently lost and wound suggests that some factor, perhaps trauma from burial.

Blackleg can be successfully prevented by the use of Signs

commercially available *Cl. chauveoi* vaccines and all animals over six months of age should be vaccinated

The disease is usually sporadic involving single or small numbers of animals. All ages of cattle are affected and prior to being turned out in the spring. There are, however, considerable advantages in the use of multi-

clinical signs appear within 48 hours of infection. The

valent vaccines containing the antigens of Cl. chauveoi, clinical signs will vary with the site of infection, but

Cl. novyi and Cl. septicum, which offer maximum protection in all cases, anorexia, depression and fever are very

effective in cattle against blackleg, malignant oedema

marked. A local lesion develops at the site of infection

and black disease (p. 1005).

consisting of a swelling, which becomes tense and

depending on the type of infection may become emphy-

sematous. Lameness, stiffness and muscle tremors may

Malignant oedema

be evident. Animals usually die within 48 hours.

Malignant oedema is considered to be an acute wound

When infection is associated with parturition, the

infection caused by organisms of the genus Clostridium

vulva and perineum swell and there is a bloodstained

(Radostits et al., 2000). However, if blackleg is restricted discharge from the vulva. Death is rapid, usually within

to cover endogenous Cl. chauveoi infection then malignant-24–36 hours after the onset of signs.

malignant oedema must also include those incidents, albeit

relatively few in number, arising from activation of

Pathology

latent Cl. novyi and Cl. septicum, which undoubtedly occur.

In malignant oedema there is little opportunity for the laboratory examination of specimens taken from affected animals. As in blackleg, it is necessary for a

Aetiology

post-mortem examination to be carried out as soon

Clostridium chauveoi, Cl. novyi, Cl. perfringens, Cl. sep-
after death as possible, because of the bacterial invasion

ticum, Cl. sordelli and other clostridia have been iso-of the carcass and rapid onset of putrefactive changes.

lated from, or demonstrated in lesions of clostridial

The site of infection is surrounded by an extensive myositis in cattle. Williams (1977) in a survey of 173 oedema of the subcutaneous tissues and intramuscular

cases in Wales demonstrated Cl. chauveoi in 75 (43 per fascia. It may be possible to identify the initial wound,

cent), Cl. chauveoi and Cl. septicum in 22 (13 per cent), but tissue damage is usually so extensive that the only

Cl. novyi in 53 (31 per cent), Cl. novyi and Cl. septicum trace is a small wound in the skin. The oedema fluid may

in nine (5 per cent), Cl. septicum in 11 (6 per cent) and be clear and gelatinous in Cl. novyi infections with very Cl. sordelli in three (1.7 per cent). In this series Cl. per-little muscle damage. Infection with Cl. septicum pro-fringens was

isolated from about 50 per cent of the
duces an extensive bloodstained frothy oedema, with
lesions, but its presence was not considered to be of sig-
the underlying muscle a dark red colour permeated

Bacterial Conditions • 725

with gas. *Clostridium sordelli* produces changes similar cattle and occasionally sheep, and can be readily cul-to those produced by *Cl. novyi* except that the oedema tured from the urine of affected and carrier animals.

is more bloodstained and has a foul odour. The lesion

Goudswaard and Budhai (1975) identified four

produced by *Cl. chauveoi* is similar to that described serotypes, of which type 1 is the most pathogenic.

under blackleg.

It has been demonstrated that the pathogenicity of *C.*

All body cavities contain bloodstained fluid and

renale is dependent to a large extent on the presence of the parenchymatous organs show degenerative changes

pili on the organism, which assist its adhesion to the

and post-mortem decomposition. If the infection

urinary epithelium. This process is pH dependent and

involves the reproductive tract, the uterus will contain

Takai et al. (1980) showed that the proportion of pili-a large volume of foul-smelling bloodstained fluid and

ated organisms adhering to bladder cells is high at a pH the uterine and vaginal walls will be greatly thickened, above 7.6, but significantly lower at a pH below 6.8, permeated with bloodstained fluid and gas.

a factor that is important in the pathogenesis of pyelonephritis.

The disease is considered to be the result of an

Diagnosis

ascending infection, involving successively the bladder,

The clinical signs and necropsy findings are so charac-

ureters and kidneys. Hiramune et al. (1972) established teristic that diagnosis can be readily reached. It will,

infection in cattle after the introduction of organisms

however, be necessary to resort to laboratory examina-

into the bladder and the characteristic lesions devel-

tion of lesions for the identification of the organisms by

oped. Females are far more susceptible than males to

fluorescent antibody tests or culture.

infection and the short length of the urethra in the

female is thought to be a major factor in the establish-

ment of infection. Stasis of urine is an important pre-

Treatment and prevention

disposing factor in the pathogenesis of pyelonephritis

Affected animals should be treated with high doses of antibiotics, preferably parenteral penicillin or broad spectrum. In addition, wounds should be drained and tract occurs through the presence of calculi or pressure irrigated with antiseptic solutions, and packed with a exerted by a gravid uterus.

suitable antibiotic preparation.

The disease is widely recognized in Europe and

Trivalent vaccines, containing the antigens of Cl.

North America, but the prevalence of infection is

chauveoi, Cl. novyi and Cl. septicum are available largely unknown. Morse (1950) found that C. renale

and are effective in preventing malignant oedema

could be recovered from the urine of 22.7 per cent of (p. 1005).

cattle in herds in which pyelonephritis had been con-

Harwood (1984) highlights the dangers from intra-

firmed, whereas only 10.7 per cent of cattle in other muscular injections administered to cattle, particularly herds were infected. Clinically infected or carrier cows if infection is introduced and if large volumes are used, are the principal source of infection, the disease being and under such circumstances routine vaccination is mainly transmitted by direct contact, although Morse recommended.

(1950) reported that infection was transmitted by indirect contact from affected and carrier cows tethered in stalls to those in adjacent stalls. In some herds there

Contagious bovine pyelonephritis

is circumstantial evidence to support the existence of venereal spread and the organism has been readily This disease is a specific infection of the urinary tract of isolated from the prepuce of normal bulls (Hiramune cattle, which results in a chronic purulent inflammation et al., 1975).

of the kidneys, ureters and bladder.

Signs

Aetiology

In affected herds, clinical cases appear sporadically with Corynebacterium renale is considered to be the specific animals under three years of age rarely affected. Most causal agent, although other organisms, especially streptococci, staphylococci, Arcanobacterium (Actinomyces, usually in winter. Corynebacterium) pyogenes and Escherichia coli, as There is considerable variation in the clinical signs well as C. renale are present in the urine of some observed from case to case, particularly during the early animals with pyelonephritis and may also be implicated. Bloodstained urine may be passed intermittently cated. Corynebacterium renale is an obligate parasite of by an apparently healthy animal over a period of weeks,

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before other signs appear. In other animals, one, two or

Diagnosis

more attacks of acute colic lasting up to 6 hours or more

The diagnosis of pyelonephritis is based on the clinical

may be the first sign. More frequently, however, the

signs, the changes in the urine and the presence of C.

onset is insidious. The most common signs are a gradual

renale in the urine together with the detectable abnormal loss of condition, a slowly declining milk yield, fluctu-

malities in the urinary tract. Enzootic haematuria

ating or capricious appetite, intermittent fever, and

(p. 947) has some clinical features in common with

the intermittent passage of bloodstained urine. As the

pyelonephritis, but it is afebrile, lesions are confined to

disease progresses, urination becomes more frequent

the bladder and the urine from such cases is sterile or

and painful with the passage of small volumes of urine

negative for C. renale. Similarly, non-specific cystitis containing blood and tissue debris. During the later

may also resemble pyelonephritis, but the bladder only

stages it may be possible to detect, by rectal examina-

is affected and the urine is sterile or negative for C.

tion, enlargement of one or both kidneys, a thickened

renale.

bladder and enlargement of one or both ureters,

particularly the terminal portions where they cross

the neck of the bladder. Frequently, palpation of

Treatment and prevention

the kidneys induces a pain response. The course

of the disease may run from a period of weeks to two

Corynebacterium renale is sensitive to a range of antibi-or more months, death resulting from a combination

otics but penicillin remains the antibiotic of choice for

of kidney failure and blood loss with an extensive

the treatment of pyelonephritis in the bovine. A com-

loss of condition.

plete recovery can be achieved if treatment is

commenced during the early stages, when little tissue

damage has occurred. In advanced cases, however,

Pathology

when there is considerable tissue destruction, only a

The urine of clinically affected animals is turbid and in

temporary recovery can be achieved through antibiotic

the early stages intermittently bloodstained, but in the

therapy, although this may enable an animal to be fat-

later stages the urine is almost constantly bloodstained.

tened and subsequently sent for slaughter. Large

Microscopic examination of the centrifuged deposit of

doses of procaine penicillin G should be administered,

the urine reveals the presence of erythrocytes, leuco-

e.g. 10 000–15 000 iu/kg daily for at least 10 days.

cytes and epithelial tissue debris. Corynebacterium

The acidification of the urine by the administration of renale can be readily demonstrated in Gram-stained monobasic sodium phosphate is still considered by smears and Ado & Cook (1979) have reported on the some as useful supportive therapy and 100 g daily for value of fluorescent antibody tests for the identification a period of five days during antibiotic treatment is of the organism. It can be readily isolated on blood agar recommened.

and other media in common use. The clinical signs

There are no specific control measures other than are so characteristic that haematological examination isolation of affected animals and thorough cleansing and blood chemistry estimations are seldom considered and disinfection of the contaminated environment. In necessary. In any case, such examinations are unlikely affected herds where natural breeding is practised, the to reveal any abnormalities until the disease is well introduction of artificial insemination may achieve a

developed when anaemia and uraemia are the most reduction in the number of clinical cases.

prominent findings.

Animals dying from pyelonephritis are usually in poor condition and the carcass pale and anaemic. Spe-

Endocarditis

cific lesions are confined to the urinary tract. One or both kidneys are enlarged with less well marked lobu-

Endocarditis may be defined as inflammation of the lation than normal and a markedly thickened capsule.

endothelial lining of the heart. The inflammatory

The surface is mottled by greyish white necrotic areas.

processes usually result in valvular insufficiency or

On section the renal pelvis is greatly dilated and con-

stenosis that interfere with the flow of blood into and

tains varying amounts of blood, pus and mucoid fluid.

out of the heart, leading to congestive heart failure.

Greyish white streaks of necrotic tissue radiate from

the pelvis towards the cortex and there may be numer-

Aetiology

ous abscesses in the cortex and medulla of each lobule.

*The ureters are grossly enlarged and distended by
Most cases of bovine endocarditis appear to be caused
blood, pus and mucus. The bladder wall and the urethra
by bacterial infection. Several species of bacteria have
are thickened and the mucosa oedematous, haemor-
been incriminated, but streptococci especially entero-
rhagic and necrotic.*

cocci of Lancefield's group D, A. pyogenes, staphylo-

Bacterial Conditions • 727

*cocci, Mannheimia and Pasteurella species are the com-
monest (Evans, 1957; Larsen, 1963).*

endocarditis may be insidious.

It would appear that a persistent bacteraemia is nec-

A recurrent or persistent fever of 40–41°C

essential for the development of endocardial lesions and

(104–106°F), anorexia, depression and a reluctance to

move are the usual early signs. Pinching of the withers

a primary focus of infection in the form of mastitis,

and ballottement of the sternum ventral to the heart

metritis, reticulitis, limb abscesses, etc. can be identified elicit a pain response. The heart rate is accelerated to at post-mortem examination (Evans, 1957; Andersen, 100–120 beats/minute and in due course the jugular 1963; Larsen, 1963).

vein becomes engorged, which is followed by oedema

Although it is generally accepted that the causal of the brisket and submandibular areas. The detection organisms are transported to the heart via the blood- of a heart murmur on auscultation is an important clin- stream, the method by which they reach the endocar- ical feature, but this is not easily detected when the right dial lesion is still uncertain. It is possible that some

atrioventricular valves are involved. Lacuta et al. (1980) bacteria may be able to adhere to the intact endothe-have reported on the value of electrocardiography

lium. However, it is more likely that they adhere to and echocardiography in the diagnosis of endocarditis.

damaged endothelium and it is assumed that trauma

Whilst echocardiography is of value in that it will and debility are the main factors in producing sufficient demonstrate reflected echoes from the vegetative

damage for the bacteria to localize in the endothelium. lesions and detect abnormal valve movements, electro- This hypothesis is based on the fact that the usual sites cardiography is less so because the abnormalities shown of the lesions are on the free edges of the valves are not diagnostic of endocarditis.

exposed to the blood flow and that they are in apposi- As the disease progresses, secondary involvement of tion to others. The heart valves of the bovine have their other organs and systems occurs, leading to pneumonia, own blood supply and it is thus possible for the bacte- nephritis, arthritis, etc. Progressive weight loss, anaemia ria to produce emboli in the capillaries, which form a and weakness inevitably is followed by recumbency and focus of infection.

death. The course of the disease may extend from one The early lesions of endocarditis are seldom seen or two weeks to two or three months.

except in experimental infections. Initially, the leaflet of the valve becomes swollen and an irregular ulcer devel-

Pathology

ops on its surface, in which the bacteria localize. From this ulcerated area the characteristic vegetative structures develop. Blood samples from affected animals show a leucocytosis and a shift to the left, although in the more chronic thrombi, except that they contain few platelets. Several cases these changes are less well marked. Plasma fibrin layers of thrombus-like material are deposited on the valve. Fibrinogen levels are elevated (Wuijckhuise-Sjouke, 1984) affected valve in response to the bacterial activity, but this is a feature of inflammation of serous membranes so that vegetations assume a cauliflower or wartlike appearance as well as endocarditis. The causal organism can be isolated on blood culture during periods of fever but the valves become distorted and shrunken and functionally incompetent. Fragments of at least 20 ml of blood are necessary and the cultures of the vegetation may become detached to form emboli, which lodge in other organs.

The heart lesions found at necropsy are fairly con-

stant (Plate 44.3). The pericardial sac is distended with varying amounts of oedematous fluid and the heart

Signs

is enlarged and distorted, due to hypertrophy of the

All ages of cattle are affected although Larsen (1963)

myocardium and dilatation of one or more chambers,

found that nearly half of the 53 bovine cases he encoun-

tered were in animals between two and three years of

atrioventricular valves. In the majority of cases the right

age. Both Evans (1957) and Biering-Sørensen (1963)

atrioventricular valves are affected. The affected valves

found that 3.9 per cent of cattle examined in abbatoir

are shrunken and thickened, particularly in the later

surveys had either died from, or been slaughtered

stages, and attached to them there are wart-like or

because of, endocarditis.

cauliflower vegetations.

The published descriptions of the clinical signs of

The parenchymatous organs may show pathological

bovine endocarditis are varied and reflect the stage to

changes. The lungs are passively congested with fre-
which the endocarditis had progressed and also the
quently a number of embolic infarcts, and the liver is
extent to which the clinical signs were attributable to
usually enlarged due to passive venous congestion and
the primary focus of infection (Evans, 1957; Power &
may show evidence of cirrhosis. Numerous small haem-
Rebhun, 1983). Thus the initial signs may not indicate
orrhagic foci are scattered over the surface of the

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kidneys and within the cortex and there may be a
of Great Britain in 1926. The lesions in the affected
number of infarcts or abscesses in the cortex.

animals were those of a bronchopneumonia and

although *Pasteurella* was isolated from the lungs it was not fully typed but likely
to have been type B. Since

Diagnosis

then there have been no further reports of haemor-

The clinical diagnosis of endocarditis is dependent upon
rhagic septicaemia in Great Britain. In the USA only
the detection of heart murmurs, which may be

four outbreaks, three in bison and one in cattle, have extremely difficult when the lesions involve the right been confirmed and Carter (1982) speculated on its atrioventricular valves. Thus it may be difficult or disappearance from that country.

impossible to differentiate between endocarditis and

Pasteurella multocida is not a very resistant organism pericarditis (p. 731) (John, 1947) or other causes of con- and it is unlikely to survive for long periods outside the

gestive heart failure. Cardiac lesions of enzootic bovine animal body. In soil and mud, for example, where com-leukosis (EBL) (p. 695) may also produce signs of competition from other organisms is strong it does not gestive heart failure, but affected animals show a per-survive for more than 24 hours (Bain, 1963). Toxins sistent lymphocytosis and are positive to the agar gel have been demonstrated in culture filtrates, but all the immunodiffusion and ELISA tests for EBL.

evidence suggests that the most significant of these are endotoxins, which are important in producing the clinical disease and rapid death.

Treatment

The main source of infection is the carrier animal, the

The organisms associated with bovine endocarditis are

organism localizing in the nasopharyngeal mucosa and

sensitive to a range of antibiotics. However, the nature

tonsils. In herds that experienced haemorrhagic septi-

of the heart lesions is such that therapeutic concentra-

caemia 44.4 per cent of healthy cattle were carriers,

tions of antibiotic may not penetrate through to the

whereas the carrier rates in three herds in which the

bacteria. Furthermore, the permanent damage inflicted

disease had not been confirmed were 3.89 per cent, 5.5

on the heart valves and the embolic lesions in other

per cent and nil (Mustafa et al., 1978). Furthermore, the organs cannot be

effectively repaired. Thus treatment

carrier rate was higher in cattle under two years of age

of ailing animals is unlikely to lead to complete recov-

than in adult cattle. It is estimated that approximately

ery. Although temporary improvement may occur after

10 per cent of carrier animals become immune.

intensive antibiotic treatment for seven to ten days,

Spread of infection is through the ingestion of feed

a relapse within seven days is the usual outcome.

contaminated by carrier or clinically affected animals.

However, Power and Rebhun (1983) reported that nine

The role of biting and blood-sucking ectoparasites in

cows, in which an early diagnosis had been made,

the spread of infection is still unclear, although

responded to long-term penicillin therapy.

Macadam (1962) suggested on the basis of experimen-

tal work in rabbits that ticks could transmit the disease.

It is generally accepted that environmental stress

is an important factor in precipitating outbreaks of

Haemorrhagic septicaemia

disease. Outbreaks occur when animals are exposed to

cold and wet weather, housed under poor conditions or

Haemorrhagic septicaemia, or more appropriately sep-

exhausted by prolonged periods of work. Under such

ticaemic pasteurellosis of cattle, is a peracute disease,

circumstances the immunity of carrier animals wanes,

which is characterized by a septicaemia and a very high

allowing a rapid multiplication of the organism and

mortality rate.

spread within the carrier animal and its subsequent dissemination to susceptible contact animals.

Aetiology

Signs

Carter and Bain (1960) and Carter (1982) highlighted the confusion and conflict in the terminology of dis-

*The disease may be peracute with death frequently
eases attributed to Pasteurella infection in bovines. Thus occurring so rapidly that few, if any, clinical signs are*

*it is difficult to assess the accuracy of many of the early
observed. In the acute form, there is a sudden onset of
reports on haemorrhagic septicaemia. It is now consid-
fever (41–42°C, 106–108°F), severe depression, oedema
ered to be a primary pasteurellosis caused by Pas-
of the throat, profuse salivation and rapid death, usually
teurella multocida capsular serotypes B and E, which
in less than 24 hours. The oedematous form of the
appears now to be confined largely to the tropical coun-
disease is also acute with the development of hot
tries of Asia. Shirlaw (1938) reported on the occurrence
painful swellings of the head, throat, brisket, perineum*

of septicaemic pasteurellosis in the Northumbrian area and limb(s). Severe oedema of the head and throat may

Bacterial Conditions • 729

result in dyspnoea and eventually death through neous swellings may develop in some animals and asphyxiation rather than death from a septicaemia. after the administration of some batches of vaccine, anaphylactic shock has occasionally been recorded.

*A live streptomycin-dependent mutant of *P. multocida* Pathology vaccine has been developed (Wei & Carter, 1978) and Large numbers of *Pasteurella* organisms can be demonstrated in the nasal discharges and saliva of clinically affected animals. The course of the disease is usually as yet.*

*less than 14 hours, which limits the opportunities for The observations of Sawada et al. (1985) on naturally carrying out laboratory examination on specimens from acquired immunity to *P. multocida*, capsular types B ailing animals.*

and E, are of considerable interest. They found, on the
On post-mortem examination the most prominent
basis of serological and passive immunity tests in mice,
features are widespread petechial haemorrhages on the
that 81 per cent of feeder calves were immune to type
serous membranes and in various organs, especially
B and 91 per cent to type E. The immunity appeared to
the lungs and muscles, and a subcutaneous oedema
develop in the absence of either of these serotypes in
of the throat region. The lungs are oedematous, may
the microbial flora of the calves.

also show an early interstitial pneumonia and often
there is a haemorrhagic gastroenteritis. In the oedema-
tous form of the disease, there is widespread oedema of

Infectious necrotic hepatitis

the head, tongue, brisket and/or one or more limbs as
(black disease)

well as widespread petechiation. The spleen is not
greatly enlarged.

Infectious necrotic hepatitis is a highly fatal acute or
peracute infectious disease of sheep and cattle, charac-

terized by the presence of one or more necrotic areas

Diagnosis

in the liver, in which *Cl. novyi* type B has multiplied and The regional occurrence of the disease is of some aid

produced lethal toxins.

in diagnosis, but the clinical signs, rapid course and necropsy findings are similar to those of other diseases, e.g. anthrax (p. 717), clostridial myositis (p. 723), acute

Aetiology

leptospirosis (p. 735), so that confirmation can only be

The causal organism is *Cl. novyi*, which like other achieved by isolation and identification of the causal

clostridia is a Gram-positive spore-bearing bacillus,

organism. *Pasteurella multocida* can be readily isolated widely distributed in the environment, particularly in

from heart blood, spleen, liver and other sites, but

soil. The more pathogenic types, however, are more

identification of the specific serotypes may require

prevalent in those areas where infectious necrotic

submission to specialist laboratories.

hepatitis occurs. After ingestion, spores are transported

to the liver and other locations, where they remain as a

latent infection. On the basis of toxin production a

Treatment and prevention

number of types are recognized, but infectious necrotic

Pasteurella multocida is sensitive in vitro to a range of hepatitis in cattle is caused by type B, which produces

antimicrobial substances, including oxytetracycline,

large amounts of the lethal alpha toxin.

florfenicol and penicillin/dihydrostreptomycin and, in

The disease occurs in cattle and sheep in those areas

theory, therefore treatment of clinically affected

where liver fluke infection also occurs (p. 276), and it

animals should be feasible. However, the sudden onset

has been demonstrated experimentally in sheep that

and rapid course of the disease are such that treatment

migrating immature liver fluke produce liver damage

during the very early stages only is likely to be suc-

and through this an environment suitable for the

cessful. Even if all the organisms are eliminated, death

activation of the latent infection. It is assumed that

from Pasteurella endotoxaemia may still occur (Jubb et the same sequence of events occurs in cattle, although

al., 1985).

Williams (1964) noted the absence of liver fluke

The only effective method of control is vaccination

damage in some confirmed cases. It has been demon-

of all herds at risk. A dead vaccine in an adjuvant base

strated that the survival of Cl. novyi and Fasciola containing paraffin and lanolin has proved highly effective. Fasciola is favoured by the same type of soil environment when used prophylactically and is also of value in

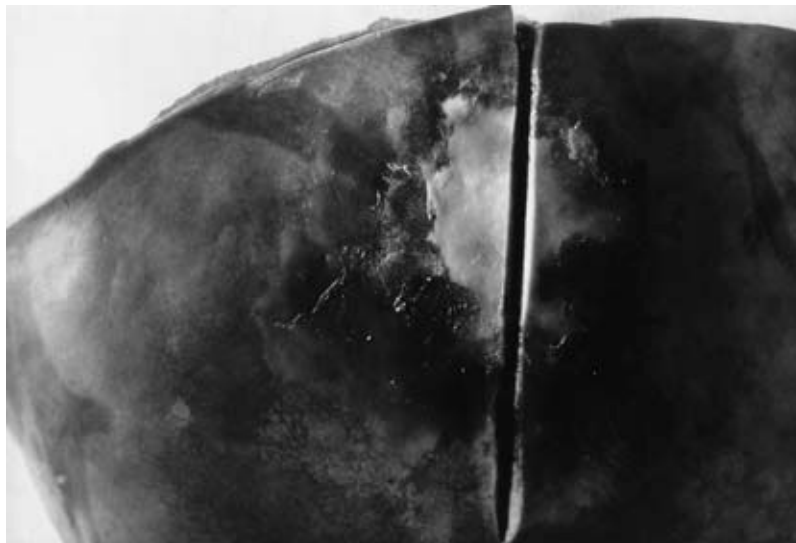
ment (Bagadi & Sewell, 1973). Other migrating para-

reducing losses if used during an outbreak, immunity

sites, e.g. the intermediate stages of certain canine

persisting for at least 12 months. Persistent subcuta-

cestodes, may also activate latent Cl. novyi infection.



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Fig. 44.2

A typical black disease lesion in

the vertical lobe of a bovine liver. Note the central necrotic area surrounded by a zone of congestion.

The organism multiplies rapidly in the damaged liver clear gelatinous fluid in variable amounts is present in and large amounts of the lethal and necrotizing toxin the axillary and inguinal regions, and over the brisket. are formed, which leads to the production of the char-Fluid, sometimes bloodstained and containing fibrin acteristic lesion. The toxin is rapidly absorbed, leading strands, is present in the pleural and peritoneal cavities to a systemic toxæmic and it is only rarely that the and in the pericardial sac. Haemorrhages are scattered organism can be recovered from other organs in the over the endocardium and sometimes over the epi-fresh carcass.

cardium, parietal pleura and peritoneum. The mucosa The disease has been confirmed in most countries of the abomasum is usually congested and often the where fascioliosis occurs. Cattle of all ages may be abomasal wall is distended by a gelatinous oedema. The

affected; Williams (1964) found that in a series of 46 duodenum and jejunum may show a patchy mucosal cases, one was in a six-month-old calf, two were in cows congestion.

over seven years old and the remainder in bullocks and The pathological changes in the liver are characteris- heifers between one and two years old. There is little tic. The organ is a dark brown colour due to venous con- information on morbidity rates in cattle, but experience gestion and the gall-bladder is usually distended. One, in the UK indicates that in most herds only single two or more sharply demarcated yellowish necrotic animals succumb. It is likely, however, that the disease areas up to 8 cm (3 inches) (Fig. 44.2) or more in is becoming more common in some areas.

diameter may be identified, most frequently on the surface but also in the depths of the parenchyma, where they may be missed unless the liver is carefully palpated

Signs

and sectioned with a knife. The necrotic area is sur-

Most cattle with infectious necrotic hepatitis are found

rounded by an obvious zone of deep congestion.

dead but, in some, illness may last for one to two days

Lesions may be found anywhere in the liver, but the

(Herbert & Hughes, 1956). Affected animals develop

majority tend to be located in the ventral lobe. Evidence

severe depression, are disinclined to move, with a

of liver fluke migration in the form of subcapsular haem-

normal or slightly elevated temperature. Signs of dis-

orrhages and greenish yellow scars (accumulations of

comfort are exhibited on palpation of the liver region.

eosinophils) may be evident near the lesion.

Microscopical examination of sections prepared from

the liver lesions show a central core of necrotic tissue

Pathology

demarcated from the congested parenchyma by a leu-

Because of the acute nature of the disease it is

cocytic barrier (Fig. 44.3). Within the barrier and at the

not possible to carry out ante-mortem laboratory

periphery of the necrotic tissue, large numbers of

examinations.

vegetative clostridia are evident. The hepatic cells

Animals dying from the disease are usually in good condition. The subcutaneous vessels are engorged and immediately adjacent to the leucocytic barrier show degenerative changes.



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Fig. 44.3

A section through a black disease lesion demonstrating the central necrotic area and the clearly stained leucocyte barrier. (Haematoxylin and eosin, ¥5.)

Diagnosis

Two doses of vaccine at an interval of not less than one month are required to produce a satisfactory immunity

The post-mortem findings of the characteristic lesions

(see p. 1005). Because of the association with liver fluke provide a firm basis for arriving at a diagnosis. Fluorescent antibody techniques can be used for the rapid identification of this parasite. (see p. 278).

identification of Cl. novyi in impression smears made from the periphery of the liver lesion as described by

Batty et al. (1964). Toxins may also be demonstrated in the liver lesions and peritoneal fluid (Williams, 1964),

Pericarditis

although the laboratory tests for these are laborious and time-consuming. If the post-mortem findings are Infection of the pericardial sac by micro-organisms inconclusive and the carcass shows evidence of decomposition results in a purulent or non-purulent pericarditis with position then the fluorescent antibody and toxin tests the accumulation of varying amounts of fluid and cannot be reliably used, since Cl. novyi may have multiple masking or muffling of heart sounds, and multiplied in the liver and other locations after death may lead to congestive heart failure.

(Bagadi & Sewell, 1974), and some of the strains found

in livers are of low pathogenicity (Williams, 1964) but

Aetiology

are detected by fluorescent antibody techniques.

Other cases of sudden or rapid death have to be

A wide range of micro-organisms are associated

considered in differential diagnosis and these include

with bovine pericarditis and they include A.

anthrax (p. 717), other clostridial infections such as

pyogenes, Haemophilus somnus, Mycobacterium bovis, clostridial myositis (p. 723), lead poisoning (pp. 906,

Mannheimia and Pasteurella species, staphylococci, 944) and metabolic disorders (Chapter 46). It is vital

streptococci and Mycoplasma species.

that blood smears for anthrax diagnosis be examined

Infection with pyogenic bacteria is nearly always

before post-mortem examination is carried out and

primary and frequently associated with traumatic reti-

thereby prevent environmental contamination with

culitis. The organisms are introduced during penetra-

anthrax spores.

tion of the pericardial sac by a foreign body originating

from the reticulum, or infection may be the result of

direct spread from a traumatic mediastinitis. In some

Treatment and prevention

instances a purulent infection may be superimposed on

There is little opportunity for effective treatment,

an original non-purulent fibrinous pericarditis. Infec-

although in those cattle that are ailing for one to two

tion of the pericardial sac also occurs through the local-

days, the administration of broad-spectrum antibiotics

ization of a blood-borne infection or through the direct

*or penicillin and *Cl. novyi* antiserum may be of value.*

extension of a myocarditis or pleurisy.

Effective vaccines have been developed and in areas

During the early stages of a purulent pericarditis

where liver fluke occurs their use should be advocated.

there is a marked hyperaemia and deposition of a fib-

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rinous exudate on the epicardium. Varying amounts of

the overall haematological picture depends to some

fluid accumulate within the pericardial sac and, as the

extent on the causal agent and other lesions that may

volume significantly increases, both atria and the right

be present, so that the total white cell count may vary from only marginally above the normal range to more than 30 000/ml. The cardiac lesions found at post-mortem examination depend on the type of infection and the duration of illness. In acute purulent pericarditis the pericardium and epicardium are thickened and covered by a fibrous deposit, and the pericardial sac distended with fluid, which varies from a dirty grey to a yellowish green colour with a foul odour. The atria and the right ventricle are compressed. When the condition is associated

results. A severe toxæmia may also develop. In some instances, however, the volume of exudate is small and adhesions develop between the pericardium and pericardium, which become organized, resulting in complete attachment of the epicardium and pericardium, and epicardium are thickened and covered by a fibrous deposit, and the pericardial sac distended with fluid, which varies from a dirty grey to a yellowish green colour with a foul odour. The atria and the right ventricle are compressed. When the condition is associated

marked distension of the pericardial sac. The epicardium with traumatic injury it may be possible to identify a foreign body, but should this be wire it may have disintegrated. Occasionally, a rupture of one of the coronary vessels, an atrium, or ventricle may occur in traumatic pericarditis and under such circumstances the pericardial sac contains a mixture of blood and purulent fluid. In more chronic cases, the grossly thickened pericardium and epicardium are closely adherent except for loculi containing inspissated or thick creamy pus giving Signs

a 'bread and butter' appearance. Signs indicative of The early clinical signs of pericarditis are often difficult to identify because they are obscured or dominated by congestive heart failure are also identifiable and these

include congested liver and lungs, engorged jugular
those of the primary disease, e.g. pleurisy, traumatic
veins and subcutaneous oedema of the submandibular
reticulitis, and the onset of pericarditis may therefore
space, brisket and inguinal region. If the pericarditis is
be insidious. Arching of the back, reluctance to move,
associated with other conditions, e.g. traumatic reticuli-
shallow and rapid abdominal respirations and elevation
tis, pneumonia or pleurisy, then lesions attributable to
of body temperature to 40–41°C (104–106°F) are the
them will also be present.

initial signs. The pulse rate is increased, and percussion
Evidence of a non-purulent or fibrinous pericarditis
and palpation over the cardiac area of the thoracic wall
is sometimes seen in apparently healthy animals dying
elicits a pain response. It may be possible to detect a
from other causes. In such animals there is a patchy
rough pericardial friction sound by auscultation during
or diffuse thickening of the pericardium with similarly
the early stages, but this is by no means easy and may
distributed adhesions to the epicardium, which can be

be missed.

easily broken down. There is no evidence of interfer-

*As the condition progresses, fluid accumulates in the
ence with the cardiac function and the lesions are of no
pericardial sac and the heart sounds become muffled,
pathological significance.*

although sometimes a splashing sound may be detected.

The signs of congestive heart failure, engorged jugular

Diagnosis

*veins, a jugular pulse and subcutaneous oedema of
the submandibular space, brisket and inguinal region,*

*The clinical diagnosis of pericarditis is not easy because
develop and most animals die within one or two weeks.*

the signs may be dominated or obscured by those of the

*Some animals survive the acute phase with antibiotic
primary disease and it may be difficult to recognize the*

*treatment, and the signs of congestive heart failure
pericardial sounds. The characteristic friction sounds in*

abate slowly. However, complete recovery seldom if

the early stages may be confused with those of pleurisy,

ever occurs and recovered animals should be sent for

*although the latter is synchronized with the respiratory
slaughter at the earliest opportunity.*

*movments. Similarly, the pericardial sounds may
resemble the murmurs (p. 727) produced by valvular
lesions, but unlike the murmurs, they persist for the*

Pathology

*whole of the cardiac cycle. The heart sounds may also
Blood samples from animals affected with a purulent
be muffled by effusion associated with pleurisy but
pericarditis, especially traumatic pericarditis, show a
under such circumstances there are signs of respiratory
marked leucocytosis and a shift to the left. However,
involvement.*

Bacterial Conditions • 733

Treatment

*and a lowered oxygen tension, immediate growth and
toxin production will occur. However, such conditions
Although long-term therapy with antimicrobial agents
may only be attained after healing at the surface has
is indicated, it seldom results in complete recovery,
occurred, so that multiplication and toxin production*

especially when the pericarditis is traumatic in origin. may be delayed. Thus the incubation period may vary Thus Blood and Hutchins (1955) report that only about from a few days to four weeks or more. In idiopathic 50 per cent of cattle with traumatic pericarditis treated tetanus it would appear that the neurotoxin is produced with sulphamezathine and/or penicillin responded in the rumen (Smith & Holdeman, 1968). and were sufficiently recovered for salvage. Surgical From the site of production the neurotoxin reaches drainage of the pericardial sac either by pericardiocentesis or pericardiotomy as described by Horney (1960) trunks. The exact mechanisms of transport are not may also have to be considered. However, it must be known, nor are the means by which the toxin exerts its emphasized that pericardiocentesis offers only temporary influence on the nervous system. Smith & Holdeman (1968) state that the neurotoxin can also reach the There are a number of reports on the successful treat-

central nervous system via lymph and blood.

ment of fibrinous and traumatic pericarditis by pericar-

Individual or small numbers of animals are usually

diotomy (Jennings & McIntyre, 1957; Krishnamurthy et

affected in a herd, but in outbreaks of idiopathic tetanus

al., 1979; Mason, 1979).

many animals may be affected.

Tetanus

Signs

As the incubation period may vary quite considerably

Tetanus has long been recognized as a highly fatal

it is not always possible to relate the onset of clinical

disease of all species of farm livestock. The disease is

disease to specific incidents of injury or surgical inter-

produced by the toxin of Clostridium tetani and is char-ference. The first signs are those of apparent stiffness

acterized by hyperaesthesia, tetany and convulsions.

and reluctance to move, accompanied by muscle

tremors that become more pronounced. Another early

sign in some cattle is prolapse of the third eyelid, which

Aetiology

becomes more prominent with handling of the head.

Clostridium tetani is a spore-bearing organism and is These signs are progressively followed by the appear-considered to be one of the least fastidious clostridia as

ance of a slight but persistent ruminal tympany, elevation of the tail, unsteady gait of the hindlimbs, especially are extremely resistant to environmental conditions.

when turning, and trismus with saliva drooling from the

The organism has two main habitats, namely the soil mouth. Because of inability to adopt the normal urinating posture, urine is retained. Further progression has been suggested that the main reservoir of infection leads to generalized muscular tetany with the adoption for animals is soil. The organism produces a number of a 'rocking horse'-like posture. Attempts at walking of toxins, but the neurotoxin is the one of principal lead to lateral recumbency and an inability to rise. importance.

Tetanic convulsions and opisthotonus soon develop.

The portal of entry is usually through a deep punc-

Initially, the convulsions are triggered by external ture, although in cattle introduction into the genital stimuli, but later these occur spontaneously. The dura-tract at the time of parturition is also important. The tion of fatal disease in young cattle is four to five days, organism may also gain entry into surgical wounds, e.g. but older cattle may survive for up to 10 days. Non-fatal after castration, and may be introduced into muscle cases do occur, but generally these do not progress to during vaccination and other injections. There are the convulsive stage and recovery is slow over a period however outbreaks of disease in cattle in which the of weeks or sometimes months.

organism has not localized in any tissue site but remained within the gastrointestinal tract, and such

Pathology

outbreaks are designated as idiopathic tetanus (Wallis, 1963).

There are no tests available for the diagnosis of tetanus

After gaining entry into the tissue, the organism

in the live animal and any tests undertaken would be

remains localized. Multiplication will only occur if for the purpose of eliminating those conditions that optimal conditions develop at the site of infection. If at produce similar clinical signs. Nor are there any gross the time of initial injury there is sufficient tissue damage or microscopic pathological findings that would confirm

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tetanus, although attempts should be made to identify duration. The recommended drugs (Radostits et al., the site of infection and culture the organism.

2000) are chlorpromazine (0.4 mg/kg body weight intravenously, 1.0 mg/kg body weight intramuscularly) three to four times daily and acetyl promazine (0.05 mg/kg

Diagnosis

body weight), administered twice daily for eight to ten Because of the distinctive clinical signs, classical tetanus days or until the severe clinical signs have disappeared. is seldom confused with other diseases. Clinical hypo- Animals that are treated should be kept in dark quiet magnaemia (p. 255) in calves and cattle (p. 787) is surroundings with ample bedding and sufficient space

accompanied by tetany and convulsions, but there is no
to avoid injuring themselves if convulsions occur.

prolapse of the third eyelid or ruminal tympany, and

On farms where tetanus is a problem, vaccinations
low blood calcium and magnesium levels are diagnos-
should be routinely undertaken (see p. 1005).

tic. Cerebrocortical necrosis (CCN) (polioencephalo-
malacia) (p. 261, 903) may produce a similar clinical
picture, except that again there is no prolapse of the
third eyelid and no ruminal tympany. Also, in CCN the

Leptospirosis (other than *Leptospira*

erythrocyte transketolase activity is decreased and

hardjo infection)

blood pyruvate and lactate levels are increased, and
classical lesions are obvious at necropsy. Some cases of

Aetiology

lead poisoning (pp. 906, 944) may also show similar clin-

There are many different serotypes of *Leptospira*

ical signs, but elevated blood and kidney/liver lead

interrogans present in cattle.

values are diagnostic. Strychnine poisoning is extremely

rare in cattle in Great Britain and is usually associated with the ingestion of earthworms treated with strychnine. Strychnine is used for the destruction of moles,

Aetiology

and can be identified in the abomasal contents. It is used

Many serotypes do occur but probably the most impor-

in other counties, often illegally, to kill wildlife. Bovine

tant are *L. interrogans* serovar *icterohaemorrhagiae* spongiform encephalopathy (p. 909) may also need to

and serovar *canicola*. *Leptospira interrogans* serovar

be differentiated but the signs usually develop over a

pomona has been found in many countries to be the

longer period than tetanus.

commonest infection of farm animals. It has only

recently been found in Britain and the strain appears to

Treatment and prevention

be different from the one causing abortion in pigs. In

many cases there are carrier animals and often these are

Cattle appear to respond better to treatment than

rodents. The leptospires survive in the environment

horses and sheep, although it is unlikely that fully devel-

*if conditions are wet. However, they are inhibited at
open tetanus will respond and therefore under such
a pH less than 6 or greater than 8. Temperatures
circumstances euthanasia should be considered.*

below 7–10°C (44.5–50°F) or above 34–36°C (93–96°F)

When treatment is undertaken it should have three

are detrimental. The organisms can survive over four

*objectives: elimination of *Cl. tetani*, neutralization of months under wet
conditions but only 30 minutes when*

unfixed neurotoxin and the induction and maintenance

the soil is dried. It often survives in the environment in

of muscle relaxation until all the neurotoxin has been

average conditions for about one and a half months.

destroyed or eliminated.

Most infection is spread via contaminated feed or

Large doses of penicillin administered parenterally is

water. It must be remembered that most serovarieties

*the recommended treatment for *Cl. tetani* infection and are able to infect many
species including man.*

this should be supplemented by treatment of the

Mortality rate is usually low at about 5 per cent.

infected site (if located) by irrigation and topical appli-

cation of antibiotics. Thus treatment should continue for at least seven days.

Signs

The administration of antitoxin for the neutralization of unfixed neurotoxin is considered to be of little value

Acute: The cow is often ill with a pyrexia of 40–41°C (104–106°F) with dullness and anorexia. The milk yield drops and often there are haemorrhages under the local injection of the antitoxin may be of value.

mucous membranes, and there is often jaundice and

A number of drugs have been used to relieve muscle haemoglobinuria. In some cases there is synovitis or a tetany, but because of the need for long-term relaxation necrotic dermatitis. Occasionally, meningitis has been some are not suitable because their activity is of short recorded.

Bacterial Conditions • 735

Subacute: In this form the signs are milder with a

Leptospira hardjo infection

temperature of 39–40°C (102–104°F). The animal has a

(flabby bag) (see p. 580)

reduced milk yield and is dull. Jaundice occurs in some

cases and there is usually some haemoglobinuria. In

Aetiology

some cases abortion occurs about a month later.

Infection with *Leptospira borgpetersenii* serovar *hardjo* (*Hardjo bovis*) and *Leptospira interrogans* serovar *Chronic*: This is not common and results in abortion.

hardjo (*Hardjo prajitno*), spiral organisms.

These are a common cause of abortion in Britain,

Necropsy

although they have only recently been readily diag-

nosed. *Leptospira borgpetersenii* sv *hardjo* appears to Acute: There are often submucosal and subserosal

be better adapted to cattle; it is excreted in

haemorrhages, jaundice, anaemia and haemoglobinuria.

large numbers in urine although it is less fre-

In some animals there is ulceration of the abomasal

quently isolated from aborted fetuses whereas *L. inter-*

mucosa and emphysema. Histological examination

rogans sv *hardjo* appears to be less well adapted in often shows an interstitial nephritis as well as cen-cattle (possibly more in sheep), while isolated from

trilobular hepatic necrosis.

*Britain, India, Nigeria, Malaysia and the USA, it has
Subacute or chronic cases are more likely to show a
not been seen in Australia and New Zealand; urine
progressive interstitial nephritis with white foci on the
shedding of organisms is low, but it is more frequently
surface of the renal cortex.*

*found in aborted fetuses and is relatively more
pathogenic.*

Up to 50–60 per cent of British farms may be

Diagnosis

infected. In Northern Ireland 41.6 per cent of randomly

This depends on finding the organism. Otherwise,

*selected aborted fetuses were infected (Ellis et al., 1982) paired serum samples
can be examined for a rise in*

and the level rose to 68.9 per cent of fetuses from farms

Leptospira titres.

with abortion problems. A very common problem in

New Zealand, most cases are in the dairy herd rather

than beef cattle. Humans can be infected but they must

Differential diagnosis

be exposed to concentrated infection, i.e. contact via urine while milking. Although infection is present in Any form of haemoglobinuria will need to be eliminated, including babesiosis (p. 748), which is usually the milk, it quickly dies off once taken from the udder. Meat does not carry infection. Spread occurs seen in the summer in tick areas. Kale poisoning (p. 941) and postparturient haemoglobinuria (p. 792) more rapidly in wet seasons in low-lying areas. Colostrum-derived protection normally lasts about should be partly diagnosed by the history and the low three months. Serological rises in *L. hardjo* titres fol-plasma phosphorus levels in the latter. Bacillary lowing infection tend to be short-lived, i.e. a few months haemoglobinuria (p. 719) will be differentiated by the to a year or so. It was originally considered to be a presence of clostridia. winter disease. However, carriers often stop or reduce excretion on silage. Most infection now occurs in the Treatment summer, often with abortion in the summer or early

autumn. Serology in individual animals is difficult to interpret because the bacteria are in the lumen of the kidney or uterus.

The main therapy is usually antibiotics such as dihydrostreptomycin or the tetracyclines. Treatment should be given as soon as signs develop. Blood transfusion

The spread of infection takes place cow to cow via urine, fetuses and uterine discharge, and from bull to human infection.

often helps. The farmer should be warned of possible cow by infected semen. The source of infection is via carrier cows or infected calves, which may be chronically infected, but it may possibly be spread by con-

Control

taminated water or sheep on the farm.

Separate the infected cow. If it is partly due to rodent build-up, control the rodents. Ensure removal of brackish water and provide good drainage. Vaccination

Signs

could be used, as with *L. interrogans* serovar *canicola* or *icterohaemorrhagiae*, but as cases are usually sporadic. There are two main syndromes, the udder form and

radic this does not tend to be used.

abortion.

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Severe udder form: In a cow or heifer the udder signs diagnosis. It can also be isolated from abortion or post will not be apparent until the animal has calved. This calving discharge. Culture of the bacteria can be under- form occurs soon after the infection enters a herd. taken. Fetal serology can also be used. Antibodies may There is a sudden drop in milk yield affecting all four be detected in post calving discharges and cervical quarters, with pyrexia usually between 40 and 41.5°C mucus. Serology of the dam can help but often antibody (104–107°F). The udder secretion becomes thickened titres may be static or falling by the time of abortion. It and clotted, occasionally it is bloody or it can be yellow is very difficult to detect carrier animals, as up to 25 per and colostrum-like. The udder itself is not swollen or cent are serologically negative. Bulk herd milk samples inflamed but tends to be flaccid. In a six to eight-week can indicate the presence of infection and also give

period, 30–50 per cent of the herd may be infected.

some suggestion of infection levels.

*The condition usually resolves over seven to ten days
(see p. 580).*

Differential diagnosis

Mild udder form: Many cows are infected and show

*Other leptospiral infections (p. 734), salmonellosis
only a slight drop in milk yield.*

*(Chapter 15), foot-and-mouth disease (p. 700), and mas-
titis (Chapter 23) must all be considered in the udder
form. In abortion, (p. 578) salmonellosis, mucosal*

*Abortion: This usually occurs six to twelve weeks after disease, brucellosis,
Neospora carinum infection (pp.*

*the dam is infected. Abortion can occur on its own or
283, 584) and infectious bovine rhinotracheitis are some
be preceded by the milk drop syndrome. Most cases of
common causes to be differentiated.*

*abortion occur in the second half of pregnancy. If infec-
tion occurs late in pregnancy then an infected calf may
be born. There may be some apparent infertility in the*

Treatment

herd (see p. 580).

Large doses of dihydrostreptomycin (25 mg/kg body weight) may help remove the organism and prevent

Necropsy

kidney and liver damage. All cattle due to calve should be vaccinated. In countries where dihydrostreptomycin

Abortion: There are usually no useful macroscopic features in the aborted fetus.

effective.

Diagnosis

Control

Udder form: History helps in diagnosis, in particular

Vaccination is possible with a killed strain (p.1015). The

the sudden onset of the problem. The signs, sudden loss

vaccines available may contain Hardjobovis or Hard-of milk, flaccid udder, are useful. The Californian Milk

joprajitno strains; there is not always cross-protection Test is positive and there is a high milk white cell count.

between the two strains. Hardjoprajitno vaccines may

Identification and culture of the organisms from urine

produce a longer duration of immunity (G.S. Dhaliwal,

(it can occasionally be isolated from milk and blood in pers. comm.). Both vaccines will control abortion and the acute stages) is a definitive diagnosis. Paired serum improve fertility in endemic herds. Vaccination of samples can be used for the complement fixation test, young stock may also produce benefits. Vaccination microscopic agglutination test and plate agglutination involves two initial doses at least four weeks apart. test. High titres over 1/300 indicate recent exposure to If cattle are young when vaccination commences then infection. Microagglutination titres are found from 10 two doses are required after five months of age. An days after infection; their peak and duration depend on annual booster is recommended but two vaccinations the serotype and route of infection. IgM titres appear a year may be required in herds that calve in the first but peak two to three weeks after infection whilst autumn. It does not affect animals that already have IgG levels appear later and peak 12–30 weeks post milk drop syndrome. Vaccination does help prevent infection (G.S. Dhaliwals, pers. com.). Serum and

abortion but infected cows may still excrete bacteria.

vaginal mucus antibody tests are also available.

It is therefore advisable to treat all adult cattle with dihydrostreptomycin before commencing a vaccination

Abortion: Identification of the bacterium in the fetus, programme.

especially in the lungs, kidneys or adrenal glands, by

Any bought-in animals, and especially bulls, should

fluorescent antibody studies, is the main method of

be isolated and treated with antibiotics and vaccine

Bacterial Conditions • 737

before entering the herd. Reducing exposure to poten-

Treatment and control

tially contaminated water supplies and to pasture

High doses of intravenous tetracyclines for at least

grazed by sheep is a sensible management control.

three days can be useful. Otherwise ampicillin, amoxy-

cillin, florfenicol, novobiocin, sulphonamides or poten-

Haemophilus somnus infection

tiated sulphonamides can be administered. Once

disease is diagnosed, extra observation must be under-

(see p. 907)

*taken of in-contact animals. Control is difficult,
although in North America an aluminium hydroxide*

Aetiology

adjuvenated vaccine is now used.

*A Gram-negative small rod-shaped organism. Most
strains of the organism are antigenically similar.*

Pyaemia

Epidemiology

Pyaemia is defined as a clinical or pathological state

*It is seen in North America and Europe. Inapparent
characterized by the formation of multiple secondary
infection occurs more frequently than clinical disease.
abscesses in a number of organs and/or tissues.*

Problems occur more frequently in younger animals.

The primary pyogenic infection may occur in a

*The portal of entry of infections is often via the respira-
number of sites and the formation of metastatic lesions
tory tract although it is also found in the reproductive
in other organs and tissues follows the entry of organ-
and urinary tracts. Disease is probably a septicaemia
isms into the circulation. Small numbers of organisms*

with localization. Single or several cases may occur.

may intermittently gain entry via the lymphatic drainage of the primary lesion, to produce a bacter-

Signs

aemia, followed by localization and the formation of secondary abscesses. More frequently, however, a septic

The peracute form often causes sudden death. The

thrombus is formed within the primary lesion and por-

acute form is often seen as the sleeper syndrome

tions of this become detached to form emboli, which are

or thromboembolic meningoencephalitis (TEME) in

then arrested in the capillary bed of an organ or tissues

growing cattle. The animal is depressed, with closed

to form the metastatic lesions. The most frequent sites

eyes, and recumbent. Usually there is pyrexia (40–42°C,

for secondary abscess formation are the valvular endo-

104–108°F). Nervous signs can be of muscle tremors

cardium, myocardium, lungs and joints, although the

and hyperaesthesia. The animal may be blind with

liver and kidneys may sometimes be involved.

retinal haemorrhage. Other syndromes include synovi-

*In cattle, pyaemia is often associated with septic
tis with initial lameness. Pneumonia is a common
metritis and mastitis, and A. pyogenes is the organism finding, particularly in
calves, and often associated with
most frequently incriminated. Staphylococcal mastitis
pleurisy (see Chapter 17). Abortion can occur and so
may also lead, in some cases, to pyaemia. On occasions,
can chronic bloat.*

*hepatic abscesses and foul in the foot caused by
Fusobacterium necrophorum
may also result in*

*Pathology
pyaemia.*

*Because pyaemia is a form of generalization of a
There may be focal or diffuse cerebral meningitis
primary infection, the clinical signs, diagnosis and treat-
with characteristic haemorrhagic infarcts in the brain.
ment must be considered in relation to the primary
Haemorrhages can occur in other organs. In the joint
infections referred to above and which are discussed in
form the synovial membranes are oedematous with*

the appropriate sections of the text.

petechial haemorrhages. There is often inflammation of the pericardium, peritoneum and pleura which may be serofibrinous or fibrinous. Histologically there is often

References

a vasculitis and thrombosis, often with infarcts and accumulation of neutrophils.

Anthrax

Prognosis

Baliey, W.W. (1953) Anthrax: response by terramycin therapy.

Journal of the American Veterinary Medical Association, 122, Recumbent cattle often do not recover and usually, 305–6.

if there is no response after three days' treatment, the

Campbell, A.D. (1969) Anthrax: A problem of the livestock condition is irreversible.

industry. Veterinary Record, 85, 89–90.

738 • Chapter 44

Greenough, P.R. (1965) Anthrax and antibiotics. Veterinary Muller, J. (1961) Type C botulism in man and animals – inci-Record, 77, 784–5.

dence in cattle and horses. Medlemsblad for den Danske

Hugh-Jones, M.E. & Hussaini, S.N. (1975) Anthrax in England

Dyrlaegeforening, **44**, 547–57.

and Wales 1963–1972. *Veterinary Record*, **97**, 256–61.

Notermans, S., Kozaki, S., Dufrenne, J. & Scothorst, M. van Lincoln, R.E., Walker, J.S., Klein, F. & Haines, B.W. (1964) (1979a) Studies on the persistence of *Clostridium botulinum* Anthrax. *Advances in Veterinary Science*, **9**, 327–68.

on a cattle farm. *Tijdschrift voor Diergeneeskunde*, **104**, Schlingham, A.S., Devlin, H.B., Wright, G.G., Maine, R.J. & 707–12.

Manning, M.C. (1956) Immunising activity of alum pre-

Notermans, S., Kozaki, S. & Scothorst, M. van (1979b) Toxin cipitated antigen of *B. anthracis* in cattle, sheep and production by *Clostridium botulinum* in grass. Applied and swine. *American Journal of Veterinary Research*, **17**, 256–

Environmental Microbiology, **39**, 767–76.

61.

Prevot, A.R. & Sillio, R. (1955) A biological enigma: resist-Sterne, M. (1959) In *Infectious Diseases of Animals*, 1st edn ance of cats to *Clostridium botulinum* toxin. *Annales de* (ed. by A.W. Stableforth & I.A. Galloway), Vol. 1, pp. 16–52.

l’Institut Pasteur, **89**, 354–7.

Butterworth Scientific Publications, London.

Smith, L.D. (1977) *Botulism, the Organisms, its Toxins, the Disease*, pp. 15, 91–6. Charles C. Thomas, Springfield, Illinois.

Bacillary haemoglobinuria

Blood, D.C., Radostits, O.M. & Henderson, J.A. (1983) Veteri-

Clostridial myositis: blackleg and

nary Medicine, 6th end, p. 548. Baillière Tindall, London.

malignant oedema

Roberts, R.S. (1959) In *Infectious Diseases of Animals*, 1st edn Blood, D.C., Radostits, O.M. & Henderson, J.A. (1983)

(ed. by A.W. Stableforth & I.A. Galloway), Vol. 1, p. 200.

Veterinary Medicine, 6th edn, pp. 541–5. Baillière Tindall, Butterworth Scientific Publications, London.

London.

Smith, L.D. (1957) *Clostridial diseases of animals*. Advances Harwood, D.G. (1984) Apparent iatrogenic clostridial myosi-in *Veterinary Science*, **3**, 463–524.

tis in cattle. *Veterinary Record*, **115**, 412.

Smith, L.D. & Holdeman, (1968) *The Pathogenic Anaerobic* Harwood, D.G. (1994) *Clostridial myositis – an old fashioned*

Bacteria, pp. 339–48. Charles C. Thomas, Springfield, Illinois.

disease? *Cattle Practice*, **2**, 323–9.

Williams, B.M. (1964) *Clostridium oedematous infections* Jubb, K.V.F., Kennedy P.C. & Palmer, A. (1983) *Pathology of (black disease and bacillary haemoglobinuria) of cattle in*

Domestic Animals, pp. 180–4. Academic Press, London.

mid Wales. *Veterinary Record*, **76**, 591–6.

Kerry, J.B. (1964) A note on the occurrence of *Clostridium chauveoi* in the spleens and livers of normal cattle. *Veteri-*

Botulism

nary Record, **76**, 396.

Radostits, O.M., Gay, C.C., Blood, D.C. & Hinchcliff, K.W.

Appleyard, W.T. & Mollison, A. (1985) Suspected bovine bot-

(2000) *Veterinary Medicine*, 9th edn, pp. 763–4. W.B.

ulism associated with broiler litter waste. *Veterinary Record*, Saunders, London.

116, 535.

Williams, B.M. (1977) Clostridial myositis in cattle: bacteriol-Breuknik, H.I., Wagenaar, G., Wensing, T., Notermans, S. & ogy and gross pathology. *Veterinary Record*, **100**, 90–1.

Poulos, P.W. (1978) Food poisoning in cattle caused by the

ingestion of brewers grains contaminated by *Clostridium*

Contagious bovine pyelonephritis

botulinum type B. *Tijdschrift voor Diergeneeskunde*, **103**, 303–11.

Ado, P.B. & Cook J.E. (1979) Specific immunofluorescence

Clegg, F.G. & Evans, R.K. (1974) Suspected case of botulism of *Corynebacterium renale*. *British Veterinary Journal*, **135**, in calves. *Veterinary Record*, **95**, 540.

50–4.

Clegg, F.G., Jones, T.O., Smart, J.L. & McMurty, M.J. (1985) Goudswaard, J. & Budhai, S. (1975) Some aspects of the

Bovine botulism associated with broiler litter waste. *Veteri-immunological response in cattle with Corynebacterium*

nary Record, **117**, 22.

renale. Identification of a new serotype, type IV. *Zentralblatt* Davies, A.B., Roberts, T.A. & Bradshaw, P.R. (1974) Probable fur *Veterinarmedizin*, **22B**, 473–9.

botulism in calves. *Veterinary Record*, **94**, 412–14.

Hiramune, T., Invi, S., Murase, N. & Yanagawa, R. (1972)

Egyed, M.N., Shlosberg, A., Klopfer, V., Nobel, T.A. &

Antibody response in cows infected with *Corynebacterium*

Mayer, E. (1978) Mass outbreaks of botulism in ruminants

renale. *Research in Veterinary Science*, **13**, 82–6.

associated with ingestion of feed containing poultry waste:

Hiramune, T., Narita, M., Tomonari, I., Murase, N. &

Clinical findings. *Refuah. Veterinerath*, **35**, 93–9.

Yanagawa, R. (1975) Distribution of *Corynebacterium*

Ektvedt, R. & Hanssen, I. (1974) Outbreak of botulism in

renale among healthy bulls, with special reference to inhabi-cattle. *Norske Veterinaertidsskrift*, **86**, 286–96.

tation of type III in the prepuce. National Institute of

Fjolstad, M. & Kluna, T. (1969) An outbreak of botulism

Animal Health Quarterly, Tokyo, **15**, 116–21.

among ruminants in connection with ensilage feeding.

Morse, E.V. (1950) An ecological study of *Corynebacterium*

Nordisk Veterinarmedicin, **21**, 609–13.

renale. *Cornell Veterinarian*, **40**, 178–87.

Haagsma, J. & Laak, E.A. (1977) Type B botulism in cattle fed Takai, S.,

Yanagawa, R. & Kitamura, Y. (1980) pH dependent grass silage. Report of an outbreak. *Tijdschrift voor Dier-adhesion of piliated Corynebacterium renale to bovine geneeskunde*, **102**, 330.

bladder cells. *Infection and Immunity*, **28**, 669–74.

Bacterial Conditions • 739

Endocarditis

Infectious necrotic hepatitis

Andersen, H.K. (1963) Investigations on pathogenesis, the

Bagadi, H.O. & Sewell, M.M.H. (1973) An epidemiological

aetiology and topography of endocarditis in cattle. Nordisk survey of infectious necrotic hepatitis of sheep (black

Veterinarmedicin, **15**, 668–90.

disease) in Scotland. *Research in Veterinary Science*, **15**, Biering-Sørensen, V. (1963) Incidence of endocarditis in cattle 49–53.

and its seasonal variation. *Nordisk Veterinarmedicin*, **15**, Bagadi, H.O. & Sewell, M.M.H. (1974) Influence of post

691–5.

mortem autolysis on the diagnosis of infectious necrotic

Evans, E.T.R. (1957) Bacterial endocarditis in cattle. Veteri-hepatitis (black disease). *Research in Veterinary Science*, **17**, nary Record, **69**, 1196–1202.

320–7.

John, F.V. (1947) Verrucose endocarditis. *Veterinary Record*, Batty, I., Buntain, D. & Walker, P.D. (1964) *Clostridium oede-59*, 214.

matiens, a cause of sudden death in sheep, cattle and pigs.

Lacuta, A.Q., Yamada, H., Nakamura, Y. & Hirose, T. (1980) *Veterinary Record*, **76**, 115–17.

Electrographic and echocardiographic findings in four cases

Herbert, T.G.G. & Hughes, L.E. (1956) Black disease (infec-of bovine endocarditis. *Journal of the American Veterinary tious necrotic hepatitis) in a heifer. Veterinary Record*, **68**, Medical Association, **176**, 1353–65.

223–4.

Larsen, H.R. (1963) *Clinical observations on endocarditis in*

Williams, B.M. (1964) *Clostridium oedematiens infections cattle. Nordisk Veterinarmedicin*, **15**, 645–67.

(black disease and bacillary haemoglobinuria) of cattle in

Power, H.T. & Rebhun, W.C. (1983) *Bacterial endocarditis in mid-Wales. Veterinary Record*, **76**, 591–6.

cattle. *Journal of the American Veterinary Medical Associa-*Williams, B.M. (1976) *Infectious necrotic hepatitis of sheep:*

tion, **182**, 806–8.

An epidemiological survey on Welsh farms. British Veteri-

Wuijckhuise-Sjouke, L.A. van (1984) *Plasma fibrinogen con-*
nary Journal, **132**, 221–5.

centration as an indicator of the presence and severity of

inflammatory disease in horses and cattle. Tijdschrift voor Diergeneeskunde, **109**, 869–72.

Pericarditis

Blood, D.C. & Hutchins, D.R. (1995) *Traumatic pericarditis of*

Haemorrhagic septicaemia

cattle. Australian Veterinary Journal, **31**, 229–32.

Horney, F.D. (1960) *Surgical drainage of the bovine pericar-*

Bain, R.V.S. (1963) Haemorrhagic septicaemia, Agricultural dial sac. *Canadian Veterinary Journal*, **1**, 363–5.

Studies No. 62. Food and Agricultural Organization, Rome.

Jennings, S. & McIntyre, W.M. (1957) Pericardiectomy in a

Carter, G.R. (1982) Whatever happened to haemorrhagic septic-
cow. *Veterinary Record*, **69**, 928.

ticaemia. *Journal of the American Veterinary Medical Asso-*

Krishnamurthy, D., Nigam, J.M., Peshin, P.K. & Kharole, M.U.

ciation, **180**, 1176–7.

(1979) Thorapericardiotomy and pericardiotomy in cattle.

Carter, G.R. & Bain, R.V.S. (1960) Pasteurellosis (*Pasteurella* *Journal of the*
American Veterinary Medical Association, **175**, multocida). A review stressing
recent developments. *Veteri-*714–18.

nary Review Annotations, **6**, 105–28.

Mason, T.A. (1979) Suppurative pericarditis treated by peri-

De Alwis, M.C.L. & Carter, G.R. (1980) Preliminary field trials cardiotomy in a
cow. *Veterinary Record*, **105**, 305–1.

with streptomycin dependent vaccine against haemorrhagic

septicaemia. *Veterinary Record*, **68**, 223–4.

Jubb, K.V.F., Kennedy, P.C. & Palmer, N. (1985) Pathology

Tetanus

of Domestic Animals, 3rd edn, Vol. 2, pp. 488–9. Academic Radostits, O.M., Gay,
C.C., Blood, D.C. & Hinchcliff, K.W.

Press, London.

(2000) *Veterinary Medicine*, 9th edn, pp. 754–7. W.B.

Macadam, I. (1962) *Tick transmission of bovine pasteurellosis*.

Saunders, London.

Veterinary Record, **74**, 689–90.

Smith, L.D. & Holdeman, L.V. (1968) *The Pathogenic Anaer-Mustafa*, A.A., Ghalib, H.W. & Shigidi, M.J. (1978) *Carrier obic Bacteria*, pp. 256–81. Charles C. Thomas, Springfield, rate of *Past. multocida* in cattle herds associated with an Illinois.

outbreak of haemorrhagic septicaemia in Sudan. *British*

Wallis, A.S. (1963) *Some observations on the epidemiology of*

Veterinary Journal, **134**, 375–8.

tetanus in cattle, *Veterinary Record*, **75**, 188–91.

Sawada, T., Rimler, R.B. & Rhoades, K.R. (1985) *Haemor-*

rhagic septicaemia: naturally acquired antibodies against

Past. multocida types B and E in calves in the United States.

***Leptospira hardjo* infection**

American Journal of Veterinary Research, **46**, 1247–50.

Shirlaw, J.F. (1938) *Haemorrhagic septicaemia: A criticism of*

Ellis, W.A., O'Brien, J.J., Neill, S.D., Ferguson, M.W. & Hanna, the present position with an account of investigations into

J. (1982) *Bovine leptospirosis: Microbial and serologi-*

the problem in the Northumbrian area. Veterinary Record, cal findings in

aborted fetuses. *Veterinary Record*, **110**, 50, 1005–9.

147–50.

Wei, B.D. & Carter, G.R. (1978) Live streptomycin dependent *Past. multocida* vaccine for the prevention of haemorrhagic septicaemia. *American Journal of Veterinary Research*, **39**, 1534–7.

Chapter 45

Ectoparasites, Tick and Arthropod-

borne Diseases

S.M. Taylor, A.G. Hunter and A.H. Andrews

ECTOPARASITES

740

Other problems

775

Insects

740

Tick paralysis

775

Hypodermatosis (warbles)

740

Sweating sickness

776

Lice (pediculosis)

741

Mhlosimge

777

Arachnids

742

Magudu

777

Mange

742

Stomatitis–nephrosis syndrome

777

Tick infestations

744

Ixodoidea

744

Fly problems

745

Mosquitoes

745

ECTOPARASITES

Blackflies

745

Midges

745

Insects

Horseflies and deerflies

745

Houseflies

746

Hypodermatosis (warbles) (see p. 875)

Bush flies

746

Face fly

746

Hypodermatosis is the term used to describe infection

Head fly

746

with and the lesions caused by the larvae of two species

Stable fly

746

of the fly genus Hypoderma, H. bovis and H. lineatum.

Horn flies and buffalo flies

747

The disease is characterized by damage to dorsal hides,

Horse louse flies

747

oesophagus, central nervous system and occasionally

Tsetse flies

747

death due to anaphylaxis (see p. 927).

Tumbu fly

747

Blow flies and screw-worm flies

747

TICK AND ARTHROPOD-BORNE DISEASES

748

Aetiology and epidemiology

Protozoal diseases

748

Babesiosis (redwater)

748

The adult flies are distributed in the northern hemi-

Theileriosis

750

sphere, excluding the most northerly arctic regions.

Besnoitiosis

756

There are two species, H. bovis and H. lineatum. Hypo-Trypanosomosis

756

derma bovis favours the northern parts of their habitat Rickettsial diseases

761

areas and H. Lineatum the southern and warmer areas,

Anaplasmosis

761

but both species can occur simultaneously. The adult

Tick-borne fever

763

Bovine petechial fever (Ondiri disease, ondiritis)

763

flies have a yellow abdomen characterized by a broad

Heartwater (cowdriosis or malignant rickettsia,

stripe of black hairs. They are active in warm weather,

blacklung)

765

generally in Europe between the months of June and

Jembrana disease (Tabana disease)

766

August. Their presence upsets cattle, which run to avoid

Viral diseases

767

them and in doing so injure themselves on fences or

Bovine ephemeral fever (BEF)

767

other obstacles. The females of H. bovis deposit eggs Lumpy skin disease (LSD)

768

singly on hairs, frequently above the hocks on the hind

Rift Valley fever

769

legs; the eggs of H. lineatum are laid in rows, whence its Tick-borne encephalitides (flavivirus infections)

770

name was derived, usually on the lower part of the body

Tick-borne encephalitides (Near East encephalitis)

771

and in places where both adult and eggs are difficult for

Japanese encephalitis (flavivirus)

771

cattle to dislodge. The eggs adhere strongly to the hairs.

Other arthropod-borne diseases

771

Parasitic diseases

774

They hatch in four to six days and the first stage larvae

Onchocercosis (worm nodule disease)

774

crawl down to the skin, which they penetrate, and

Stephanofilariosis

774

proceed to migrate through soft tissues towards their

Thelaziosis

775

overwintering sites. This movement takes several

740

Ectoparasites, Tick and Arthropod-borne Diseases • 741

weeks, and by late autumn H. bovis has reached the

Treatment

epidural fat within the vertebral column and H.

The larvae are susceptible during their migration to lineatum the submucosa of the oesophagus. The larvae systemic organophosphorus insecticides and the remain in these sites during the winter, moulting to their anthelmintics ivermectin, doramectin, eprinomectin, second stage of development. At the end of the winter abamectin and moxidectin. Care should be taken not to they resume migration both reaching the subdermal treat cattle when larvae are in their overwintering sites, tissue along the back in the spring. They make a hole in as death in situ and subsequent lysis may result in the overlying skin through which they respire while anaphylaxis. For that reason statutory eradication po-developing to the third stage, which reaches a length of licies have compulsory treatments applied before mid 2–3 cm and is characterized by rows of small spines on November, coupled where necessary with inspection the posterior margin of most of its segments and pos-and further treatments in the following spring. terior spiracles on a terminal tuberosity. The creamy-

The organophosphorus treatments are normally white larvae emerge from the back of the animal in applied dermally as pour-on preparations. A large April and May and fall to the ground to continue development by pupation, eventually emerging as adults phosmet and crufomate. Mass treatment has resulted five weeks later. They have a short lifespan as adults, in eradication from many islands although it has mating, laying eggs and dying within two weeks of proved more difficult on large land areas. Avermectins, emergence.

although highly effective, are not normally used in mass eradication campaigns since their primary use is as an Pathogenesis

anthelmintic, although if a single animal in a herd is found to be infected the entire herd is usually treated The larvae are most pathogenic during two phases of with them. Although prophylaxis against adult flies is their development. The first is during the late autumn not actively undertaken, slow-release devices such as

and winter, when they are in epidural fat or ear tags impregnated with organophosphorus compounds or oesophageal wall respectively. If present in large numbers, or if treated with synthetic pyrethroids, the possibility of ovi-position by adults. reactions may cause damage to the spinal cord that can result in either spinal paralysis or difficulty in swallowing and eructation, which can lead to bloat. The second

Lice (pediculosis)

is during their pre-emergence development, where they damage the subdermal tissue and skin of the back and neck. Lice are ubiquitous parasites of cattle. In large numbers they cause subsequent downgrading of hides and trimming of carcasses, and cause irritation and loss of growth in anaemia and may act as vectors of pathogenic organisms (see p. 880).

isms (see p. 880).

Signs

Aetiology and epidemiology

In the late autumn and winter, occasionally there can be

Lice are host-specific parasites and are classified into damage to the spinal cord resulting in paralysis, difficulty two types: (i) sucking lice and (ii) biting lice. Five in standing or bloat. In the spring, there are swellings species infest cattle, four sucking and one biting. under the skin 55–75 cm either side of the midline

The former are Haematopinus eurysternus and H. containing pus, a breathing hole and a third stage larva.

quadripertusus, ‘short-nosed’ sucking lice; Lignognathus vituli, ‘long-nosed’ and Solenopotes capillatus,

‘small blue’ sucking lice. The biting species is Damalinia Diagnosis

bovis. As might be construed the sucking lice have

If disease results during their winter development, the

mouthparts adapted for piercing skin and sucking

symptoms have to be differentiated from other causes

blood; the biting lice ingest skin and hair detritus, blood

of spinal or oesophageal paralysis. There is an ELISA

and scabs. Each species has a preferential area on

test available that detects antibody to migrating larvae

animals, but if large numbers are present can be found

and, although originally designed to aid eradication

anywhere on the body surface. Normally, *H. euryster-*
programmes, it may be helpful in diagnosis. When
nus and *D. bovis* are found on the poll, neck and head larvae are in their pre-
emergence sites on the back their
and the latter also along the mid dorsal region of the
presence is unmistakable since no other condition
body. *Lignognathus vituli* and *S. capillatus* also favour causes similar lesions,
especially if a heavy infection is
the head, neck and frequently the ventral surface of the
present.

neck, dewlap and axillae. *Haematopinus quadripertusus*

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is restricted to subtropical areas and is usually found in
pyrethroids is generally of longer duration. This aspect
the area of the tail and posterior lumbar regions.

is important because for complete removal of all lice

The life cycle of lice is direct. Adults live for approxi-
the activity must not only kill adults but nymphs emerg-
mately one month during which time they lay a few
ing from eggs. The anthelmintics ivermectin, dora-
hundred eggs, which are tightly attached to hair by a
mectin, eprinomectin and abamectin because of their

glue-like substance produced by the louse. The eggs long half-life are also extremely effective, removing all hatch within a few days and the first nymph, which sucking and more than 98 per cent of biting lice. If used resembles a small softer adult, emerges. It undergoes as a pour-on preparation they are even more effective, three moults to second and third stage nymphs and and will remove 100 per cent of both species. For pre-finally adults, each nymphal stage taking one week. The ventative purposes, housed cattle can be treated at entire life cycle from egg to adult occupies approxi-housing, sometimes complemented by removal by elec-mately three weeks.

tric clippers of a 15 cm band of hair from the poll to the There are seasonal variations, especially marked in root of the tail.

temperate areas, in the number of lice found on cattle. In these areas numbers on susceptible cattle increase during autumn and early winter, peaking late winter

Arachnids

and early spring. There are two reasons for this: (i)

cattle are frequently housed in autumn and lice are

Mange

easily transferred from infested to non-infested

Mange is the descriptive term used for infection of animals, and (ii) climatic conditions favour louse activity; strong sunlight and high skin temperatures have been shown to inhibit lice and the converse occurs in infected non-infected animals by direct contact. There are four species that parasitize cattle and two others those that carry heavier burdens than others, and it is which are facultative parasites of them and other generally accepted that very large louse populations are animals (See p. 881).

indicative of stress or intercurrent illness, poorly fed cattle parasitized by nematodes and trematodes being frequently and characteristically louse infested.

Aetiology and epidemiology

The pathogenic effects of louse infestation have been

Although of the same order, the mites have different the subject of much research. It is now considered that morphologies and habitats, and some are classified as light or moderate infections have little effect on cattle. 'burrowing' and others as 'non-burrowing'. For these Heavy infestations lead to skin irritation, scratching and reasons they are described separately.

rubbing, with resultant damage to hides. Some reports have indicated that large sucking lice burdens may also

Non-burrowing mites: One species, Chorioptes bovis, is cause anaemia and weight loss, although it has also been

specific to cattle. Psoroptes ovis, although primarily a noted that these effects can be prevented by adequate

parasite of sheep, can become permanently adapted to

high quality nutrition. Lice may also act as vectors for

cattle. Non-specific mites are those species of harvest

blood-borne organisms, and have been implicated in

mites present on pasture, the larvae of which can infest

the transmission of Eperythrozoon wenyonii.

many different species of animals: the mite species are

Neotrombicula and Eutrombicula.

Signs and diagnosis

(1)

Chorioptes bovis. The most common mite found on cattle. Louse infestations are usually evident by the presence of eggs adhering to hairs, on the edge of bald areas and nizable by its cup-shaped suckers on short unjointed on parting of hair careful examination of preferential pedicels. It is seen most frequently during autumn and niches with a magnifying lens will reveal adults and winter in housed cattle, frequently on the hind legs but nymphs.

also on the neck, the head and root of the tail. It is a surface feeder and produces mild hair loss, lesions

Treatment and control (see p. 1030)

increasing in size only slowly. The lesions are obviously itchy as affected cattle will rub affected areas on walls, Lice are fairly easily killed by the application of doors, etc., incurring damage to the skin and subsequent organophosphorus, amitraz or synthetic pyrethroid quality of the hide after death.

preparations, either as sprays, pour-ons or dusting

powders, although resistance to the last of these has

(2)

Psoroptes ovis. The mite is normally found on

been reported. The residual activity of synthetic

sheep and is the cause of sheep scab, which is notifiable

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and government regulated in many countries. The mite

found in other areas. Early small lesions exhibit hair

is a similar size to C. bovis but is characterized by thinning and slightly thickened scaly skin, but soon the

long jointed pedicels with funnel-shaped suckers on the

irritation and resultant rubbing combined with expan-

legs. Although similar to P. ovis from sheep, which is sion of infected areas result in total loss of hair and

transferrable experimentally both to cattle and rabbits,

thickened, crusted and excoriated skin. Affected

the rarity of cases of mites transferring from sheep to

animals may become so preoccupied with the irritation

cattle and vice versa in nature has led to the conclusion

that they reduce their food intake, leading to loss of

that some adaptation to specific hosts takes place,

weight or milk production, and secondary bacterial

and that infection of sheep by cattle strains does not infections may ensue on the most severely rubbed occur.

lesion.

The mite, although a surface feeder, is much more

(2)

Demodex bovis. This mite is considered to be a

irritant than C. bovis since its mouthparts are adapted normal commensal found on bovine skin; as for all

for piercing skin, causing the formation of serious vesi-

*members of the family Demodicidae it becomes path-
cles, which can coalesce and eventually become scabs.*

ogenic only when the efficiency of the immune controls

The life cycle is typical for both P. ovis and C. bovis.

of its host is reduced. The mites are characteristically

*Female mites lay approximately 90 eggs. The hatching
cigar-shaped, 0.2 mm long, with four pairs of short legs*

of eggs and subsequent development from larva to

close to the anterior end. Their preferred niches are in

nymph to adult occupies approximately 10 days. Adult

hair follicles and sebaceous glands and because of this

mites can live for a maximum of six weeks.

deep location are not transmitted unless prolonged
Affected cattle become restless because of the skin
contact occurs. For this reason it is presumed that the
irritation. The areas commonly affected are the
young become infected shortly after birth during
abdomen, tail root and perineum, and they can become
feeding from their mother, and as a result lesions are
further damaged by scratching. In extreme cases
usually seen in cattle on the muzzle, head, neck and
animals may cease to feed adequately and lose weight.
back. The lesions take the form of small, 0.75 cm diam-
eter nodules and are follicles or glands filled with mites
(3)

Forage mites. The adults and nymphs of the
and caseous pus. In some countries a high proportion
family Trombiculidae are free-living on pasture. Eggs
of hides may be affected, but in the UK the average
are laid on soil, and larvae crawl on to vegetation and
prevalence is 17 per cent (Urquhart et al. , 1987). The to animals that lie on or
brush through the foliage. The

lesions do not normally seriously affect cattle and larvae are skin-piercers, causing vesicles. Animals treatment is not usually necessary.

become hypersensitive to their secretions and the subsequent rubbing and scratching increases the damage to skins.

Diagnosis

All of the mites are too small to be easily seen with the

Burrowing mites:

naked eye, and microscopic examination of skin scrap-
(1)

Sarcoptes scabiei. Mites of this species are host
ings is necessary, as it is for differentiation of most skin
specific although they are morphologically identical to
conditions. Mites are normally most easily found in
those infesting other animal species. The adults are
scrapings taken from the edge of lesions. The scrapings
slightly larger than half the size of P. ovis and C. bovis.
are boiled in sodium or potassium hydroxide and
They are characterized by a rounded shape and the
centrifuged prior to examination of the sediment by

presence of triangular scales on the posterior of the microscope.

dorsum. Adult females burrow tunnels in the epidermis.

They lay eggs in tunnels, hatching taking place within a week. The hatched larvae crawl to the surface and

Treatment and control (see p. 1030)

create further epidermal tunnels in which they moult

Mites are affected by amitraz, some organophosphorus

through the stage of nymphs to become adults. Males

compounds, synthetic pyrethroids and the anthelmintics

and females mate either in tunnels or on the skin

ivermectin, doramectin, eprinomectin and moxidectin.

surface and the cycle resumes, the entire length of which

For topical applications the problem is to ensure that occupies three weeks.

the chemical can come into contact with the parasite

Sarcoptes scabiei infections cause extreme irritation.

and it may be necessary to remove scabs before appli-

On cattle the preferred sites are the neck and the

cation. The organophosphorus compounds used fre-

lumbar area adjacent to the tail, resulting in the collo-

quently are phosmet, propetamphos and diazinon, and
quial description of 'neck and tail mange', but it can be
the synthetic pyrethroids flumethrin, deltamethrin and

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cypermethrin. Organophosphorus compounds and

There are three genera: *Argas*, *Ornithodoros* and ivermectin, doramectin and
moxidectin should not be

Otobius.

used on dairy cows giving milk for human consumption
unless withdrawal periods are observed. Eprinomectin
is safe to use in such cattle.

Ixodidae family

These have a hardened scutum that covers almost all
the dorsum in adult males but only one-third of the

Tick infestations (see p. 1030)

dorsum in unfed larvae, nymphs and adult females. The
mouthparts are visible. There are nymph and larval

In tropical regions ticks play an important role in
stages. All stages including the adult females feed only
causing and spreading disease. Many actively suck
once. Adult males remain on the host and feed often.

blood and can cause death by anaemia. Some ticks

Up to half the engorged weight can be taken in with

*cause paralysis, particularly in young animals. The life
one feed.*

cycles vary considerably, with some ticks spending all

The family includes one-, two- and three-host ticks

their time on a single host, others are only parasitic at

and there are ten ixodid genera of which seven have

certain stages and some spend each stage of the life

veterinary importance:

Amblyomma,

Boophilus,

cycle on a different host. Those spending all their time

*Dermacentor, Haemaphysalis, Hyalomma, Ixodes and on a single host (one-host
ticks) are easier to control*

Rhipicephalus.

than those on different hosts for each development

stage (three-host ticks)

Control: This can be directed at the ticks on or off the One-host ticks:

Boophilus spp.

host. The latter is easiest to perform, but is ecologically

Two-host ticks:

Hyalomma spp.

damaging as other non-dangerous insects are also

Rhipicephalus bursa

affected. When off-the-host-control is performed it

R. evertsi

includes the use of pasture spraying with anti-tick dips,

Three-host ticks:

Amblyomma spp.

which involves keeping stock off the area for as long as

Argas persicus spp.

possible to allow death of the ticks. It works best where

Dermacentor spp.

the ticks are short-lived as in warm arid conditions. It

Haemaphysalis spp.

is best performed by treating pastures in rotation. The

Hyalomma spp.

effectiveness of pasture spraying depends on whether

Ixodes spp.

alternative hosts, either domestic or wild, are available.

Rhipicephalus spp.

The practice often means underuse of pasture. However, the overuse or intensification in grazing often increases the tick population. Another method of con-

Ixodoidea

trol often under-rated is that of pasture burning.

In most areas the only effective method of tick

Their life cycle involves egg, larva, nymph and adult.

control is on the host. This is usually done by hand

Almost all ticks at each development stage require a dressing, spraying or dipping, and in some countries by blood meal. The unfed stage varies in its ability to vaccination. Hand dressing is laborious and has to be survive depending on the amount of moisture present. done efficiently. It can be done by the use of dusting The engorged stage drops off the host and then does powders, creams, pastes or in a liquid form by hand not move from where it is deposited. The group is spraying, paint brushing or application by washes. In divided into two main families, the Argasidae ticks and large herds sprays or dips are used. In general, dips the Ixodidae or hard ticks.

provide a more effective overall control. Both effective spray races and dips are expensive to install. The compounds used tend to be organophosphates, synthetic

Argasidae family

pyrethroids and traditional compounds. The last group

These have a cuticle without a hardened dorsal scutum

includes arsenic preparations such as sodium arsenite.

and their mouthparts cannot be seen from above. They

It is cheap but has no residual effect and it can be toxic.

are mainly found in arid or semi-arid areas. When

Resistance to arsenic dips occurs in some areas. Or-

feeding they rapidly engorge with blood. They are able

ganophosphorus dips are effective but have limited re-

to survive for long periods, i.e. months or years, without

sidual activity. They can be toxic, especially when used

the presence of suitable hosts for feeding. The nymphs

by unskilled staff. Organochloride compounds are now

and adults can repeatedly feed, which allows greater

prohibited in most countries because they are persist-

capacity for transfer of infection.

ent for long periods and affect the human food chain.

Synthetic pyrethroids are good and relatively non-toxic used. Otherwise, for small groups of animals the use of but some resistance to them is developing, and occurs mosquito screens will stop contact.

also when ticks are DDT resistant.

The number of dippings depends on several factors

Blackflies

and often means the interval between dippings varies according to the season of the year. It depends on the

These belong to the family Simuliidae and include

duration of the ixodicide's activity, the toxicity of the

blackflies, buffalo flies and sandflies. They are small

dip, the seasonal activity of the ticks, the time the ticks

(<5 mm long) and black or grey in colour. They occur

spend on the host and whether it is required to control

in most countries and when in large numbers they are

or try to eradicate the parasite. Thus one-host ticks,

an annoyance. It is thought that there may be toxic

which remain on the host for three weeks, are far easier

factors in the saliva. The bites result in vesicles and

to deal with than three-host ticks, which only feed for a day or two. Generally, at its height, dipping will be once a week for one-host ticks and twice a week for three-host ticks. Such frequent dipping does allow tick selection for resistance. However, often where tick control is not achieved it is more likely due to not dipping frequently enough or the dip being too weak, improperly mixed or the wrong type or concentration for the stage. Occasionally they stampede. Cattle may wallow in mud of the tick to be killed. Where resistance does occur it or kick up dust to keep off the flies. Control depends on attacking the larval stages by spraying breeding dip. Many farmers alternate the type of dip used on a

sites with insecticide. However, rapid reinfection of the routine basis.

areas will occur. Treatment of the adult is difficult but

Vaccination is carried out in Australia and a few other a repellent such as dimethyl phthalate is helpful.

countries to the one host tick *Boophilus microplus*. The Regular dipping of cattle for ticks, etc. also reduces fly

vaccine consists of recombinant antigens to the levels.

microvilli of the tick intestinal epithelium. As a result ingestion of blood from immunized cattle disrupts the

Midges

digestion of the tick which leads to death or reduced fertility. After some years of vaccination the local tick

These are extremely small flies (1–3 mm long) of the population is therefore drastically reduced. Vaccination

family *Ceratopogonidae*, the important genera being needs to be repeated at least annually as the immunity

Culicoides and *Lasiohelia*. These suck the blood of is not boosted by antigens from natural infection.

animals and man. They transmit ephemeral fever, blue-tongue and other pathogenic viruses. The larvae are

found mainly in swamps, where they live either in

Fly problems

wet mud or free-standing water. Some occupy more

(see p. 1030)

restricted habitats such as rotting vegetation. Besides

acting as vectors of arboviruses they can cause hyper-

Mosquitoes

sensitivity reactions.

There are many species of mosquito including Aedes

Control is by draining the breeding sites. Otherwise,

spp., Anopheles spp., Culex spp., Mansonia spp. and spraying the areas with oil or DDT gives good results.

Psorophora spp. They can cause problems when in large numbers due to annoyance of man and animal.

Mos-

tive on a short-term basis. Mosquito screens are of no

quitoes cause some loss of blood by feeding as well as

use. Fires at times of biting activity will assist in herd

transmitting diseases such as Rift Valley fever and prob-

tection.

ably other conditions such as lumpy skin disease and

bluetongue. The mosquito life cycle is aquatic for the

Horseflies and deerflies

larva and pupa and takes 5–21 days to reach the adult stage.

Horseflies are of the Tabanus spp. and are also called Control of the insect over large areas involves

March flies or breeze flies. Deerflies are of the Chrysops drainage of still surface water or by treating water with

spp., Haematopota spp. and Pangonia spp. They are oil, synthetic pyrethroid or other insecticide. Oil is

large, brown, robust flies that bite and suck blood. They

easiest to use and can involve waste engine oil with

act as vectors of diseases such as anthrax, anaplasmosis

kerosene or diesel oil. Applying repellents to cattle

and trypanosomiasis. Haematopota spp. are thought to

tends to be too expensive and self-applicators are often

help transmit summer mastitis. The eggs are laid on the

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leaves of plants growing close to or in water. The larval

A repellent such as dimethyl phthalate can be of use.

and pupal stages occur in the water or mud and the life

Synthetic pyrethroids can be successful.

cycle takes about four to five months to complete. The

flies tend to be active during the day in summer months,

Face fly

particularly when it is hot and sultry, and are mainly found on the ventral abdomen and legs. Control is dif-

This is Musca autumnalis a small fly resembling, and difficult but depends on drainage of wet areas. Breeding slightly bigger than, the housefly, found in Europe, Asia sites can be treated with oil or insecticide. Otherwise, and North America. Numbers tend to be greatest in the repellents such as dimethyl phthalate and g-dimethyl summer and particularly cattle outside are worried.

tolumide are useful but last only a few days. Some Fresh cattle faeces are the only fly breeding grounds. products are available to apply to the cows' udders.

Flies are particularly seen on the face around the nostrils and eyes where they feed on the secretions. They are thought to transmit infectious bovine keratocon-

Houseflies

conjunctivitis. There are no wholly successful control measures although plastic insecticide-impregnated ear

The common housefly (Musca domestica) has a world-

tags are useful and powders or cream containing wide distribution. They cause worry to animals by set-organophosphorus compounds assist.

ting on them as well as acting as vectors of diseases such as anthrax, erysipelas, brucellosis and possibly summer mastitis. They cause aggravation at wounds or

Head fly

other areas where there is blood, exudate, pus, etc. They

The fly *Hydrotaea irritans* is the same size as the house-fly but discharges some of their stomach

contents, so-called vomit drop, on to the food to

found in Great Britain and Europe. It is a nuisance fly

moisten it and often they also defaecate. This means

and does not bite although it does feed off exudate

that they are able to transfer many pathogens effec-

around wounds. It occurs in large swarms from July

tively. Eggs are laid on rotting vegetable material or

to September. The life cycle is annual and it involves

faeces. The life cycle involves an egg producing a larva

periods of development in the soil. Sores on animals are

(a maggot), which takes about 10 days to mature, made larger due to self-inflicted aggravation because thereby completing the cycle in about 12–14 days in of irritation. *Hydrotaea irritans* is incriminated in the warm weather. transmission of summer mastitis (see Chapter 24). It is Control methods include removing all manure and probably also concerned with infectious bovine kerato-organic material at least every three days. This can be conjunctivitis. Control is difficult and involves the use stacked so as to ferment, ideally in a bunker or pit. Oth- of parasite sprays and dipping. Plastic insecticide-erwise, it should be turned over every few days or better impregnated ear tags are useful.

still treated with insecticide. Faeces can be burnt. Fly traps (Baber's) can be used to collect flies and larvae.

Buildings should be kept as clean as possible and can

Stable fly

be sprayed with insecticides, or insecticide strips can be

The stable fly, *Stomoxys calcitrans*, is about the size of placed in areas with little air movement. Electrocutors

the housefly. It is grey in colour, has a sharp proboscis

can be used to which the flies are attracted by an and when it settles it sits with its head upwards. It is a ultraviolet light.

Cattle can have insecticide-bloodsucker and feeds on the host causing great irritation. Wounds often bleed freely after the flies have fed. impregnated ear tags but these are not too effective against the housefly.

The eggs are laid in faeces, rotting hay or straw and the life cycle is complete in two to three weeks. The larval and pupal stages take place in organic matter. Warm

Bush flies

damp environments encourage the flies' growth and

These include *Musca sorbens*, *M. fergusonii*, *M. ter-surround*. They cause considerable nuisance and worry

raeregina and *M. hilli*. They are found particularly in with reduced milk and meat production and possibly

Australia. They tend to be found all the year round in anaemia. In cattle there can be a hypersensitivity of the northern Australia but only in the summer periods in forelimb skin, which in turn has blisters that coalesce to the south. The flies often appear in large numbers and

form bleeding sores. *Stomoxys calcitrans* can transmit can be found on the lips, eyelids and other mucous

anthrax. Another species, *S. nigra*, occurs in South membranes and by wounds. They are considered to Africa.

transmit infectious bovine keratoconjunctivitis. Control

Control of the fly involves the frequent removal and is difficult but involves fly spraying cattle and buildings.

disposal of bedding and faeces from buildings. Destruc-

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tion of the flies is difficult because they feed for only a

of environments but are usually found in one of three

short time. It can be helped by the use of insecticide-

terrains: (i) forest, where they inhabit thickly wooded

impregnated plastic ear tags or spraying with suitable

areas with a high humidity; (ii) riverine, where they live

sprays, spraying of walls and shaded areas of pens.

on the edges of forest but by streams, lakes and rivers;

and (iii) savannah grasslands, where the tsetse are the

most important species for spreading animal disease.

Horn flies and buffalo flies

Both the male and female tsetse flies feed on blood

*Horn flies include *Liperosia* or *Haematobia irritans*, *H.**

and most of the species are attracted to certain host

*minuta and *L.* or *H. exigua*. This last is also known as species. In tsetse flies the eggs hatch in the uterus and*

the buffalo fly. The flies are greyish, smaller and less

so they are larviparous. The larvae are deposited on

active than stable flies. At rest their wings are com-

shady dry soil and pupate there. One larva is produced

pletely closed but are held away from the body. They

every 10 days and the pupal stage lasts 28–56 days. As

thus spend more time on the host except when laying

the adult female survives three or four months eight to

eggs, etc. Many have a limited geographical range. Thus

twelve larvae are produced.

**H. irritans* is found in the USA and Hawaii, *H. minuta* Control depends on surveying an area to determine*

*in Africa and *H. exigua* in Australia and south-west*

the species of the tsetse fly present and their habitats.

Asia. They are most common on cattle and buffalo

Then the possibility of control can be assessed. Methods

and occur in large numbers, causing much irritation.

of eradication or control are direct using insecticides to Although bloodsuckers they are not known to transmit remove adult flies, removal of pupae and use of repellents on cattle. The indirect approach depends on death. They also produce large sores.

removal of nesting sites and dry weather refuges, and The eggs are laid in fresh faeces and can only survive the use of traps. Insecticides including DDT and diel- with high humidity and warm temperature. They pupate drin are of long duration but have been considered by in the soil and the life cycle takes eight days to three some to cause too much long-term pollution. Shorter weeks to complete. Control of the flies is difficult but term insecticides that are biodegradable such as syn- removal of dung is important. The cattle and buildings thetic pyrethroids. are now favoured (see p. 760).

should be sprayed frequently with insecticide. If tick dipping is undertaken this reduces fly numbers. The

Tumbu fly

anthelmintics doramectin, ivermectin and eprinomectin

are effective because of their residual activity, and will
The tumbu or mango fly is *Cordylobia anthropophaga*.
protect cattle for periods of up to three weeks. The use
It infests man and animals but rarely cattle. It is about
of plastic insecticide-impregnated ear tags is helpful.
the size of a housefly and lays eggs in the soil or sand.
Otherwise, applying insecticide at regular intervals will
The hatched larva attaches to the skin of a host where
assist.

it produces a painful irritant swelling with a dark central
hole. Once mature the larva passes out to the soil where
it pupates. The adult lives on food and animal excreta.

Horse louse flies

In light infestations each larva can be manually
These include *Hippobasum equina*, *H. rufipes* and *H.*
removed by covering up the airhole with liquid paraf-
maculata. *Hippobasum equina* is the most common, is fin in Vaseline. The
parasite will push its posterior
slightly larger than the housefly and is a reddish-brown,
abdomen out of the hole and it can then be pressed
fast, glossy fly that causes problems to cattle and horses.

out gently. In severe infestations avermectins such as doramectin, eprinomectin and ivermectin will control legs and the perineum. The eggs are laid and develop in dry soil. They can act as mechanical vectors of compounds, can be applied.

disease. Plastic insecticide-impregnated ear tags are of use in control as are spraying or dipping of cattle.

Blow flies and screw-worm flies

These result in blow fly infection or myiasis. The flies

Tsetse flies (see p. 760)

involved include several genera: *Calliphora*, *Callitroga*, The *Glossina* species are an important African fly that *Chrysomya*, *Lucilia* and *Sarcophaga*. Cattle are less affected than sheep. The flies are really scavengers and

are 6–13.5 mm long, thin-bodied, yellow or brown flies.

mainly live on dead meat. However, true screw-worm

The wings are folded over each other and there is a

flies only lay their eggs in fresh wounds. They are,

slender proboscis. There are 20 species of *Glossina* and however, attracted to

dying flesh on live animals.

all are found in tropical Africa. They occur in a variety

Even the small wounds of ticks attract the flies. The

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larvae feed and grow quickly and mature in three to

starts to suck blood. Bloodsucking is not continuous but

five days.

takes place in short periods of activity until the tick

A local dressing of a larvicide and an antiseptic is

becomes fully engorged. Full engorgement for larvae

useful after clipping away the hair. The most effective

requires three to five days, for nymphs five to six days

treatments are the avermectins doramectin, epri-

and for adults seven to ten days. Babesia spp. are not ivermectin and ivermectin.

They normally promote a

transmitted by infected ticks until the end of the

complete kill in about 12 hours. All wounds should be

engorgement period because the babesial stages in

treated with an antifly preparation. Dipping or spray-

the tick have to develop and move to the salivary gland

ing the animals with insecticides can be helpful. Other-

prior to becoming infective sporozoites. The sporo-

wise, the number of flies can be reduced by trapping and zoites are injected into the host with saliva and then quick disposal of carcasses. Chromosomal translocation invade erythrocytes. They proceed to divide asexually of males to produce sterile or lethal mutant offspring in red cells, forming two pear-shaped merozoites. Each has been tried with some success. The American screw-erythrocyte ruptures when the merozoites leave to worm fly *Callitroga americana* has been controlled by infect new cells. The reproduction time in erythrocytes the release of irradiated males. They mate with the is in the order of 12–15 hours depending on the species. females, which lay sterile eggs, thereby ultimately When sufficient multiplication has taken place for par-reducing or eliminating the fly population.

asites to be visible in very low numbers in blood smears the animal will show a febrile reaction. The length of time required is usually approximately one to three

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weeks after infection, but depends on the number of *Babesia* in the infective inoculum, and in very large

Protozoal diseases

syringe-passed infections may be as short as two to three days. Thereafter the parasitaemia may build up

Babesiosis (redwater)

and in extreme cases more than 20 per cent of erythrocytes may be infected, although the percentage infected

Babesiosis is caused by a intra-erythrocytic protozoan

varies with the species involved, e.g. the parasitaemia of

of the genus *Babesia* transmitted by hard ticks of the *B. bovis* is usually less than 1 per cent in venous blood family Ixodidae (see p. 744). Unlike many other parasites whereas *B. bigemina* and *B. divergens* on average reach sitic diseases, it affects adults more severely than young

3–8 per cent. At this point the affected animal has a

cattle in which infection is frequently subclinical. It

febrile reaction and may exhibit the characteristic

causes fever, haemoglobinaemia, haemoglobinuria,

haemoglobinuria that produced the colloquial term of

anaemia and death.

‘redwater’ for the disease, and unless treated it may

die. After treatment the animal becomes a carrier of

Aetiology and epidemiology

the organism and may suffer from occasional recrude-

scences of parasitaemia. Ticks become infected by

The four most important species of *Babesia* that affect feeding on parasitaemic cattle. Infected adult female

cattle are *B. bovis*, *B. bigemina*, *B. divergens* and *B.*

ticks pass the infection to their eggs, the infection being

major, the first three being much more significant than termed transovarial, and the larvae, nymphs and adults the last. Two are considered 'small' *Babesia* and two

up to the F2 generation. Some *Babesia* spp. may remain

'large', and there is one large and one small species for

infected in the absence of feeding on carrier bovines,

both tropical and temperate climatic areas (Table 45.1).

although for *B. bovis* infection ceases with larvae.

The parasites are transmitted by hard ticks, which are

Transmission from larva to nymph to adult is termed

also affected by their role as intermediate hosts in the

transtadial infection, and is observed with *B. bigemina*

babesial life cycle. When an infected tick attaches to the

and *B. divergens*.

skin of cattle its mouthparts penetrate the skin and it

The epidemiology of the *Babesia* spp. is governed by

the local climate and behaviour of its tick vectors. As a

result they merit separate consideration as tropical or

Table 45.1

Babesia spp infecting cattle.

non-tropical species.

Species

Size

Climatic preference

Tropical species: *B. bovis* and *B. bigemina*. These species are found in Australia, Africa, South and Central

B. bovis

Small

Tropical or subtropical

America, Asia and the very south of Europe. In Aus-

B. bigemina

Large

Tropical or subtropical

tralia and the Americas the tick *Boophilus microplus*

B. divergens

Small

Temperate

is the sole vector, in Africa other *Boophilus* and

B. major

Large

Temperate

Rhipicephalus species. Boophilus spp. are one-host

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ticks, i.e. all stages of the life cycle take place on one

dark in colour and reduced in quantity. The faeces may

animal, only the engorged female dropping to the

return to normal but less tends to be passed although

ground before laying eggs. Some vectors in Africa, e.g.

there is still spasm of the anus.

R. evertsi, are two-host and R. appendiculatus is a three-host tick.

Late: In another 24–36 hours the rectal temperature

Babesia bovis merozoites in erythrocytes measure

is often subnormal with the animal having blanched

2 \times 1.5 mm and those of B. bigemina 4.5 \times 2 mm. Para-

mucous membranes, a poor appetite and drinking little.

sitaemia in venous blood is low with B. bovis but it may There is marked constipation and a greatly increased

be high in capillaries and cause sludging of blood, which

heart rate.

if in the brain causes early death. It also produces

Pregnant cows may abort following infection.

enzymes with severe effects on the blood coagulation system, and is generally considered the most pathogenic

Necropsy

of the bovine Babesia spp.

Babesia bigemina infection results in much higher

The carcass may be very blanched and there is some-venous parasitaemia but it has few other effects other

times jaundice. The liver is often swollen and pulpy, with than to cause a febrile reaction and straightforward

the kidneys dark and enlarged. The bladder contains haemolytic anaemia.

red-brown urine. There are ecchymotic haemorrhages under the epicardium and endocardium.

Temperate species: B. divergens and B. major. Babesia divergens, the merozoites of which measure 1.5×0.4

Diagnosis

mm, is common in areas of permanent pasture in north-

Cattle suffering from babesiosis frequently have a

western Europe and is transmitted by the three-host

history of recent movement to tick-infested pastures

tick Ixodes ricinus. Babesia major (3.2 = 1.5 mm) is either through grazing management or after purchase,

found only in south-eastern England and on islands

and in Europe may have suffered from tick-borne fever

off the coast of The Netherlands and is transmitted by

a week or so before babesiosis is evident. Examination

Haemaphysalis punctata.

of the cattle, especially the preferential feeding sites of

Babesia divergens behaves rather similarly to B.

the vector ticks, will reveal evidence of recent tick bites

bigemina, i.e. it can cause a high parasitaemia, which or engorging ticks. Clinical babesiosis is unlikely to be

results in fever and severe haemolytic anaemia. It has

observed in cattle less than nine months old; such cattle

little effect on blood coagulation systems in comparison

can be infected and show febrile reactions but the

to B. bovis. Its epidemiology is closely bound to the

resultant parasitaemia remains low and haemoglobin-

ecology of its vector I. ricinus. In Europe, I. ricinus is uria mild. In areas of large tick populations, most cattle

generally active only between May and November,

are infected at an early age and become immune there-
and in most areas has spring and autumn population
after, the situation being described as enzootic stability.
increases, although in the most northerly climates it
In the early stages of the disease, haemoglobinuria may
may only have one in midsummer. The ticks quest more
not be present and diagnosis requires careful examina-
actively in warm conditions and outbreaks of babesio-
tion of stained blood smears (see Fig. 45.1). Once
sis are frequently observed two weeks after fine
haemoglobinuria is present, the parasitaemia may be
weather. The epidemiology of *B. major* is still only
more obvious. Differential diagnosis requires elimina-
slightly investigated, but such isolations as have been
tion of other conditions causing haemoglobin uria, e.g.
reported have taken place in May and June.

anaplasmosis (p. 761), eperythrozoonosis, leptospirosis
(p. 734), postparturient (p. 792) and bacillary haemo-

Signs

globinuria (p. 719).

Early: There is slight dullness with a pyrexia often of Treatment and control (see

p. 1029)

40.5–41°C (105–106°F). The animal shows diarrhoea

and because of spasm of the anal sphincter there is

There are two aspects to treatment: firstly, treatment

a narrow stream of diarrhoea (pipe-stem diarrhoea).

with a babesicide and secondly, the need for supportive

There is also haemoglobinuria. Slight dehydration is

therapy such as blood transfusion and fluid replace-

often seen as a slightly sunken eye.

ment. There are few babesicides now available. The

only treatment now licensed in the UK is imidocarb,

Mid: After 24–36 hours the mucous membranes tend

which is given at a dose rate of 1 mg/kg body weight. It

to become pale and the pulse rate is increased. The

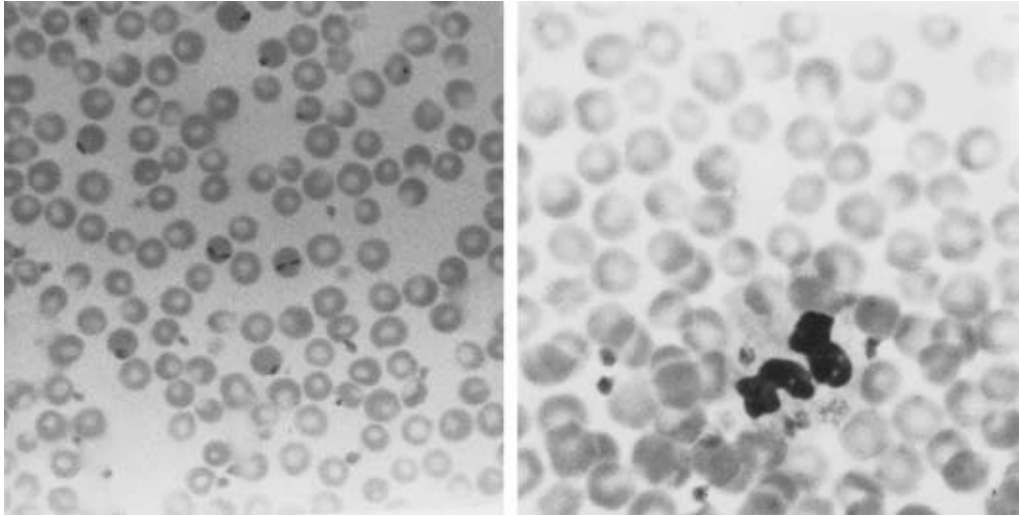
is used widely in South America and other countries. It

animals tend to slow up and there is a reduction in

is highly effective and relatively non-toxic, but does

appetite and thirst. The urine tends to become very

have tissue residues for several weeks after its use.



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(a)

(b)

Fig. 45.1

(a) Bovine blood infected with *Babesia divergens* (¥630). (b) Bovine blood smear showing *Ehrlichia phagocytophila* in a neutrophil (¥630).

It can also be used at twice the therapeutic dose as a

Table 45.2

Species of the genus *Theileria* in cattle.

chemoprophylactic, giving protection from infection for up to six weeks. It is used in this way to administer to

Species

Synonym

Disease

cattle that will be exposed to ticks, or that have been

T. annulata

T. dispar

Mediterranean coast

*vaccinated with live Babesia with the hope that cattle T. turkeistomica
infection*

will become mildly affected but protected from clinical

T. lawrencei

T. bovis

Corridor disease

illness and immunity to further infection will be

T. mutans

T. orientalis

Benign bovine theileriosis

stimulated.

T. buffeli

Live vaccines for B. bovis and B. bigemina have been T. parva

East Coast fever

available in many countries for many years, notably

T. sergenti

Mediterranean coast fever

Australia and South America. For B. bovis the vaccines (Russian)

consist of live organisms made avirulent by repeated rapid syringe-passage through splenectomized calves. In the case of *B. bigemina* rapid passage did not reduce

Theileriosis

virulence and vaccines available are either developed by 'slow' passage from recrudescences of parasitaemia

This comprises a group of infections caused by proto- or are fully virulent organisms, which are used in

zoan parasites of the genus *Theileria* (Table 45.2) and schemes involving infection and subsequent treatment.

transmitted by ixodial ticks. They occur in a variety

There are no vaccines currently available for *B. diver-* of ruminants and wild animals. Both members of the gens, although much research is being carried out to

genus *Babesia* and the genus *Theileria* occur within red develop inactivated recombinant vaccines for that

blood cells. They are collectively called piroplasms and species and *B. bovis* and *B. bigemina*.

the infections caused by the two are thus sometimes

Blood transfusion is frequently required for severely known as 'piroplasmosis'.

affected adult cattle and is normally achieved by col-

lection of 5 l of blood from an unaffected healthy cow

East coast fever (ECF) (pp. 73, 753)

*into a 22 per cent solution of the anticoagulant acid
citrate dextrose (ACD), the mixture being immediately*

*The disease is a major constraint for production in
transfused into the recipient animal. Such single trans-
countries where it occurs.*

*fusions without cross-matching of blood are usually
successful, but repetition can lead to problems of*

*Aetiology: The cause is a protozoan parasite, Theileria incompatibility of blood
antigens.*

parva. There is some disagreement about its classifica-

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*tion. Theileria lawrencei, a parasite of buffalo, causes a delay of three to five
days after attachment before this*

*high mortality in cattle, but if passaged through cattle
occurs. If the tick infects a susceptible animal there is a
it reverts to a parasite indistinguishable from T. parva
period of five days before the parasite can be detected
and producing a syndrome similar to ECF. Thus it
in the local drainage lymph node closest to the tick bite.
has been suggested that the cattle parasite should be*

What happens during this period is not known but it is called *T. parva parva* and that of the buffalo *T. parva* likely that sporozoites rapidly enter target lymphocytes.

lawrencei.

By doing this the sporozoites escape the phagocytic, lytic and immunological defences of the host. Then

Epidemiology: The disease occurs across a large area

there is a transformation of the lymphocyte to lym-

of East and Central Africa and is endemic in Burundi,

phoblast with larger, less dense nuclei and increased

Kenya, Malawi, Sudan, Tanzania, Uganda, Zaire and

cytoplasm caused by the parasite, which in turn differ-

Zambia. It may also occur in Ethiopia and southern

entiares into a macroschizont in the cell cytoplasm. As

Somalia. It is possible to eradicate the disease and this

the disease progresses the macroschizont grows to an

has been done in Mozambique, South Africa, Swaziland

average size of 4.8 mm. The lymphoblasts rapidly pro-

and Zimbabwe, although it can return if preventive

liferate, probably stimulated by the presence of the

measures are not maintained. It is a disease of cattle but

parasite, which becomes aligned along the spindle and can infect Indian buffalo (*Bubalus bubalis*) and African divides by synchrony. As the infected lymphoblast buffalo (*Syncerus caffer*). Infection is restricted to coun-divides both daughter cells are infected. Some tries with a temperature and rainfall suitable to allow macroschizont-infected cells degenerate releasing free the survival of *Rhipicephalus appendiculatis*, a three-macroschizonts, which invade other uninfected lymphoid cells. How this occurs is not known but it may be (7000 feet) provided there is adequate vegetation and through membrane fusion of cells in close apposition. a rainfall in excess of 50 cm (20 inches). In many areas From about day 14 after tick attachment macroschizonts differentiate to microschizonts. The mechanism follows the onset of rain and thus tick activity. In some of differentiation is not known. Microschizont-infected highland areas or close to water or sea, where the rain-fall is more or less constant, tick activity and ECF can

1.5 mm in diameter). The released micromerozoites occur virtually all the year round. Although *R. appendiculus* enter erythrocytes where they form piroplasms *diculatis* is the main arthropod host, eight species of 3–5 mm), which tend to be rod or comma-shaped. The the genus *Rhipicephalus* and three species of the genus piroplasm-infected erythrocytes are then available to *Hyalomma* can be experimentally infected. In areas infect ticks feeding on the blood of the cattle host. where there is a constant challenge then the cattle will *Theileria parva* piroplasms rarely divide within erythrocytes, which is different from *T. mutans* and also challenge is heavy then the calves die. However, those *Babesia* spp. that survive are resistant to further challenge and they Following feeding on the infected bovine the tick, can thrive in these areas. In marginal areas where challenge is intermittent or seasonal then cattle previously blood that is around the stage of gametogamy. The tick exposed may lose or have reduced immunity. Thus if a is a three-host tick with all three stages (larva, nymph

heavy challenge occurs at the start of the rainy season and adult) feeding on separate hosts. Infection is trans-situational so an infected larva can transmit infection as disease. Immunity is only to challenge by a similar nymph but not as an adult. There appears to be no strain. Cattle previously exposed may be partly or transovarially transmitted as occurs with *Babesia* spp. totally susceptible to infection by other strains. Recovery. Once in the tick gut the erythrocytes lyse releasing the merozoites. Infected cattle have a sterile immunity, which lasts more than three years. Levels of immunity to piroplasm anti-merozoites develop into male microgametes or female gametes peak often four to six weeks after infection and microgamonts. Fusion of these by prokaryotic anisocytogony persist for six months.

Sexual reproduction is then thought to occur to produce zygotes, which invade gut cells and thus differentiate into a motile

Life cycle: The life cycle of *T. parva* is still not completely known. The kinete breaks out from

pletely understood but most of it is now known or the gut cell and enters the haemolymph. The stage of extrapolated. Firstly, at the stage of introduction of sporonts is then reached. The kinetes invade the acinar infection to the cow there is the period of schizogony. cells (usually type III acinars) of the salivary glands. Sporozoites from infected ticks are injected with saliva The kinetes round off and nuclear division occurs to into the cattle while the tick feeds. There is usually a produce a sporont or primary fusion body. The sporont

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invaginates and forms buds. Further development is ered by some to be due to a massive infection in par- delayed until such time as the tick starts to feed in tially immune cattle.

its next instar. When this occurs primary sporoblasts develop from the sporont buds and form cystomeres or Chronic: This is usually seen in animals that are par- the secondary fission stage. There is hypertrophy of the tially immune or exposed for long periods to low levels host cell and the cell nucleus. There is then division of

of infection. It often occurs in calves in endemic areas. the primary sporoblasts to produce secondary sporob- The lymph nodes tend to be enlarged and there is inter- lasts or the tertiary fission stage and sporozoites (1– mittent pyrexia, anorexia and loss of condition. These 1.5 mm) are produced. This stage is rapidly completed in animals frequently recover. In cases of concurrent three days from the onset of the tick feeding. The sporo- helminthosis, malnutrition or other disease and a con- zoites are released into the salivary duct with peak stant population of *T. parva* some animals become sporozoite production by day 5. The host cell and severely retarded and never reach their full production nucleus degenerate and the parasite residual bodies potential.

remain. The sporozoites persist during the whole period of feeding, which may be up to 10 days for Pathology: The lesions will depend on the duration of female ticks and intermittently over a long period for signs. The most consistent finding is one of hypertrophy male ticks.

and hyperplasia of the lymph nodes initially, followed by lymph node oedema and some haemorrhage and cent, with an incubation period of 10–15 days.

necrosis later on. There is destruction of lymphocytes leading to destruction of lymphoid cells. Lymph node biopsy shows hypertrophy of lymphoid cells and often

Signs

after the 11th day they may show macroschizonts that

Peracute: There is marked pyrexia and death with increase in number and size. Damaged lymphocytes are swollen lymph nodes in a few days.

seen with free schizonts. Microschizonts are then present, either intact or branching out of cells as micromerozoites. Blood examination reveals a progres-

Acute: Usually the first sign is an enlargement of the sive panleucopenia. A noticeable rapidly developing

lymph nodes for the region draining the area where the anaemia is only seen in the terminal stages.

infected tick has fed. The preferred feeding sites for R.

The examination reveals froth at the nostrils and

appendiculatus are the ears and so usually the first the most striking feature is massive pulmonary oedema, lymph nodes to swell are the parotids. One to two days hyperaemia and emphysema. The alveoli, bronchioles, after the swelling occurs the animal becomes pyrexia bronchi and trachea are filled with frothy pulmonary with a temperature rising to 39.5–42°C (103–108°F). exudate. There can also be pleural and pericardial The temperature tends to remain high until the animal exudate. There are excessive haemorrhages and these either recovers or dies. Other lymph nodes begin to may be present on most serous and mucous membranes and this tends to become generalized. Some of the membranes. In the abomasum the mucous membrane is red superficial lymph nodes such as the parotids, prescapular and inflamed and there may be ulceration or erosions, lars and precrurals become very enlarged. Anorexia especially in the pyloric region. In chronic cases there gradually develops and there is consequent loss of condition are ulcers in Peyer's patches. The cortices of the kidneys dition. In many cases lacrimation and nasal discharge

show haemorrhage and are often congested, and occur. The breathing becomes rapid and dyspnoeic, and nodules of lymphoid tissue projecting from the kidney there is diarrhoea or dysentery. As the animal deteriorates surface may be seen and are a characteristic feature rates and approaches death, the temperature falls when they occur. The spleen may or may not show and there is severe dyspnoea and recumbency. Nasal changes and it can be enlarged or shrivelled. The liver exudate pours out of the nostrils. The animal dies of is often enlarged with mottled grey areas. Degeneration asphyxiation from lung oedema. Death is usually about of the organs is rapid after death.

18–24 days after infection, but occasionally this is reduced to 14 days. The mortality tends to be near 100 per cent in susceptible animals.

Diagnosis: The signs are relatively specific and infectious. Occasionally, nervous signs develop and this is known

tion can be confirmed by the presence of piroplasms in as 'turning sickness'. Foci of Koch's blue bodies are the blood or schizonts in lymph node biopsy smears

found in the cerebral tissue. The animal appears to turn stained with Giemsa. There is also panleucopenia and often rapidly and become giddy with collapse. The less then anaemia.

severe form involves slower turning and frequent head

Post-mortem findings are helpful but they are similar

pressing. Both nervous forms are fatal and are consid-

to those of malignant catarrhal fever. Differentiation of

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T. parva from other Theileria spp. depends on the acute-ing out due to poor immunity or deaths due to vacci-

ness of the disease, the number of piroplasms present

nation. However, animals can be vaccinated with stabi-

in the blood and the number present in lymph nodes.

late produced by freezing down emulsions from

Complement fixation, capillary titre agglutination and

infected blood. After injection of the stabilate long-

indirect haemagglutination tests have all been used in

acting oxytetracycline or buparvaquone is given; other-

diagnosis but are less reliable. The indirect fluorescent

wise short-acting oxytetracycline or paravaquone can

antibody test (IFAT) is considered more reliable. An
be used. Cattle so treated become resistant to disease
enzyme-linked immunosorbent assay (ELISA) test is
and they show little or no apparent reaction. In some
also being increasingly used.

cases more than one strain of *T. parva* is introduced, possibly together with a
strain of *T. lawrencei*. A sporozoite vaccine is now being tried.

Treatment (p. 1029): Animals in good condition prior
to infection have lower morbidity. Parvaquone and the
more recently developed buparvaquone are effective if
treatment is not delayed until the animal is too severely
Mediterranean coast fever
affected. Oxytetracycline is effective if given at the
This is in many ways similar to East Coast fever but is
same time as infection occurs. It is also able to reduce
caused by a different *Theileria* species.

the severity of clinical disease and appears most
effective when given by injection. Chlortetracycline
given at any stage of infection at a dose of 10 mg/kg

Aetiology: The cause is a protozoan parasite, *Theileria* body weight either
parenterally or by mouth reduces

annulata. Morphologically, the parasite is similar to *T.*

the severity of parasitaemia and pyrexia.

parva and the macroschizonts and later microschizonts are found in the lymphoid tissue. Theileria annulata

piroplasms in the erythrocytes tend to be oval or round

Control: In endemic areas indigenous calves have a

in shape. In Russia, there is a similar disease due to T.

high degree of resistance to disease. However, calves of

sergenti, which is different from T. annulata both mor-European breeds are very susceptible to infection and

phologically and immunologically.

often die. Those that survive often succumb to further

attacks. Adult cattle of any breed brought into the areas

are highly susceptible to disease and probable death.

Epidemiology: The condition is found around the

The immunity is more cellular than humoral.

Mediterranean including south-eastern Europe, Russia,

Thus where disease is endemic there is legislation to

the Middle and Far East, India, Sri Lanka, Egypt and

control ticks, to slaughter infected animals, quarantine

Sudan. Both cattle and water buffalo (Bubalus bubalis) cattle and restrict cattle movement. While it has been

are susceptible to the infection. The condition involves

shown in South Africa and Zimbabwe that eradication of a development cycle in ticks of the genus *Hyalomma* of ECF is possible, it is expensive and in many countries and seven species are known to be vectors. The ticks try the legislation is not enacted as rigorously as it involved are one-host, two-host or three-host ticks. It might be. However, individual farmers can do much to be believed that in most tick species there is no transovarially reduce problems on their own premises by sensible transmission. Thus infection is acquired in the cattle management and tick control. Efficient and well-maintained fencing will reduce the access of nomadic When the tick is attached to the host it must feed a considerable amount before infective stages of *T. annulata* ticks can also be fenced off. Grass burning, rotational are produced and enter the cattle. Piroplasms appear in grazing, alternate grazing with other species such as the erythrocytes shortly after the first detection of sch-goats or sheep, alternate grazing with immune cattle or

izonts. The intra-erythrocytic form remains in the blood rotating land between grazing and crop all reduce the for many years and in natural infections they tend to problem. Cattle should be quarantined on entry to the have a ring or oval shape.

farm. Should disease break out its effects can be The disease is seasonal, depending on the activity of reduced by slaughtering infected cattle, stopping move- the ticks, which hide during the winter in crevices ment of cattle, other animals, people, hay or feed from between rocks, walls, etc. The result is the infective adult infected areas and then creating a buffer area between stage being produced in late spring or early summer. the infected and clean areas.

Thus infection tends to be seen mainly in the summer It seems that for effective immunization of cattle and early autumn. Infection can also be transmitted by against ECF it is necessary for the infection to be estab- injecting blood or tissue from ill or recovered animals. lished in the host. Vaccination is not widely used as it Intra-uterine transmission has been recorded on occa-

often leads to unpredictable results, with disease break-
sion. Infection of calves in endemic areas usually

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produces a mild disease, although up to one-quarter of

Diagnosis: The area, type of animals affected and signs calves can die.

help in diagnosis. Lymph node smears show schizonts.

In the Russian form involving *T. sergenti*, infection is In some cases a liver
biopsy is needed to differentiate.

said to be transmitted by only one species of the genus

Serological tests used for *T. annulata* include the com-*Haemaphysalis*.

plement fixation test (CFT), indirect fluorescent anti-

body test (IFAT) and enzyme linked immunosorbent

assay (ELISA).

Signs

Peracute: This occurs in completely susceptible animals *Treatment:* Many cases
of *T. annulata* infection show entering endemic areas. The animals develop
marked

spontaneous recovery. There have been few controlled

pyrexia with anorexia, depression and weakness and

trials of treatment. Both oxytetracycline (by injection)

they die in three or four days.

and chlortetracycline (orally) have been claimed to give

relief. However, buparvaquone is at present the best

Acute: This is most commonly seen in susceptible therapy.

animals moved into endemic areas and in marginal areas of tick activity. The animals develop pyrexia,

Control: Following natural infection there exists a pre-which may persist for several days. It is accompanied

munity that lasts for many years. There is no cross-

by inappetence, lethargy, swelling of the superficial

immunity to other Theileria spp. Immunization is

lymph nodes, oculo-nasal discharge and ruminal stasis.

practised in several countries by taking blood from

This is followed in a few days by anaemia with pale

recently recovered cattle passaged through susceptible

mucous membranes, exercise intolerance and a rapid

cattle. This is continued until no piroplasmic forms of

heart rate. Later on jaundice may become apparent.

the parasite occur. Then citrated blood is injected into

Constipation is common when pyrexia first occurs

susceptible stock, usually calves. Most animals show

but later there is diarrhoea and bloodstained faeces.

only limited reaction to such vaccination, although a
The animals lose condition rapidly and about 90 per
few die. As there are no piroplasms in the blood ticks
cent die over a period of eight to eighteen days after
are not infected. Immunity can then be enhanced by
signs occur.

injecting a virulent strain. It has been possible to culture
schizonts on various tissue culture cell lines and these
Subacute: There is intermittent fever for two to four
have then been used to vaccinate susceptible cattle.
weeks with moderate progressive anaemia and jaun-
They do not produce piroplasms and so the animal
dice. Many of these animals die but some recover over
cannot infect ticks.

a long period. Some of these cases change into a more
Prevention otherwise involves control of the tick and
acute phase and die.

cattle movements. Cattle can be sprayed or dipped with
acaricide. Some walls and buildings can be sprayed to
Chronic: This is often an even more prolonged form of
kill off the overwintering stages. Carrier animals still

the subacute disease. Recovery can occur but is very contain infection in their blood and if moved to new protracted. However, some cases suddenly develop the farms or countries will take the disease with them to more acute form and die.

infect local ticks. Thus detection of carriers is of use and can be done by taking blood from the animal and inject-

Necropsy: There is a pale, anaemic carcass with the ing into susceptible cattle.

mucous and serosal membranes showing numerous petechial haemorrhages. The lymph nodes are enlarged, cystic and oedematous. The liver tends to be pale brown

Corridor disease

or yellow, enlarged and friable with an enlarged friable

This condition is very similar to ECF but it has a spleen. The kidneys tend to be pale and on occasions different cause.

show pseudoinfarcts. The abomasum is red, inflamed and may show haemorrhagic ulceration. There are epicardial and endocardial haemorrhages and these

Aetiology: It is caused by Theileria lawrencei, which is may be petechial or

ecchymotic. The lungs contain primarily a protozoan parasite of buffaloes. However, oedema, often red-tinged, and congestion.

it is transmissible to cattle and produces a similar type A lymph node smear may show schizonts present in of disease to ECF. The schizonts tend to be smaller and lymphocytes but they are more common in the liver and fewer in number and the piroplasms relatively rare in spleen.

the blood. If *T. lawrencei* is passaged through cattle it Ectoparasites, Tick and Arthropod-borne Diseases • 755 quickly reverts to a parasite indistinguishable from

A few lymphocytes contain schizonts and there are one

ECF. It has therefore been suggested that *T. parva* is a or two piroplasms in the erythrocytes.

cattle-adapted strain of *T. lawrencei*. It has also been proposed that the nomenclature for the two parasites

Diagnosis: The condition is similar to ECF, anaplas- should be *T. parva parva* and *T. parva lawrencei*.

mosis and babesiosis and the peracute form is similar to heartwater. The signs, and area, help in diagnosis,

Epidemiology: The disease has mainly been described

particularly of those cattle associated with buffalo.

in South Africa, Zimbabwe and Kenya. It is transmit-

However, where *T. parva* infection is common then

ted by the three-host tick *R. appendiculatus* and it is disease caused by *T. lawrencei* may be missed. A lymph possible that distribution of *T. lawrencei* is over the node biopsy helps as schizonts in the lymphocytes tend

whole area occupied by the tick. The buffalo (*Syncerus*

to be few and small in size. Blood samples show only a

caffer) is considered to be the natural host and cattle few erythrocytes to contain piroplasms. There is also no

brought in to the area tend to be susceptible. Those

marked anaemia as in anaplasmosis or babesiosis.

brought up in the region that survive the first few

months are immune but those in marginal areas or

Treatment: Use of oxytetracycline by injection reduces where tick levels have only been partially introduced

the intensity and duration of signs. Similarly, chlortet-

are not. *Theileria lawrencei* is transmissible from carrier racycline has an effect and can be given by mouth. The

buffaloes via ticks to cattle. It can be passed from cattle

naphthoquinones, parvaquone and buparvaquone are

to cattle via the ticks. The recovered cattle can then act

now successfully used.

as carriers of infection. Morbidity in susceptible cattle is variable at about 60–80 per cent.

Control: Infection can be controlled by tick dipping, controlling movement of susceptible cattle, grazing tick

Signs: The signs are similar to those seen with T. parva.

areas with less susceptible species such as sheep or goats and ensuring no contact occurs between cattle

Peracute: The animal develops pyrexia, lymph node and buffalo or other game animals. Cattle that are enlargement and dies in a few days.

exposed to infection and recover are immune.

However, there is not complete cross-immunity with T.

Acute: This is the most common form in susceptible

parva infection, although it does seem that cattle pre-cattle. There is pyrexia and swelling of the lymph node

viously infected with T. parva have a good immunity to closest to the site of the infected tick bite. Other lymph

T. lawrencei. When cattle are initially infected with T.

nodes then start to swell and there is general dullness.

lawrencei there is only limited resistance to T. parva.

There is lacrimation, nasal discharge and oedema

Stabilates of ticks infected with strains of T. parva have occurs, particularly of

the eyelids, face and throat. The animal has been injected into cattle followed by treatment with oxytetracycline or parvaquone, which has given some immunity to *T. lawrencei*. Some stabilates have included and dyspnoeic. a *T. lawrencei* strain.

Mild: In this form there is a mild rise in temperature, Benign bovine theileriosis swelling of the lymph nodes and some pyrexia.

This disease tends to be less severe than ECF, Mediterranean coast fever or Corridor disease.

Pathology: The lymph nodes are swollen and show hypertrophy and hyperplasia followed by oedema and haemorrhage. There are extensive haemorrhages on

Aetiology: The cause is a protozoan, *Theileria mutans*, most of the serosal and mucosal surfaces. The aboma-which is very similar to *T. parva*.

sum tends to be red, inflamed and with ulcers, especially in the pyloric region. The spleen may be enlarged. The

Epidemiology: The condition affects cattle as well as kidney cortex is congested and may show nodules of the Indian water buffalo (*Bubalus bubalis*) and the

lymph and tissue raised above the surface. The liver is African buffalo (*Syncerus caffer*). It occurs in most enlarged and a mottled grey colour. The lungs are grey parts of the world except countries north of latitude and contain blood and fluid with froth in the alveoli, 55°N and South America. It is thought that transmission is by a wide variety of different ticks but it has been smears show hypertrophy and hyperplasia of the node. proved to be so with *R. appendiculatus*, *R. eventsi* and

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Amblyomma variegatum. Injection of parasitized blood

Besnoitiosis

can produce infection. The disease is maintained by a

This disease was previously known as globidiosis.

premune state of recovered cattle, which allows continued infection of ticks. Infection often occurs with other diseases such as anaplasmosis, salmonellosis, heart-

Aetiology

water or babesiosis.

It is due to a protozoan called *Besnoitia besnoiti*.

Signs

Epidemiology

Acute: This rarely occurs but it has been reported in

It is mainly seen in cattle and horses as intermediate

Australia, India, Japan, Kenya, Korea and South Africa

hosts in south-west Europe and in Africa. Transmission

(where it is known as Tzaneen disease). In this case the

has not been elucidated but is probably via the faeces

disease involves fever with swelling of the lymph node

of infected cats which are the final host. Morbidity can

closest to the infected tick and then more generalized

be up to 10 per cent and recovery is often protracted.

lymph node swelling. There is a progressive anaemia

and some of the cattle die. In Africa there is a cerebral

form of the disease known as turning sickness where the

Signs

animal will tend to walk in circles or head press.

In many animals there are no signs. In the cow there

may be lesions on the teats. There is pyrexia and warm

Subacute: There are very few clinical signs other than swellings develop on the ventral parts of the body

a mild pyrexia with a swelling of the lymph nodes and resulting in reduced movement. The lymph nodes are a mild anaemia.

palpably swollen and there is diarrhoea. Pulse and respiratory rates are elevated. Pregnant cattle may abort.

Pathology: In acute cases there is hyperplasia and There may be excessive lacrimation and increased nasal oedema of the lymph nodes. There may be haemor-

rhages of the serosal and mucosal surfaces. The

There are small, white, raised nodules on the conjunc-abomasum may be reddened and ulcerated. The

tiva and nasal mucosa. There then follows severe der-kidney cortex is congested with swollen lymphoid areas.

matitis over most of the body associated with infected

The liver is enlarged and tends to be grey in colour.

cutaneous cysts.

The lungs contain fluid in the bronchioles, bronchi and trachea. The spleen is swollen.

Diagnosis

Usually in the subacute form there is mild hyperpla-

sia of lymph nodes, which contain a few schizonts.

This is based on clinical signs, especially the cysts on the

Anaemia occurs but is slight. There are only a few piro-

scleral conjunctiva, and on the geographical area and

plasms in the blood and schizonts in lymph node

can be confirmed by the detection of cysts containing

sinuses.

spindle-shaped spores in scrapings of skin lesions or the

conjunctiva.

Diagnosis: Diagnosis is helped by the mild nature of

the signs but is not helped by the presence of only very

Treatment

few piroplasms in the blood or schizonts in lymph node

Nothing specific is available.

biopsies. In addition the schizont is morphologically

similar to T. annulata and T. parva. The best method of differentiation is by serology and the most effective test

Control

at present is IFAT.

A vaccine produced by B. besnoiti grown on tissue

culture is effective.

Treatment: Quinoline drugs such as pamaquin are of use in the erythrocytic stage.

Trypanosomosis

Control: As the disease is mild, deliberate control of Trypanosomes are blood-borne protozoa with flagellae.

eradication is not usually undertaken. However, control

Infections are widespread in wild and domestic animals

of ticks will result in control of disease. Cattle maintain

and cattle are susceptible to infection with several

immunity by a form of premunity, with piroplasms

species, the most important of which are those cyclically

remaining in small numbers within the blood.

transmitted by tsetse flies (Glossina spp.) throughout

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much of subSaharan Africa, namely Trypanosoma

the life cycle and hence nagana is unique to Africa. The

congolense, T. vivax and T. brucei. Trypanosoma vivax one exception of the nagana trypanosomes is T. vivax,

infections also occur in cattle in the absence of the

as mentioned earlier. In Central and South America it

tsetse fly in Central and South America and have been

is assumed that T. vivax is mechanically transmitted by recorded in Mauritius. In

tropical and subtropical areas

biting flies. This raises the possibility that it may also be other than subSaharan Africa, cattle are commonly transmitted mechanically in Africa, and indeed there is infected asymptotically with T. evansi.

increasing evidence that this is the case.

Tsetse fly-transmitted trypanosomoses are com-

Infection with T. evansi is also transmitted mechanically by biting flies. Infection of domestic livestock is

only grouped together under the name 'nagana' (p. 1030). Their distribution lies within the tsetse fly belts

widespread worldwide throughout the tropics and

of Africa, which extend from 14°N to 20°S in south-west subtropics, but is absent from tsetse-infested areas of

Africa and 29°S in Mozambique, covering an area of 10

Africa. It is an important pathogen of camels, horses,

million km². Many species of wild animals are symp-

dogs and buffaloes but cattle, although commonly

tomless carriers of nagana trypanosomes and provide

infected, rarely suffer clinical disease. However, they

a sylvatic reservoir of infection in which the try-

may be important reservoirs of infection for other more
panosomes are cyclically transmitted naturally from
susceptible livestock.

host to host by tsetse flies. The principal carriers of
these trypanosomes are wild bovids and suids, e.g. kudu,

Aetiology

giraffe, buffalo, warthog and bushpig. Cattle, other
domestic animals and man are infected when they come

Tsetse-transmitted bovine trypanosomoses are caused
in contact with these wild animal carriers and are bitten

by *T. vivax*, *T. congolense* and *T. brucei*. All are motile, by infected tsetse flies as
a result.

extracellular, spindle-shaped, flagellated protozoan

Tsetse flies can be classified as falling into three
parasites ranging from about 10 to 30 mm in length.

groups, namely forest, riverine and savannah. The forest

Trypanosoma vivax and *T. congolense* are essentially tsetse flies are found in the
tropical rainforests of

parasites of plasma, although *T. vivax* may leave the

Central and West Africa and in scattered areas of East

circulation in small numbers and invade extravas-

Africa. Although they are efficient vectors of try-

cular tissues particularly of the heart (Losos, 1986).

panosomes, they are of least importance as cattle rarely

Trypanosoma congolense has a predilection for the

come in contact with them due to the lack of suitable

microvasculature where it attaches to the endothelium

grazing in the forest regions. Riverine tsetse flies, as

of small blood vessels, particularly the heart and brain

their name implies, infest riverine vegetation, but

(Banks, 1978). *Trypanosoma brucei* as well as being a

virtually only in river systems draining into the Atlantic

plasma parasite has a predilection for interstitial spaces

Ocean. Their distribution is thus confined to Central

and tissue fluids.

and West Africa, largely overlapping with that of forest

Trypanosoma evansi is related to *T. brucei* to which flies. Although they are less efficient vectors of try-it is morphologically similar and has a similar infection

panosomes than forest or savannah flies, because they

pattern in the animal host.

infest vegetation near essential water supplies, riverine

flies are important vectors of trypanosomosis of

Pathology

humans (Gambian sleeping sickness) and of domestic livestock including cattle.

The pathogenesis of bovine trypanosomosis is complex. Savannah tsetse flies are the most important group of and not fully understood but is characterized by a flies because they infest large tracts of land potentially chronic and progressive anaemia. Uncomplicated suitable for grazing and browsing by domestic livestock.

tsetse-transmitted infection can be considered as

They are also efficient vectors of trypanosomes and so following a course comprised of three phases (Murray, when cattle and other livestock encroach into tsetse-1978).

infested savannah, they are at risk of being bitten by the fly and contracting infection.

Phase I (fluctuating parasitaemia and fever): Following Tsetse flies become infected when they take a blood

an infected tsetse fly bite, trypanosomes multiply meal from an infected animal. The trypanosomes then locally, causing an inflammatory reaction (chancre) undergo cyclical development within the alimentary

within a few days at the site of the bite. Chancres are a system, eventually developing to the infective or meta-regular feature of experimental infections, but have not cyclic forms within the fly mouthparts. These infective been reported in natural infections. About this time, trypanosomes are then transmitted to another susceptible animal host via the saliva during the next fly feed(s). system, causing reaction and enlargement of locally draining lymph nodes. The development in the tsetse fly is an essential part of Trypanosomes then appear in

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the bloodstream, the prepatent period following initial Africa tend to cause a more chronic form of the disease. infective tsetse bite varying with species thus:

Trypanosoma brucei is the least pathogenic to cattle and normally regarded as of minor importance.

T. congolense

12–16 days

Although infection with more than one species is

T. vivax

8–10 days

commonly reported in the field, virtually no studies

T. brucei

5–20 days

have been done on mixed infections. Cattle infected

A fluctuating but diminishing parasitaemia then devel-

experimentally with T. congolense and T. brucei, either ops and parasitaemic peaks at approximately 12-day

simultaneously or one year apart, developed cerebral

intervals are associated with febrile responses. Anaemia

trypanosomosis with encephalitis and associated clini-

becomes evident early in infection and is believed to be

cal signs and both species of parasites were isolated

haemolytic in the first instance, but haemolysis

from the cerebrospinal fluid (Masake et al., 1984). The wanes and is superseded by anaemia caused by erythrocytes. Authors suggested that the higher incidence of cerebral

throphagocytosis due to stimulation and expansion of

trypanosomosis in mixed infections than in single infec-

the mononuclear phagocytic system resulting in

tions suggests an interdependence between T. con-

splenomegaly.

golense and T. brucei in the pathogenesis of cerebral This initial phase of fluctuating parasitaemia and

trypanosomosis, possibly resulting from T. congolense's fever may last from a few weeks to a few months during

predilection for the brain microvasculature facilitating

which cattle lose condition and, depending on the

the entry of parasites into brain parenchymal extravas-

severity of infection, some may die. Cattle that survive

cular spaces. The possibility of such interaction between

this phase enter the second phase.

different species in natural infections merits further research.

Phase II (low-grade parasitaemia and progressive anaemia): Over the next few months, infected cattle

Signs

have a low fluctuating parasitaemia during which the

parasites may be difficult to detect. Despite the appar-

The clinical picture of cattle suffering from nagana is

ent reduction in parasites, the erythrophagocytosis and

influenced by several factors, namely breed and health

anaemia continue, although the spleen may return to

status of cattle infected, pathogenicity of infecting

normal size and cattle continue to lose condition.

trypanosomes, duration of exposure to infection and level of tsetse fly challenge, which in itself is dictated by

Phase III (apparent aparasitaemia but continuing

several factors. Trypanosoma vivax infections in cattle anaemia): Cattle that survive the second phase suffer

in West Africa are widespread and commonly produce

chronic disease during which the parasites apparently

an acute, rapidly fatal disease in which affected cattle

disappear, although anaemia due to erythrophagocyto-

die during the initial phase of fluctuating parasitaemia

sis continues. Affected animals are cachectic and nor-

and fever. Stephen (1986) describes acute T. vivax

mally die within six to twelve months of initial infection.

infection as resembling septicaemia in which affected

Infection at any stage may lead to congestive heart

animals have body temperatures of 40–41°C

failure and death due to a combination of anaemia,

(104–106°F), depression, dyspnoea, elevated pulse and

circulatory failure and myocardial damage. At autopsy,

respiratory rates and a jugular pulse. Less severe cases

post-mortem findings are not pathognomonic. They show signs of anaemia, loss of condition (Fig. 45.2) and include emaciation, visceral pallor and enlargement of enlargement of superficial lymph nodes. Abortions and the heart. Cattle that die early in disease may have stillbirths may occur in pregnant cows.

enlarged haemorrhagic lymph nodes and splenomegaly

The situation in East Africa and parts of Central (Stephen, 1986).

Africa is different in that *T. congolense* tends to be a Despite the large volume of literature on bovine try-more serious pathogen than *T. vivax*, although this is by panosomosis, good accounts of the pathology of natural

no means absolute as strains of *T. vivax* are known to disease are scarce and the above account represents a

cause an acute haemorrhagic disease in cattle in the

brief synopsis of the generally accepted picture. Cattle

Coast Province of Kenya (Mwongela et al., 1981). Try-at risk may be infected by several species and strains of

panosoma congolense in East and Central Africa tends

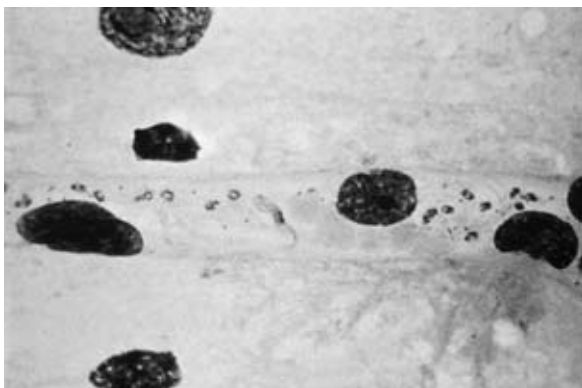
trypanosomes and the pathology and clinical signs will

to produce a chronic disease, although the clinical signs

be influenced by various factors, e.g. the age, breed and

are essentially the same as those of *T. vivax* infection nutritional status of

infected cattle, the degree of tsetse and eventual death is the usual outcome in untreated challenge and the strains and species of infective trypanosomes. In the early stages of infection appetite may be normal between periods of fever, but as the disease progresses the anaemia becomes more severe, cattle East Africa, whereas strains of T. vivax in West Africa tend to cause a more acute disease in cattle than those of T. congolense in East become depressed and lose bodily condition and in the



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Fig. 45.2

Emaciated cattle suffering from trypanosomosis in

Fig. 45.3

Trypanosoma congolense in ox brain capillary (cour-
northern Botswana (courtesy of A.G. Hunter).

tesy of CTVM Archives) (¥1000).

*terminal stages affected cattle are too weak to rise or
chronic wasting condition of cattle in contact with the
eat (Stephen, 1986). Superficial lymph node enlarge-
tsetse fly. Differential diagnoses are babesiosis (p. 748),
ment is not so pronounced as in T. vivax infections.*

anaplasmosis (p. 761), helminthosis (Chapter 19) and

Trypanosoma brucei infections of cattle, though

any condition that causes anaemia and emaciation,

common, are generally regarded as of minor signifi-

notably malnutrition. Nagana can be confirmed para-

cance and are usually mixed with the more pathogenic

sitologically by demonstrating parasites in the blood of

T. congolense or *T. vivax*. Parasitaemias from *T. brucei* infected animals and
various techniques are available

infections are lower than those of T. congolense or T.

(Fig. 45.3). These techniques were reviewed by Paris et

vivax and hence infection can be more difficult to
al. (1982) as follows.

detect, raising the possibility that disease caused by *T.*

- Microscopic examination of stained thin blood

brucei infection may not always be diagnosed. A few
smears. Different species of trypanosomes can be
reports of meningo-encephalitis have been recorded,
identified by this method.

and experimental infection produces a severe diffuse

- Microscopic examination of wet blood films; this
meningo-encephalitis resulting in depression, unsteady
must be done at the time of sampling and cannot be
gait, head pressing and circling (Morrison et al., 1983).
used to identify different species of trypanosomes.

There is a greater incidence of cerebral trypanosomo-

sis in mixed *T. congolense* and *T. brucei* infections as These techniques are not
particularly sensitive and may

mentioned earlier and the involvement of the central
not detect animals with low parasitaemias, such as those
nervous system (CNS) in natural bovine trypanosomo-
suffering chronic disease. More sensitive techniques are

sis requires investigation.

the following:

Trypanosoma vivax infections of cattle are wide-

- *Microscopic examination of the buffy coat–plasma*

spread in Central and South America and the West

interface of haematocrit-centrifuged blood, either

Indies and were probably introduced with imported

directly through the capillary tube glass or by

cattle from Africa. The importance of T. vivax in the breaking the capillary tube just below the buffy coat

New World is not clear, but epidemics of serious disease

and expressing the contents of the upper part onto

have been recorded in Venezuela and Colombia

a slide for examination by dark-ground or phase-

(Clarkson, 1976). In general, the clinical signs appear to

contrast microscopy.

be the same as chronic forms of T. vivax infection in

- *Microscopic examination of stained dehaemoglo-*

Africa and the swaying gait of infected emaciated cattle

binized thick blood smears.

may be confused with the clinical signs of rabies.

- Subinoculation of bovine blood into laboratory

Trypanosoma evansi infections of cattle, though

rodents. This is the most sensitive technique for *T.*

common throughout the tropics and subtropics, rarely

brucei and is usually good for *T. congolense*, but cause clinical disease.

most strains of *T. vivax* do not infect laboratory

rodents.

Diagnosis

In practice, many field programmes of monitoring

In tsetse-infested areas of Africa, nagana is well recog-

cattle for infection are based on routine screening of

nized and diagnosis is often based on a history of a

stained thick and thin blood films; thick films are

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examined to detect infected animals and thin films to

taining tsetse-free barriers around areas freed of

determine the species of the infecting trypanosomes.

tsetse.

Trypanosoma vivax infections in the New World

- Settlement of land freed of tsetse. Housing, crop-

may have to be differentiated from conditions causing

ping, etc. may alter the vegetation to a form unsuitable for tsetse flies, so preventing reinfestation.

and helminthosis and in addition, as mentioned earlier,

- Destruction of wild animal hosts. This is now unacceptable (p. 908; Chapter 70). In general the same techniques as described can be used to confirm diagnosis.

dors, possibly in conjunction with the third method,

Serological tests are not in general use for diagnosis above.

although several are under study. Trypanosomes display

- Release of sterile male tsetse flies to interfere with the phenomenon of antigenic variation during infection breeding of wild tsetse populations. Because of the in the mammalian host in which successive parasitaemic very low reproductive rate of tsetse flies, only small populations of trypanosomes have a different antigenic numbers of flies can be reared in colonies. Therecomposition. Thus the host mounts successive immune fore this method has limited application, usually in

responses to sequences of different antigenic populations in conjunction with other methods.

tions and a test developed to detect serum antibodies to one antigenic population may not detect serum anti-

Thus, the commonest form of prevention is avoidance of the fly by cattle herders who build up local knowl-

edge on when and where pastures are safe to graze. These variable antigens are confined to the surface glycoprotein coat of the para-

Savannah tsetse flies retreat and disperse during the dry sites and, although tests have been developed based on

and rainy seasons respectively, so that certain pastures these variable antigens, they are largely confined to

may be tsetse infested for part of the year only. In addition, grazing tends to be safest at midday, when flies are

research in antigenic variation and have little routine

diagnostic use.

least active, and most dangerous around sunset, when

Tests based on internal common somatic antigens

flies are most active (Pilson & Leggate, 1961).

may have potential diagnostic use as problems of cross-

In the absence of a vaccine cattle can be protected

reactions between species become solved by mono-

prophylactically, although effective prophylactics are

clonal technology (Nantulya et al. , 1987). Of various now limited to one drug, isometamidium chloride.

tests developed, the IFAT and ELISA appear to have

Isometamidium has certain disadvantages. It causes

the greatest potential and the ELISA can be used to

severe reactions at the site of injection, and there is con-

detect circulating trypanosomal antigens as well as anti-

siderable risk of resistance developing to the drug if

bodies (Rae & Luckins, 1984).

cattle are exposed to infection after the active ingredi-

ent in blood has fallen to below a trypanocidal level,

usually three months after injection. Thus where the

Treatment and control (see p. 1029)

risk of infection is constant, injection of the drug must

be repeated at regular intervals to maintain effective

Because of the phenomenon of antigenic variation, no

levels. Despite this, under good management cattle can

vaccine has been developed against trypanosomiasis
be efficiently reared in tsetse areas under isometamid-
and is unlikely to be in the foreseeable future. In Africa
ium protection, as has been demonstrated on the
this leaves tsetse control as the main method of pre-
Mkwaja Ranch in Tanzania over the last 30 years (Trail
vention. Tsetse control programmes are widespread
et al. , 1985).

throughout Africa but will only be considered in sum-
Treatment against trypanosomosis, in order to be
mary here. Tsetse control is usually under the direction
effective, should be given early in the disease during the
of the veterinary department and requires specialized
initial phase of fluctuating parasitaemia. As no new drugs
expertise. The methods in use are as follows:

have been developed against the disease for nearly 30

- Application of insecticide to tsetse habitat, either
years and some have been withdrawn because of resist-
on the ground by hand or from the air by helicop-
ance, treatment is now essentially limited to two com-
ters or fixed wing aircraft. This is the main method.

pounds, diminazene aceturate and homidium (either

- Use of fly traps. These are used extensively in chloride or bromide). Resistance has been recorded francophone West Africa and Zimbabwe.

against both drugs and undoubtedly will be an increasing

- Removal of the tsetse habitat. Tsetse flies have to problem. As there is little likelihood of pharmaceutical rest in certain bushes and trees, which can be companies developing new trypanocides because of cleared by felling and bulldozing, rendering the the cost and uncertainty of the market, in many of the area unsuitable for the fly. This is expensive, countries concerned the management of the existing however, and is now largely confined to main-drugs will require great care in the future.

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Trypanosomosis is normally seen as a herd problem 'trypanotolerant' breeds was reviewed by Hoste (1987). and mass chemotherapy is widely used on a herd or Trypanotolerance has also been identified in Orma area basis, ideally in conjunction with some form of

Boran cattle of East Africa (Njogu et al. , 1985) and the routine monitoring of blood smears of a percentage of

future prospects of greater utilization of breeds of cattle

cattle to indicate level of infection. Whiteside (1962)

with natural resistance to trypanosomosis are very real.

pointed out that when drugs are used regularly to treat

cattle for trypanosomosis resistance may develop and

when this occurs the drug in use must be changed to

Rickettsial diseases

one against which there is no resistance and that should

cure infections resistant to the first, i.e. a 'sanative'.

Anaplasmosis

Resistance was rarely reported against diminazene,

which consequently was recognized as the best sanative.

This is an infectious and transmissible disease of cattle

The concept of 'sanative pairs' was introduced in

that is seen in most continents of the world. It is non-

which drug usage regimens were devised to alternate

contagious and is transmitted by ticks.

diminazene with another trypanocide to minimize the

development of resistance. Thus the alternative to dim-

inazene was used for treatment of clinical cases for as

Aetiology

long as possible and then changed to diminazene for

The cause of the disease is an intra-erythrocytic

one year to ensure treatment of any resistant infections

*parasite, usually *Anaplasma marginale*. The red blood*

in circulation. Thus depending on the level of tsetse

cells contain round inclusion bodies called anaplasma

challenge, the regimen shown in Table 45.3 for treat-

and they are peripheral in location, hence the name

ment using drugs in current use was advocated.

*‘marginale’. There are three *Anaplasma* species and of In practice, it is virtually impossible to rear cattle in*

*the other two, *Anaplasma centrale* causes a mild condi-areas of very high challenge.*

*tion in African cattle, as does *Anaplasma caudatim*.*

Because treatment is now limited to diminazene and

**Anaplasma marginale*, when mature, is 0.3–1.0 mm in*

homidium and their use must be managed very care-

diameter and more than one can be present in the same

fully, Whiteside’s recommendations are possibly more

erythrocyte. The parasite transfers from cell to cell in

valid today than when first advocated.

the form of an inclusion body. This is normally oval

Diminazene aceturate is the drug of choice for treat-

in shape and measures 31 mm in diameter and can

ment of T. vivax infections in the New World.

penetrate the red cell envelope.

A member of the genus Parana-plasma, P. caudatum, Use of trypanotolerant breeds of cattle

has been found in a mixed infection with A. marginale

in cattle in the USA state of Oregon. The inclusion

It has long been recognized that dwarf humpless breeds

bodies of A. caudatum can be shown with special stain-of cattle in West Africa have a low susceptibility to try-

ing techniques to have unusual appendages such as

panosomiasis and can survive in tsetse-infested areas

rings, loops or beads in the erythrocytes. However, these

where zebu types or European breeds cannot. Until

are not found when deer erythrocytes are infected.

relatively recently these breeds were regarded as

having poor productivity and were of minor signifi-

cance, however new research indicates this may not be

Epidemiology

the case; their potential is now under extensive investigation and the situation concerning these so-called
The disease is particularly seen in tropical and sub-tropical parts of the world and it exists in some temperate areas. Africa, North, South and Central America,

Table 45.3

Regimen for treatment of trypanosomosis to minimize development of resistance.
the Far and Middle East, India, Russia and southern

Europe all have the disease present. While mainly a bovine disease, buffalo, bison, antelope, deer, gnu and

Tsetse challenge

Drug alternatives

wildebeeste can all be infected. All ages of cattle are susceptible, but calves under six months old show few

Homidium

Diminazene

if any signs. The severity of signs depends on the age and previous exposure to infection. Generally, the older

Very high

6 months

1 year

the animal at first exposure, the more severe the signs.

High

1 year

1 year

Ticks are the natural hosts of the disease and at least 20

Medium

2 years

1 year

species have been shown to transmit infection. Little is

Low

As long as possible

1 year

known about the developmental life cycle in the tick,

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although most infection is transmitted transovarially.

phology alters to include anisocytosis and poikilocyto-

The main genera of ticks concerned are Argas,

sis and leucocytosis is often present.

Boophilus, Dermacentor, Haemaphysalis, Hyalomma, Ixodes and Rhipicephalus. Horseflies (Tabanus spp.) *Diagnosis*

are experimentally and epizootologically the most important insect vector. They directly transmit the disease from an infected to a susceptible animal. Other anaplasma inclusion bodies in stained peripheral blood smears are sufficient for diagnosis. Giemsa stain is (Stomoxys), deer flies (Chrysops), housefly (Musca) and usually used but toluene blue and acridine can also be mosquitoes (Psorophora). Cattle can be carriers of the disease as well as deer. The incubation period is usually difficult and normally depends on complement fixation, two to six weeks, but it may be up to 12 weeks. capillary tube agglutination and agglutination tests.

Signs

Treatment

Peracute: This usually involves cattle over three years old experiencing infection for the first time. It is most commonly seen in high-producing purebred dairy cattle. The tetracyclines, i.e. tetracycline, chlortetracycline and oxytetracycline, are the only approved drugs that are

effective in treatment. Administration can be oral or and is frequently fatal. There is a pyrexia with rapid parenteral. Their use in the acute phase slows down the loss of milk production. Anaemia occurs with very parasitic life cycle and so reduces the crisis. Latent pale mucous membranes. The breathing is rapid with infections can be eliminated by tetracyclines. They act excessive salivation. Some cattle show nervous signs more effectively and more quickly when given by injection and abnormal behaviour.

tion rather than orally. Experimentally, other compounds of the dithiosemicarbazones have been shown

Acute: This is seen in cattle up to three years old and to be effective. It is also important to provide good

is occasionally found in cattle between one and two.

management for the animals and in valuable animals

Signs often develop unexpectedly. The animal develops blood transfusions may be necessary.

a progressive pyrexia over a few days, reaching 41°C (106°F). There is a loss of milk yield with a progressive

Control

anaemia and weakness. In addition there is depression, inappetence, dehydration and laboured breathing.

The main methods of control involve reduction in the

The lymph nodes tend to be enlarged. Some cattle will

vectors of disease, which can be done by ectoparasite

exhibit jaundice, there is frequent micturition of

dipping, but this does not entirely control the problem.

normal-coloured urine and some cows abort. Bulls may

Susceptible cattle can be separated from other carrier

show a temporary loss of fertility. Recovery takes a

cattle and wild animals or carrier cattle can be detected

period of weeks. If death occurs it is within one to four

and eliminated, although the tests used are not

days of the onset of signs.

completely reliable. Otherwise immunization can be

undertaken.

Chronic: The signs may follow an acute infection with

It was thought that cattle in indigenous areas do not

gradual emaciation.

normally show signs due to an infection immunity or

premunity. However, when there is intercurrent infec-

*Mild: This form is mainly present in cattle infected
tion or the animal is stressed in other ways then signs
under one year old. Signs are usually few with a mild
are evident, although it does seem that in premune
pyrexia.*

animals there is also a cell-mediated immune response.

*Both cell-mediated and humoral responses are required
to provide protective immunity to anaplasmosis. In*

Necropsy

addition, continuous antigenic response is dependent

The main signs at post mortem are of an acute anaemia

on a perpetual low-level exposure to infection. Immun-

and there is often jaundice. The spleen is enlarged and

ity using a live, laboratory-attenuated A. marginale

the gallbladder obstructed. The heart is usually pale and

ovine-origin vaccine gives good protection. Inactivated

flabby and petechial haemorrhages may be present on

A. marginale vaccines of bovine and ovine origin

the epicardium and pericardium. The lymph nodes are

require annual boosters. Their protective effect appears

enlarged and oedematous. The blood shows a marked

to be low. Experimentally, a more effective inactivated reduction in erythrocytes and haemoglobin. The mor-vaccine booster has been produced. Recently, a soluble Ectoparasites, Tick and Arthropod-borne Diseases • 763 organism-free cell culture derived from *A. marginale*

Signs

antigen has been developed.

Affected dairy cattle suffer an abrupt drop in milk pro-

Eradiction is possible where ticks can be removed.

duction, which may last despite treatment for four

Carrier cattle can be detected by a serological test, then

weeks, and beef cattle lose a significant percentage of

the non-infected cattle need to be kept away from

their body weight.

potential domestic or wild animal carriers. Movements

of the cattle need to be controlled and it is necessary to

reduce the level of biting flies. In such circumstances no

Diagnosis

live vaccination programme must be undertaken.

The history is usually similar to that of cattle affected

with *B. divergens*, i.e. recent purchase or movement to

Tick-borne fever

tick-infested pastures. Diagnosis can be confirmed by examination of blood smears for the presence of Tick-borne fever is caused by the rickettsial-like E. phagocytophila (see Fig. 45.1b).

organism Ehrlichia phagocytophila. It causes a prolonged febrile reaction, neutropenia and immunodepression in cattle in northern Europe (see Fig. 45.1b).

Treatment and control

Tetracyclines are the antibiotic of choice and long-

Aetiology and epidemiology

acting preparations have proved most successful.

Ehrlichia phagocytophila is transmitted by the tick

Treatment of cattle with pour-on synthetic

Ixodes ricinus during engorgement, but unlike babesio-pyrethroids has been used to prevent tick infestation,

sis, infection takes place almost immediately after

reducing the tick population for two to three weeks. It

ticks start to feed. The circumstances and timing of

has been less successful than when used on sheep, which

infections are dependent on the ecology of the tick and

retain higher concentrations of the pyrethroids on skin typically take place most often during spring or autumn and wool for longer than cattle. The fact that transmission of *E. phagocytophila* occurs early in engorgement, between April and November. Infection in ticks persists in contrast to *B. divergens*, has resulted in the observation that protection of cattle against tick-borne fever larvae.

using pyrethroids is less successful than against babesiosis. After infection the organism enters or is phagocytosed by white blood cells, usually neutrophils, but the tick before transmission.

infected eosinophils and monocytes are occasionally observed. The 'elementary body' can be seen in blood

Bovine petechial fever

smears stained with Giemsa or Leishman stains as a

(Ondiri disease, ondiritis)

pale blue dot in the cytoplasm of neutrophils. It enlarges to become a morula, after which the neutrophil

This condition is a rickettsial infection of ruminants, but ruptures and the elementary bodies of which the is restricted in area of occurrence.

morula is composed infect further cells (Fig. 45.1b). The disease becomes apparent five to nine days after trans-

Aetiology

mission by infected ticks. The animal develops a high fever, which can persist for up to 10 days. During this

The cause is a rickettsia-like organism, Cytoecetes

period the continuous destruction of neutrophils leads

(Ehrlichia) ondiri. The infection initiates in the spleen, to a severe neutropenia and for as yet unknown reasons

and there it parasitizes the circulating granulocytes and,

to a reduction of packed cell volume (PCV) of 30 per

more rarely, monocytes. The organisms are pleomor-

cent. As a result of the neutropenia the animal becomes

phic and occur in cytoplasmic vacuoles, particularly in

immunodepressed and susceptible to other infections

neutrophils. The organisms possess a rippled cell wall

such as pneumonia, infectious pododermatitis and

and they can be small (0.2–0.4 mm) or large (1–2 mm)

*babesiosis due to B. divergens, which is frequently
bodies. In some cases there are mixed groups of large
observed in susceptible cattle eight to twelve days
and small bodies.*

*after tick-borne fever. The 'parasitaemia' subsides but
recrudescences occur periodically thereafter. Immunity*

Epidemiology

*develops slowly and persists only for a few months as
cattle may be affected, albeit less severely, for some*

*The disease appears to be confined to the highlands of
years following their infection.*

East Africa. It mainly involves cattle exotic to the

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*region. Very well defined areas are often involved. The
which may be large and distributed throughout the*

*indigenous cattle and wild ruminants such as the bush-
body. There is oedema and lymphoid hyperplasia.*

*buck (Tragelaphus) and duiker (Silvicapra spp.) do not Often there is
subcutaneous and intramuscular haem-show clinical infection. The vegetation
common to all*

*orrhage and melaena. The heart shows large haemor-
sites is the edge of forest or thick bush. Experimentally,*

rhages into the epicardium and endocardium. There are infection can occur in cattle, sheep, goats, impala, bush-also haemorrhages and oedema in the respiratory tract buck, Thomson's gazelle and wildebeeste. Most of these and the mucosa and serosa of the alimentary tract. The develop latent infections. Thus carrier animals are pro- lymph nodes show hyperplasia and oedema. Both the duced from which it would seem highly likely infection liver and spleen may be enlarged and show petechiae. could be spread by an arthropod vector. However, Histologically, there is marked evidence of petechia- as yet, despite intensive investigations, the arthropod tion. Characteristically, hyperplasia of large areas of the involved (probably a tick) has not been detected. The lymphoid sinus are seen. The rickettsiae can be found incubation period is variable, from four to 14 days, and in impression smears of that surface of the spleen and in natural outbreaks disease has occurred 10 days after liver. There is a characteristic absence of eosinophils, entering into an infected area. Mortality is around 20 followed by markedly reduced numbers of lymphocytes

per cent and occurs within a few days of onset of signs, and then neutrophils.

but some animals will die two or three weeks after disease develops.

Diagnosis

Diagnosis is often difficult as the condition resembles

Signs

anthrax (p. 717), bracken poisoning (p. 946), arsenic

Peracute: These signs are seen in recently imported poisoning (p. 941), haemorrhagic septicaemia (p. 728), exotic cattle. They develop marked pyrexia and a heartwater, acute trypanosomosis (p. 756) and acute drop in milk yield. The signs usually coincide with a theileriosis (p. 750). It is usually based on the history parasitaemia. After two or three days petechial hae-

plus the area where it has occurred. Ehrlichia ondiri can morrhages occur on mucous membranes, and in some

be detected in blood or spleen smears stained with cases that are fatal there is general congestion, with pul-Giemsa, where it is particularly seen in granulocytes monary oedema, dullness, weakness and a staring coat.

and monocytes. Often by the time clinical signs develop

Most of the cattle collapse and die within four days.

the parasitaemia is low or absent. In such cases it may

be necessary to collect blood in EDTA or a suspension

Acute: These types tend to occur over a longer period.

of spleen or lung and inject this intravenously into

The temperature is high but fluctuates. There is

susceptible cattle or sheep. The granulocytes and

inappetence with reduced milk yield and abortion.

monocytes in blood smears from the recipient, stained

Although petechial haemorrhages occur in some

with Giemsa, should be examined daily for 10 days after

animals on the day after the onset of fever, in most

inoculation.

animals they have appeared by three days. The haem-

orrhages disappear to be replaced by fresh ones within

Treatment

seven to ten days. They occur on the vulva, vagina, con-

junctiva, labial surface of the gums and ventral surface

The most effective time to treat is during the incuba-

of the tongue. Any normal discharge and faeces may be

tion period when tetracyclines prevent disease. Once bloodstained. In some cases a characteristic 'poached egg eye' occurs with a swollen tense eyeball with the dosage of these antibiotics will reduce clinical signs aqueous humour containing blood, the conjunctiva and limit the parasitaemia. A single intravenous dose swollen and haemorrhagic and the eyelid everted.

of alphaethoxyethylglycoxal dithiosemicarbazone at 5 mg/kg body weight will also reduce signs and the

Subacute: Affected animals show a transient, non-fatal parasitaemia more effectively than tetracyclines.

condition with pyrexia and petechiation of the mucous membranes.

Prevention and control

Inapparent: No signs are present.

Eradication is impossible because wild animals act as a reservoir of infection. Clinically infected cattle are resistant to reinfection for several years, but they prob-

Necropsy

ably carry latent infections of E. ondiri. Losses due to The main findings are usually of an animal in good con-the disease can be reduced by restricting

access to areas

dition with submucosal and subserosal haemorrhages,

of known infection. Clearing the undergrowth and

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scrub aids this. If susceptible animals are to enter

dry season due to reduced tick activity. The incubation

infected areas they should be watched daily and when

period is variable, from 7 to 28 days, with fever starting

infection is present a single dose of dithiosemicar-

on average after 18 days. Mortality can be up to 60 per

bazone may then allow them to recover and become

cent in exotic breeds, but less than 5 per cent in local

immune to infection.

cattle.

Heartwater (cowdriosis or malignant

Signs

rickettsia, blacklung)

Peracute: This occurs in exotic breeds introduced to the This is another rickettsial condition that infects both

region. The animal appears clinically normal, but if

domestic and wild animals.

examined will have a marked pyrexia. It may then suddenly collapse, go into convulsions and die. Thoracic auscultation will often reveal oedema in the lungs and

Aetiology

bronchi.

*The infection is caused by the rickettsia *Cowdria rumi-**

*nantium and is transmitted by at least five species of Acute: The course of infection is three to six days and *Amblyomma* ticks. It is first found in reticuloendothe-consists of pyrexia (often over 41°C, 106°F), with*

lial cells and then parasitizes vascular endothelial cells.

nervous signs that may include ataxia, circling and

It is seen as close packed colonies consisting of less than

abnormal posture. In other cases signs develop only to

ten to many hundred cocci. The agent is pleomorphic

stimuli and there is then an excessive blink reflex, fre-

but the rickettsia in any one group tend to be of similar

quent tongue protrusion, a haggard, pained expression

size. The organism varies between groups from 0.2 mm

and muscular tremors. Pregnant cows may abort. If the

to greater than 1.5 mm. Division is by binary fission

condition progresses there are convulsions, paddling

and it produces morula-like colonies in the cytoplasm.

movements of the limbs, nystagmus, opisthotonus and
The small granules tend to be coccoid, with larger ones
chewing movements. Often a fetid, profuse diarrhoea is
looking like rings, horseshoes, rods and irregular
present or there may be blood in the faeces. A mild
masses. It has been suggested that differences in the size
cough may be heard. On auscultation hydrothorax,
and shape of the organisms are the result of a growth
hydropericardium and lung oedema are noted.
cycle.

Subacute: The signs are like those of the acute form but they are much less severe with a transient fever and

Epidemiology

sometimes diarrhoea. Disease may last for over a week
The disease has been reported in many African coun-
and the animal usually improves gradually. A few cases
tries south of the Sahara desert. Distribution coincides
progress to collapse and death. 'This is often the most
with that of the *Amblyomma* ticks, which require a
severe form seen in indigenous cattle and those previ-
warm humid climate and bushy grass. Experimentally,

ously infected.

five species of *Amblyomma* are able to transmit infection. These are three-host ticks. Transmission usually

Inapparent: These cattle include almost all the appears to be transtadial and so infected larvae are indigenous stock as well as some of those introduced to usually free from disease. Transovarian transmission the region. In addition they often follow cases of can occur, but is thought to be very infrequent. Infected reinfection.

larvae are found on non-susceptible animals, but if they do become infected it can pass on to both nymph and

Pathology

adult stages. Infected ticks do not transmit infection immediately; they become attached to the animals, but

The lesions present are very variable and not pathog-a variable time after they start to feed. In many cases

nomonic. In the peracute form there are few gross the level of infection is unknown as indigenous domes-

lesions, but in some there is marked lung oedema with

tic and wild animals often show no signs. It is only when

tracheal and bronchial froth. In the acute form there susceptible exotic species are introduced that infection is usually ascites, hydrothorax, hydropericardium and becomes apparent. Besides cattle, sheep, goats, Asian lung oedema. The lymph nodes are often swollen. buffalo, antelopes and deer are susceptible to infection Petechial haemorrhages can occur in the heart, lungs and disease. Indigenous cattle undergo inapparent and gastrointestinal tract. The liver is often engorged, infection. Calves under three weeks old, even from with the gallbladder distended. The spleen is occasion-susceptible stock, are difficult to infect. Heartwater can ally enlarged. There may be congestion of the occur throughout the year, but incidence declines in the meningeal blood vessels.

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Diagnosis

homogenized engorged *Amblyomma* ticks. The recipient animal is monitored and then treated with tetracycline. There is no completely specific method of diagnosis in clinics as soon as pyrexia develops. Treatment continues

the living animal. Provisional indication can be from the twice a day until the fever subsides. Pregnant cows history and clinical signs. Lymph node material can be should not be treated in this way.

aspirated to examine for vacuoles containing organisms in the cytoplasm of the reticular cells. There is a method of taking brain cortex so that the capillaries of the brain

Jembrana disease (Tabana disease)

can be examined for rickettsia. Blood can be obtained

Aetiology

and injected into susceptible animals. Eosinophils also decrease in number during the course of the disease.

The cause of the condition is not known, but it is

Serum can be examined using a capillary flocculation

thought to be a rickettsial infection. Groups of small

test. Diagnosis is easier at post mortem as the organism

coccobacillary organisms have been found in cytoplas-

can be discerned in brain tissue capillaries that have

mic vacuoles present in circulating monocytes and in

been fixed in methyl alcohol and stained with Giemsa.

impression smears of cut organ surfaces. Some consider

Differential diagnosis includes anthrax (p. 717) and the disease may have a viral aetiology.

acute theileriosis (p. 750) in peracute cases, and in nervous cases rabies (p. 908), tetanus (p. 733), strychnine poisoning, cerebral theileriosis (p. 752), cerebral

Epidemiology

babesiosis (p. 749) and hypomagnesaemia (p. 787).

The disease was first recognized in the Jembrana District of Bali Island, Indonesia, in 1964. Subsequently, the condition has only been detected in Indonesia. The

Treatment

disease is found in cattle as well as buffalo. Sheep and Therapy is most effective when carried out early in goats are infected with no apparent signs, but pigs are disease. Tetracyclines can be used and do not interfere refractory to infection. In cattle there is apparent age with development of immunity.

resistance to infection and cattle over two years old rarely die from the disease. Animals that recover are carriers, but the duration of infection is not known.

Control

There is no direct infection from animal to animal and

Disease can be prevented by controlling the vector

*so it is not contagious. *Boophilus microplus*, a pantropi-Amblyomma by dipping cattle at weekly intervals with*

cal cattle tick, appears to be the natural vector and it

reliable acaricides. However, the ticks of this genus are

is believed that infection can pass through the egg

less susceptible than those from other genera. As the

phase. The incubation period appears to be about a

tick may transmit infection after a day on the host,

month to six weeks, although it is considerably shorter

better control is obtained by applying acaricide by

after injection of infection. Mortality is about 25 per

dipping or spraying every three days. However, Ambly-

cent and is usually within a week of onset of signs. Some

omma have in some cases shown resistance to

animals relapse with infection at later dates.

organophosphorus and arsenic. Care should also be

taken not to introduce Amblyomma on infected

Signs

animals or in forage to uninfected cows.

In areas where disease is endemic most cattle are

Invariably there is a pyrexia of about 41°C (106°F) with

immune. A carrier state develops after infection and

anorexia, nasal and lacrimal discharge, which can persist

remains for several weeks. Non-infected resistance

for one to nine days. This is soon followed by enlarged

persists a variable time, lasting from a few months to

lymph nodes. Often there is excessive salivation and

several years. After this time reinfection can occur.

erosion of the oral mucosa. Petechial haemorrhages

Ideally, an effective vaccine should be used. However,

may be found on the visible mucous membranes and

at present C. ruminantium is difficult to culture serially haemorrhage is seen

within the aqueous humour of the

or to adapt to growth in laboratory animals. One

eye. Blood tends to ooze from the skin (so-called 'blood

method is to inject susceptible stock intravenously with

sweating'). Diarrhoea occurs early on and persists; it is

5–10 ml of blood from an infected animal. As infected

often bloodstained.

animals cannot always be available then infected blood

can be stored in a freezer or liquid nitrogen provided it

Pathology

is frozen rapidly after addition of dimethyl sulphonate.

The infection can also be retained in deep frozen

There are widespread haemorrhages and oedema.

brain emulsion or, more recently, in a supernatant of

Usually, generalized lymphadenopathy occurs, with the

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lymph nodes being hyperplastic and often showing dis-

or South America. The disease is transmitted by vectors

organization. Splenomegaly is common and the blood

from infected host cattle, which explains the seasonal-

vessels show vasculitis and perivasculitis. Surrounding

ity of the condition. If disease enters a new region it can

the blood vessels there is proliferation of lymphore-

cause epizootic infection with a morbidity of about 100

ticular cells in many organs except the liver. Rickettsia-

per cent, but mortality is usually less than 1 per cent. In

like organisms can be detected in impression smears.

the enzootic areas the condition is sporadic, with a mor-

bidity of 5–10 per cent.

The cause of transmission is thought to be midges,

Diagnosis

probably of the Culicoides spp. and Ceratopogonidae This is based on the area and history. Confirmation

*family. Transmission is not direct between animals, indi-
depends on the haematological changes, which include
cating the need for maturation in the vector. Spread
a progressive anaemia, thrombocytopenia and transient
mainly depends on the number of insects infected and
leucopenia, which particularly involves the lympho-
the direction of winds. Viral development in the insect
cytes. As the disease progresses 'foamy' monocytes
is suspected to be cyclic. More losses occur in adult
appear and large lymphocytes with coarse nuclei are
cattle, although calves from three months old are sus-
seen. However, confirmation is difficult unless the
ceptible. Often the disease seems to disappear only to
animal dies. It is then dependent on detection of
return again as an epizootic once resistance in the cattle
organisms in impression smears of cut organs and the
population is reduced.*

vascular changes. In some cases blood is taken into EDTA from suspect cases and then injected into sus-

Signs

ceptible cattle.

The incubation period is two to ten days and is followed

Differential diagnosis includes rinderpest (p. 707),

by a marked pyrexia of 40.5–41°C (105–106°F). This is

haemorrhagic septicaemia (p. 728) and plant poisonings

often very transient and so is missed. Other animals

(Chapter 54).

have intermittent pyrexia. There is a drop in milk yield,

which is the main loss caused by the disease, with

Treatment

anaemia, oculo-nasal discharge and increased saliva-

tion. Alimentary signs are variable and can include con-

Tetracycline injections during the course of the disease

stipation or diarrhoea. Within four days locomotory

appear to have little effect on disease severity or dura-

signs appear, including muscular tremors, which then

tion, but they do seem to reduce mortality.

develop into stiffness and weakness. The animal often

becomes lame and the hindlimbs may become stiff.

Control

Signs may resemble those of acute laminitis. Occasionally, animals show lateral recumbency.

Not enough is known about the disease to initiate

The animal usually starts to show signs of recovery

control measures. Recovered animals are often carriers after two or three days, with appetite and milk yield of infection and so resist further challenge.

improving. However, the stiffness and lameness are likely to take several more days to reduce. Recovery is uneventful. Mortality is low and is usually the result of

Viral diseases

secondary infection, or following aspiration pneumonia after regurgitation of ruminal contents or following

Bovine ephemeral fever (BEF)

lateral recumbency. Abortion does not occur, but semen

This is also known as bovine epizootic fever and three-quality in bulls is often affected for a period.

day sickness. Although signs occur they are quite mild and seem only to affect cattle. It is caused by a rhab-

Necropsy

dovirus, which can be present in the blood where it
The lesions are not specific. The main sign is enlarge-
appears to be mainly in the leucocyte-platelet fraction
ment and oedema of all the lymph nodes. Congestion
and is transmitted by insect vectors. At present there is
and petechial haemorrhages of the pleural membranes
only a single serological type of the virus, although in
may occur. Other signs are usually the result of com-
Australia several rhabdoviruses with a distinct rela-
plications such as aspiration pneumonia.

tionship to BEF have been isolated.

The condition is present in most of Africa except pos-

Diagnosis

sibly the north. However, the incidence varies widely
between regions and countries. It is also present in Asia,
This depends mainly on the signs of a mild disease with
Australia and New Guinea. It does not occur in North
pyrexia and lameness, limb stiffness and muscle

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tremors. There is a leucocytosis with a neutrophilia. A

also be by feeding at troughs or sucking milk from
fluorescent antibody test will detect virus in blood. An
infected cows. Indigenous cattle are less susceptible
agar gel immunodiffusion test has been successfully
than imported purebreds. Experimentally, giraffe and
used on serum. The virus can be isolated by serial
impala have been infected. All ages of stock are sus-
passage in Vero or BHK cell cultures or by intracere-
ceptible but calves and lactating cows are most likely to
bral injection of sucking mice.

be infected. The duration of immunity is not known and
appears variable. Reports suggest it to be from 11
months to five years, or even lifelong.

Treatment

Morbidity rates are very variable, from 5 to 80 per
Treatment is symptomatic but can include antibiotics
cent, with less than 2 per cent mortality. There can be a
for secondary complications, with the use of non-
rapid spread of the disease.

steroidal anti-inflammatory agents such as phenylbuta-
zone or flunixin meglumine for muscle stiffness. It is

Signs

best not to drench animals because of the high risk of aspiration pneumonia. Recumbent animals should be The incubation period lasts from two to five weeks in provided with adequate shade and water.

natural infection but four to fourteen days with experimental infection and is followed by a rise in temperature, which may fluctuate. In severe cases there tends to

Control

be anorexia, lacrimation and salivation. There is a clear Control of the insect vectors has not been successful.

nasal discharge, which later becomes purulent. There is

Immunity is long lasting after natural infection and has then the sudden appearance of nodules, varying in

led to the development of vaccines, which are available

number from a few to many hundred. The nodules are

in Japan and South Africa. Experimentally, live cell

firm, raised areas within the skin and vary in diameter

culture vaccines in an adjuvant base have been used

from a few millimetres to 4–5 cm. These larger areas

with success, but give rise to fears of vector transmis-

often have erect hair over them that exaggerates their
sion between vaccinated and non-vaccinated stock.

appearance. The nodules are usually of a uniform size

Control can also be achieved by dipping cows twice
on individual animals and they occur over the whole
weekly during the peak period of infection when con-
body, although they are mainly found on the back, neck,
ditions are wet, hot and humid.

brisket, legs, thighs, scrotum, udder and round the muz-
zle and eyes. Those on the mucosae of the muzzle, vulva,
prepuce, nostrils, eyes and mouth are yellow-grey in

Lumpy skin disease (LSD) (see also p. 887)

colour and soft, and if rubbed off there is an ulcer or

The disease, also called Knopvelsiekte, results in many
erosion. When the eye is affected there is keratitis and
pox-like skin lesions and has been associated with a
conjunctivitis. Respiratory lesions lead to dyspnoea and
virus that is serologically related to sheep pox and on
oral ones to salivation.

tissue culture produces three different groups of virus.

The associated lymph nodes tend to be enlarged. In

One, an orphan virus (group I), is generally present on some cases oedema develops. This can be of the lower the skin of normal cattle but does not appear to cause limbs, brisket, udder, vulva or scrotum. The enlarged the disease. The 'Allerton' virus (group II) causes a skin nodules can later ulcerate. These can then persist condition of a mild nature and is isolated from skin for several years or develop a dry surface that is lost. nodules, saliva, nasal secretions and semen. This form This becomes a deep pit, which heals by second inten- appears to be identical with the bovine herpes virus 2 tion. These wounds can become secondarily infected. of bovine mammillitis. It has often given rise to the This secondary infection can then spread to associated name of pseudo-lumpy skin disease (p. 888). The severe lymph nodes, the lungs, liver, kidney, etc. The areas disease is caused by a 'Neethling' virus (group III) and of superficial oedema also remain a long time and is found in blood, mucus, saliva and semen. can then slough, causing suppurative areas. Involve- The disease is seen in most parts of Africa and is

ment of the lung can lead to cicatrization and rupture
endemic in southern Africa. The virus is present on the
of the tracheal rings several months after onset of the
skin even when hides are salted, in blood and in saliva
disease.

and all can transmit infection.

The disease results in a chronic loss of condition and

The method of transmission has not been completely

milk yield. Secondary infection can lead to mastitis,

established, but it is believed to be by insects, particu-

abortion and sterility in bulls.

larly mosquitoes, as spread can occur without direct or

The 'Allerton' mild or pseudo-lumpy skin form of the

indirect contact. Virus has been isolated from *Stomoxys*

disease is less severe and only lasts a few weeks. There

and *Biomyia* flies. As the saliva is infected, spread can be a mild pyrexia followed
by nodule formation mainly

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on the head, neck and perineum. The nodules are char-

Rift Valley fever

acteristic with a hard, raised, rounded mass with a flat

This condition, also known as enzootic hepatitis, is a surface and a pit at the centre. Sometimes the skin zoonosis but mainly affects sheep and cattle. Camels lesions coalesce. Only the epidermis is affected, unlike can be infected and also goats to a limited extent. It is the 'Neethling' form. The problem area develops into a notifiable in many countries. It is caused by an insect-hard, dry, necrotic lesion after a week to nine days. This borne RNA virus of one main antigenic strain of is then lost over the next 10–14 days leaving a hairless the family Bunyaviridae, and genus phlebovirus. The area. Hair will grow again.

condition is transmitted between animals by insect vectors, usually mosquitoes. The disease was first identified in the East African Rift Valley, which gave rise to its name.

'Neethling' lesions include the whole depth of the skin, The condition is only found in Africa, particularly the producing a hard white-grey mass. The skeletal muscle central and southern regions, but the mosquitoes that

'Neethling' lesions include the whole depth of the skin, The condition is only found in Africa, particularly the producing a hard white-grey mass. The skeletal muscle central and southern regions, but the mosquitoes that

may contain grey nodules. Oedema of this is seen as are responsible for transmission occur on other conti-yellow jelly-like liquid. If the oral mucosa is affected it nents. Thus there is a possibility of spread. Disease is contains soft grey-yellow nodules with necrotic epithe-transmitted by various types of mosquito including lium. Necrotic ulcers with surrounding inflammation Aedes spp. Other biting arthropods would also cause can occur in the nasal cavities. In the lungs there are spread.

firm grey nodules and the whole tract may show ero-
Man can be infected by contact with infected animals, sions and ulcers. Pulmonary lesions may lead to oedema such as when undertaking necropsies. Infection may and a purulent pneumonia. When lymphadenitis is enter skin abrasions. The signs are of an influenza-like present the nodules are swollen, pale and oedematous. disease with pyrexia, severe headache, nausea, joint
The rumen and abomasal wall can show ulcers and ero-pains, flushing of the face, sometimes epistaxis and per-sions. Microscopically, the stratum papillomis, stratum

manently impaired vision due to retinal haemorrhage.

reticulis and subcutis are involved. Secondarily, the

Death is rare, but in some instances complications such

surface epithelium, hair follicles and their associated

as sight impairment can occur.

glands are infected. In the subcutaneous tissues there is

Usually, an epizootic outbreak occurs in cattle fol-

oedema, fibroplasia and perivascular inflammation with

lowed by long periods, often of five years, between out-

mononuclear cells, which usually results in thrombosis

breaks. The persistence of infection between epizootics

and overlying necrosis.

is unknown, but as cattle at the edge of and within

forests tend to seroconvert consistently it is considered

Diagnosis

disease is maintained in wild fauna.

Disease is more common in warm wet seasons. Young

The rapid spread of the disease and the sudden appear-

animals are more severely afflicted than the adults.

ance of skin nodules after pyrexia are relatively char-

Mortality tends to be high in young calves, often reach-

acteristic signs. However, a biopsy can be taken and
ing 70 per cent. In the adult, a mortality of 10 per cent
inclusion bodies can be found in skin lesions and the
can occur but the main problem is abortion.

virus can be cultured. Other diseases that may be con-
fused are the 'Allerton' form, urticaria (p. 883), der-
matophilosis (p. 886), demodicosis (pp. 743, 881),

Signs

onchocercosis (p. 774), besnoitiosis (p. 756) and severe
tick and insect bites.

The signs that develop depend partly on the age of the
animals. They tend to be most severe in the calf, where
the incubation period varies from 12 hours to three

Treatment

days. Occasionally, death occurs within 24 hours with

There is no specific treatment but secondary infection
few characteristic signs other than collapse and colic.
may require the use of antibiotics or sulphonamides.

Others show high fever, incoordination and collapse.

The rare renal form is acute and can occasionally occur
in adult cattle. In this type there is pyrexia, a profuse

Control

mucopurulent discharge, vomiting and prostration. Up to 70 per cent of affected cattle can die.

In some countries a sheep pox virus tissue culture

In adult cattle abortion is more common than the vaccine has been found to be of use. Otherwise, an acute form. However, up to 10 per cent can develop vaccine produced from attenuated 'Neethling' virus on marked pyrexia and die. Other lesions can include kidney tissue culture has been used successfully.

erosion of the oral mucosa and dry thickening of the

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unpigmented areas of the teats, udder and scrotum.

and fever (Chapter 51). The causes are included within

Hyperaemia of the coronets also occurs.

a complex of flaviviruses. The infections tend to be contained in various geographical areas. As a result there are a number of diseases that are identified by the area

Pathology

where the disease occurs. Thus, in Europe there is

The main lesion is one of hepatic necrosis with white or Russian spring-summer encephalitis, Omsk haemorrhagic foci in the subcapsular area. Other lesions are rhagic fever and Central European encephalitis. Tropical problems include Kyasamar Forest fever in India the pleura, pericardium, endocardium, lymph nodes and and Lanyot in Malaysia.

gut. There is often also oedema, congestion or haemorrhages of the gallbladder. Microscopically, there is focal in ticks, small rodents and insectivores. In the endemic or diffuse necrosis of the liver.

cases infection of wild and domestic ruminants is inapparent. However, the introduction of new ruminants can result in disease. As yet the infections of Kyasamar

Diagnosis

Forest fever, which have been found in man and horses, The history of an outbreak is helpful in that it is in a have not resulted in disease in native domestic cattle. mosquito area, there is an abortion storm plus calves

Infection in cattle would be via ixodial ticks. Man can becoming ill with many of them dying. Post-mortem be infected by ticks or, more commonly, via drinking examination of calves may show typical hepatic necro-infected goat's or sheep's milk. Infections from masis. There may also be evidence of human infection and terial submitted for laboratory diagnosis also occur.

if sheep are present then again in lambs there is disease Immunity following infection is good and lifelong.

of high fever, incoordination, collapse and high mortal-Colostrum results in passive immunity being conferred ity, with abortion in ewes. There is usually a severe to the young calf.

leucopenia. Serological confirmation is by means of a serum neutralization or complement fixation test.

Signs

Disease is almost always only apparent in animals intro-

Treatment

duced to the tick area. The incubation period is one

There is no known successful treatment, although inter-or two weeks. Pyrexia suddenly occurs and is usually

feron is effective in vitro and may eventually become diphasic. It is not until the second period of tempera-

ture rise that nervous signs appear. There is then inco-ordination, muscle tremors, ataxia, photophobia and hypersensitivity. The nervous signs become progres-

Control

sively worse. There can then be a flaccid paralysis, with

The passage from country to country is difficult to

death within a week. Those animals that recover are

control without the exclusion of susceptible animal

often subsequently debilitated.

species. Control of the insect vectors would be uneco-

nomic and impracticable. It is possible that humans

Necropsy

could act as carriers. A vaccine from neurotrophic virus

passage through mouse brain has been used success-

There are few gross lesions. On histological examina-

fully. However, it can cause abortion. In consequence,

tion there is necrosis of the neurones, particularly in the

a killed vaccine produced on BHK cells has been devel-

Purkinje layer of the motor nuclei, vestibular nuclei,

oped (p. 1018). This involves two injections to produce cerebellum and ventral nerves of the spinal cord.

adequate immunity. Good immunity has been produced Perivascular inflammation occurs throughout the white in sheep and humans by virus grown in rhesus kidney matter.

cell cultures and then inactivated by formaldehyde. A human vaccine is now available from virus raised in
Diagnosis

diploid fetal lung cells and inactivated with formalin. The diagnosis is based on the area, presence of ticks, signs and lesions. The virus can be isolated from the

Tick-borne encephalitides

central nervous system, particularly the brain stem and
(flavivirus infections)

spinal cord. Differential diagnoses include bovine

There are a series of diseases of animals resulting from spongiform encephalopathy (p. 909), rabies (p. 908; tick-borne infection and characterized by nervous signs
Chaptre 70),

neuromycotoxins,

plant poisoning

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(Chapter 54), tetanus, hypocalcaemia (p. 781) and

Diagnosis

hypomagnesaemia (p. 787).

The signs and post-mortem picture are not specific.

However, cerebral injection of infected brain tissue into

Treatment

rabbits will aid virus identification. The major disease

to be differentiated is rabies (p. 908).

There is no effective form of treatment.

Control

Treatment

There is no effective form of treatment, only sympto-

Cattle should not be introduced to endemic areas

matic.

unless they are vaccinated. The vaccines available

provide protection for at least a year. Control of ticks

by improving grazing and reducing bracken, etc., is

Control

useful.

There are no suitable control measures that can at present be suggested.

Tick-borne encephalitides

(Near East encephalitis)

Japanese encephalitis (flavivirus)

This condition is still not fully elucidated. However, there are several diseases that have been recognized for This is mainly a disease of man and has been given many years. The disease is endemic in some countries several local names, including Japanese B encephalitis, including Syria, Lebanon, Israel and Egypt and a similar Russian autumnal encephalitis and summer encephali-condition is recorded in India and Russia.

tis. Infection of animals is often from the human source.

In most cases the accidental infection of ruminants

Most disease is seen as encephalitis in horses, followed results in few cases of disease. Usually, the virus cycles by pigs. Mosquitoes are the natural vectors. Usually, inapparently within birds and ticks. Most clinical disease infection in cattle and other ruminants is inapparent is in Equidae, but occasionally problems have been

and of little overall significance.

recorded in sheep and cattle. The tick vector of Near East encephalitis is Hyalomma anatolicum. Infection

Other arthropod-borne diseases

passes through all stages of tick life including the egg.

All infection is tick mediated and it cannot be trans-

*There are well over 300 diseases that are arthropod
mitted directly between animals. Immunity follows
transmitted. Many are of limited geographical distribu-
infection in cattle but its duration is unknown.*

tion. Frequently areas do not produce overt disease.

*However, where signs do occur, they can be non-
specific with malaise and pyrexia. Often indigenous*

Signs

animals and wild animals develop inapparent disease

*The incubation period for the disease is unknown, but
that does not cause them any hazards or reduce pro-
may be four weeks. The severity of signs is variable and
duction. However, the introduction of new animals will
in the acute form there is a drowsiness and a pyrexia of
frequently result in overt disease, which can reach a*

about 40°C (104°F). However, in a few horses there are high morbidity level.

epileptiform fits with a progressive paralysis. Collapse and death can soon occur. In the subacute form pyrexia Akabane virus (Asian virus)

is usually transient and mild. Nervous signs are slight, but in some cases they can persist. Inapparent infection This bunyavirus is mosquito borne and related to Simbu tions are common.

virus. It is recorded in the Far East as well as Kenya and Australia. The condition in cattle is thought to produce arthrogryposis and hydrocephalus as well as abortion.

Necropsy

The alimentary and urogenital tract of the body show Bhanja virus (African virus)

mucosal congestion, but there is meningeal blood vessel congestion. On histological examination there is dif-

This is mainly an infection of goats but it has occasionally been isolated in cattle, particularly in southern microglial proliferation and neuronophagia and occa-

*Nigeria, and has also been reported in India. Infection
sional rarefaction of tissues.*

*has been found in Haemaphysalis ticks. Experimentally,
772 • Chapter 45*

*calves injected with Bhanja virus have developed a
infected experimentally and show a short-lasting
viraemia for several days and leucopenia.
viraemia.*

Bunyamwera virus (African virus)

Germiston virus (African virus)

*This is mainly a condition of goats, but serological evi-
This is a bunyavirus and is transmitted by mosquitoes.
dence has been found in cattle and sheep. The infection
The virus causes infection in humans, usually in labora-
occurs throughout Africa and is considered to be mos-
quitoes, but in cattle there are usually no signs but sero-
quito borne.*

*conversion. The condition is present in cattle, egrets and
herons.*

Cache Valley virus (American virus)

This is found in mosquitoes in Central and North

Harana virus (Asian virus)

America. Disease has not been recorded but sera of

The virus is transmitted by Hyalomma ticks and spread cattle are often positive.

to other animals by migrating birds. This virus resem-

bles, and may be identical to, Azo virus. It results in a

California encephalitis virus (American virus)

severe infection of humans, often with haemorrhages

and fever. It can be fatal. In cattle the disease is

In America there is a high incidence of antibody in the

transient and usually results in anorexia, pyrexia and

sera of cattle. Evidence of infection can also be found

depression.

in deer and horses. It is a bunyavirus transmitted by

mosquitoes. The virus is the most common cause of

human viral encephalitis in the USA.

Ibaraki virus (Asian virus)

It is thought to be arthropod borne and is a double-

Calovovirus (European virus)

stranded RNA virus. Antibody surveys show the infec-

tion to be widespread in South East Asia and the Far

This is a bunyavirus that is mosquito borne. It results in

*East. The condition is very similar to that of bluetongue.
cattle infection.*

*Disease is severe and only recorded in Japan. Signs
include pyrexia with oedema and haemorrhaging in
Congo virus (African virus)*

*the mouth, abomasum and around the horn/skin
junction. Degeneration of the muscles follows. There is
This is a disease of humans and also affects goats and
then dehydration and emaciation due to difficulty in
cattle resulting in anaemia, depression and pyrexia. The
swallowing. Death is commonly due to inhalation
virus is transmitted by Hyalomma ticks and infection
pneumonia.*

*between countries is probably accomplished by migrat-
ing birds. It is likely that the condition is related to
Haza virus and others in Europe and Asia. The disease
Jos virus (African virus)*

*is severe in horses and is often fatal. However, in cattle
and goats indigenous to Africa it results in a short
This unclassified virus is transmitted by Amblyomma
period of illness that may be seen as anorexia, fever and*

variegatum ticks. The disease signs are not really known depression.
but the virus has been isolated from indigenous
Nigerian cattle.

Corriparta virus (Australian virus)

Kodam virus (African virus)

The virus is mosquito borne, particularly by *Culex* spp.

The disease level is unknown but antibodies are found

This flavivirus is found in *Rhipicephalus parvus* ticks.

in the sera of cattle, horses and man as well as kanga-

The signs of disease are unknown but antibody is found
roos and wallabies.

in cattle.

Dughe virus (African virus)

Kokobera (Australian virus)

The virus appears to be spread by *Amblyomma varie-*

The virus has been found in mosquitoes in Queensland.

gatum ticks, *Culicoides* and mosquitoes. It is the most It is a flavivirus.
Antibodies have been detected in sera

frequently isolated arthropod-borne virus in Nigeria. In

from cattle and horses, although there is no indication

most cases there are no signs in cattle, but calves can be

that the virus is a pathogen.

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Kowanyama virus, Mapputta virus, Trubanaman

*spread by mosquitoes. The virus appears to be related
virus (Australian viruses)*

to Rwamba virus.

*These are all found in tropical Australia and can be iso-
lated from the Anopheles mosquito. Their pathogenic-*

Rwamba virus (African virus)

ity is not known but antibodies are found in the sera

*Cattle sera will show antibodies to this virus but disease
of cattle, sheep and pigs as well as kangaroos and
is not apparent. The name also applies to infection in
wallabies.*

humans, donkeys, goats and sheep. The condition is

spread by mosquitoes. The virus appears to be related

Kunjin virus (Australian virus)

to Pongola virus.

This is a flavivirus found in mosquitoes in tropical

Australia. There is serological evidence of infection in

Sabo virus (African virus)

humans, horses, cattle and poultry. In calves experi-

This virus is found in Culicoides in Nigeria. It is also mentally infected a mild, non-purulent encephalitis

frequently isolated from Nigerian cattle and has been develops.

isolated from goats. There is a transient fever and listlessness following infection. The virus is a member

Kotonkan virus (African virus)

of the Simbu group of bunyaviruses.

This is a rhabdovirus found in Culicoides. The disease resembles ephemeral fever virus. The infection is par-Sango virus (African virus)

ticularly present in Nigeria.

This virus is found in Culicoides in Nigeria. It is also frequently isolated from Nigerian cattle. There is a tran-

Lokern virus (American virus)

sient fever and listlessness following infection. The virus is a member of the Simbu group of bunyaviruses.

Antibodies to the bunyavirus have been found in cattle, horses and sheep. The infection is found in Californian sandflies and mosquitoes.

Shamonde virus (African virus)

This virus is found in Culicoides in Nigeria. It is also fre-Middelburg virus (African virus)

quently isolated from Nigerian cattle. There is a transient fever and listlessness following infection. The virus While antibodies can be found in domestic cattle in is a member of the Simbu group of bunyaviruses.

South Africa, disease has not been recorded. Man is not affected. The virus is spread by *Aedes* mosquitoes.

Shuni virus (African virus)

Murray Valley encephalitis virus (Australian virus)

This virus is found in *Culicoides* in Nigeria. It is also frequently isolated from Nigerian cattle and has been

This is a flavivirus found in mosquitoes and wild birds.

isolated from sheep. There is a transient fever and

It can produce epidemics of human encephalitis in listlessness following infection. The virus is a member

Australia and New Guinea. The infection can be of the Simbu group of bunyaviruses.

detected serologically in cattle, horses, pigs and dogs.

When calves are experimentally infected the disease is symptomless.

Sindbis virus (African, Asian and Australian virus)

The life cycle is believed to involve *Culicoides*, mos-

quitos and wild birds. In most cases cattle exhibit no

Obodhiang virus (African virus)

signs but they do develop antibody titres. Infection

This is a rhabdovirus found in Culicoides. The disease of humans results in illness. It is a species of the

resembles ephemeral fever. It is found in mosquitoes in alphaviruses.

Sudan and the pathogenicity is not known.

Thogoto virus (African and European virus)

Pongola virus (African virus)

The virus is found in ticks in Nigeria and in Boophilus

Cattle sera will show antibodies to this virus but disease

decoloratus ticks in Kenya. It has also been isolated in is not apparent. The name also applies to infection in

the tick Rhipicephalus bursa in Sicily. The infection can humans, donkeys, goats and sheep. The condition is

also involve humans and sheep. Calves, when experi-

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mentally infected, show viraemia and leucopenia but

Treatment

usually there are no other signs.

There is no specific treatment but diethylcarbazine

citrate 4 mg/kg in the food can be helpful, otherwise

PARASITIC DISEASES

ivermectin, doramectin and eprinomectin can be used.

Onchocercosis (worm nodule disease)

Control

Aetiology

The control of insect vectors is not practicable.

This is a filarial infection of the genus Onchocerca. In cattle, Onchocerca gibsoni affects the subcutaneous

tissues, particularly the brisket and lower limbs; O.

Stephanofilariosis

liendis (synonyms O. gutturosa, O. bovis) is found in the Aetiology

ligamentum nuchae, other ligaments and stifle joints; O.

ochengi causes a dermatitis and O. armillata is found This is caused by parasites of the genus Stephanofilaria.

in the aortic wall. The worms are thread-like and

These include S. assamensis, which occurs in India

often measure 6 cm long with microfilariae 200–400 mm

and Pakistan, S. kaeli, found on the legs of cattle in long.

Malaysia, S. stilesi, affecting the ventral surface of the body and found in many parts of Asia, and S. dedosi,

which occurs in Indonesia affecting the head and neck.

Epidemiology

The parasites are small (2–9 mm long) and filariform in

*The condition occurs worldwide, but is more common
shape.*

in the tropics and subtropics. The life cycle is indirect.

*Transmission is by midges (*Culicoides* spp.), sandflies and blackflies (*Simulium* spp.), which ingest microfilaria*

in the skin and subcutaneous tissue. They develop to

The parasite occurs in cattle and buffalo in many parts

the infective larval stage and then infect the final host

of South East Asia as well as the tropics. The life cycle

when the vector again feeds. The larvae migrate to

is indirect and involves anthomyid flies as intermediate

*the predilection site where *O. gibsoni* develops into*

hosts. Open skin lesions develop from which the flies

nodules and the others become enclosed in a fibrous

feed and ingest the microfilariae. In the fly they develop

cyst. The females produce microfilariae that remain in

to the infective larval stage and are then transmitted to

the skin or subcutaneous tissue.

the final host when the fly feeds. There appear to be

more flies when conditions are moist either due to rain

Signs

or presence of rivers, streams or irrigation.

There are few clinical signs except for the presence of nodules up to 3 cm diameter under the skin, particularly

Signs

in the brisket. In O. liendis infections there are few signs in the ligamentum nuchae but the stifle joint may be

There are only superficial lesions, seen initially as a swollen. Disability can occur due to the supporting lig-

popular dermatitis. There is then exudate and haemor-

aments being affected. Infestation with O. gibsoni can rhyme lasting many months with the skin becoming

result in nodules.

thickened and dry.

Pathology

Diagnosis

There are nodules present. Onchocerca armillata is

This is based on the lesions and recent wet weather.

present in nodules in the wall of the thrombic aorta; O.

Scrapings or smears from skin lesions reveal adult

liendis is found in the ligamentum nuchae, ligaments, worms and microfilariae.

stifle, the omentum, splenic ligament and capsule.

Diagnosis

Treatment

The presence of infection can be detected by examin-

Topical application of organophosphorus compounds

ing skin biopsies. Differentiation from skin tuberculo-

is effective, including trichlorophan 6 per cent and

sis (p. 743) and demodectic mange (p. 781) is necessary.

coumaphos 2 per cent.

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Control

Control

There is no effective control but in some cases fly repel-

The condition is not usually severe and so control is not

lents are used. However, these are expensive for routine

normally undertaken particularly because of the ubiq-

use. In adult animals that have been repeatedly infected

uitous nature of the vector.

with S. stilesi there is evidence that further infestation is reduced or stops.

Other problems

Thelaziosis

Tick paralysis

Aetiology

Aetiology

Disease is caused by infestation with spirurid nema-

todes of the genus Thelazia, in cattle by T. rhodesii. The This occurs with a number of different ticks and results

worms are thin, white and can be up to 20 mm long.

in an ascending flaccid paralysis either due to acetylcholine failure at neuromuscular sites or a lack of conduction within the nerve fibres.

Epidemiology

It occurs in the conjunctival sac of mammals in many parts of the world. In the species that have been studied

Epidemiology

the worms are viviparous and produce first-stage

This is seen in ticks in Australia (Ixodes holocyclus), larvae that are infective for various Diptera, including

North America (Dermacentor andersoni, D. acciden-

Musca spp. The flies act as the intermediate hosts where talis) and South Africa (I. pilosus, I. rubicundus, the parasite develops to a third-stage infective larva,

Haemaphysalis punctata). Most cases coincide with the

which is transferred to the final host when the flies

peak tick populations and particularly at maximum feed.

adult activity. At times only one feeding tick may cause the problem.

Signs

There are often no signs, otherwise there may be

Signs

lacrimation that can be unilateral or bilateral and is

The signs involve a change in temperament followed often purulent. There can be conjunctivitis, keratitis, by slight incoordination. Then the animal starts to drag corneal opacity, ulceration, protrusion of the eyeball its hindlimbs and as the forelimbs are progressively and photophobia. Abscesses may develop on the involved it becomes recumbent. There is respiratory eyelids.

distress and then the animal dies of respiratory failure.

Pathology

Necropsy

There may be conjunctivitis, keratitis and corneal ulceration and scarring.

There are no characteristic signs at post mortem.

Diagnosis

Diagnosis

This depends on the signs and finding the worms.

If the ticks are removed the animal makes a recovery.

Treatment

Treatment

The most effective treatments are the avermectins iver-

*Remove as many ticks as possible by hand, then treat
mectin, doramectin and eprinomectin at 200 mg/kg. Lev-
with an ixodicide.*

*amisole can be given orally at 5 mg/kg body weight or
as a 1 per cent eye lotion. Otherwise, local anaesthetic
can be used and the helminths removed manually. Irri-*

Control

*gation of the unanaesthetized eye can be undertaken
with 1 in 8000 aqueous iodine or 2 per cent boric acid,*

*Although susceptibility decreases with age, immunity
3 per cent piperazine adipate or 0.2 per cent diethyl
varies from a few weeks to years. Prevention really
carbamazepine.*

involves the proper use of dips, sprays, etc.

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Sweating sickness (sweetsiekte,

Inapparent: These animals show few signs but there is

notkalersiekte, vuursiekte,

a form of residual immunity to the problem.

schwitzkrankheit, la dyhydrose tropicale,

foma, ol macheri)

Pathology

This appears to be a toxicosis with a dermatotropic

toxin related to tick infestations. Removal of the ticks

The lesions depend on the duration of the condition

results in a rapid clinical recovery. The problem is not

and any concurrent disease. There is a disseminated

transferred from animal to animal by the introduction

intravascular coagulopathy with microthrombi (Van

of blood, saliva or tissue from ill animals. Animals

Amstel et al. , 1987). The oral mucous membranes show

appear to be susceptible or non-susceptible. In the

ecchymotic dermatitis, inflammation and superficial

former case cattle become ill with each exposure and

necrosis. The nasal cavity, pharynx, larynx, oesophagus can die. Only certain strains of the tick *Hyalomma truncatum*, known as the boat-legged tick, are implicated. In the thorax there is hydrothorax, hydropericarditis with oedema and emphysema. The liver displays fatty degeneration and the abomasal, small and large intestine. Although cattle are involved, particularly calves, sheep, goats, pigs and dogs are also affected. The signs vary mucous membranes show hyperaemia.

according to individual susceptibility, age of the animal and the number of the correct strain of tick present. The

Diagnosis

inactive period is about six days, but varies from four to eleven. The morbidity of the condition is very variable This is dependent on the area and local history of the and mortality ranges from 30 to 70 per cent. Immediately following recovery from sweating sickness immunisation. Pathological signs are helpful and there is an

nity persists for well over a year and in some cases lasts increased prothrombin clotting time. Peracute cases up to four years. There appears to be no passive transfer of immunity to calves in colostrum.

(p. 748), anaplasmosis (p. 761), heartwater (p. 765) and poisonings (Chapter 54).

Signs

Treatment

Peracute: This is fatal in 48–72 hours, with a sudden rise in rectal temperature, hyperaemia, anorexia, dyspnoea,

There is no specific drug that will affect sweating sick-

hyperaesthesia of the visible mucous membranes and

ness, but hyperimmune serum is useful in treatment.

muscle tremors. There tends to be excessive lacrimation

The inflammatory nature of the disease suggests the

and nasal discharge.

use of anti-inflammatory agents, but corticosteroids

are probably contraindicated as there are often high

circulating cortisol levels in affected cattle. The use

Acute: This only occurs in animals under a year old and of antibiotics is indicated because of the likely

presents as a sudden rise in temperature to 40–41°C immunosuppression that can occur in the presence of (104–106°F) for a period of up to eight days. The high cortisol levels. As there is severe intravascular pyrexia may be continuous or intermittent and lasts coagulation the use of heparin at 10–20 iu/kg body longer in the latter case. The mucous membranes weight may be helpful. The liver is often affected and become hyperaemic and there is lacrimation, salivation so multivitamin injections, particularly containing and nasal discharge. The hair is in poor condition and cyanocobalamin, choline and methionine, may be of wet eczematous areas develop involving the head, par-use.

ticularly the cheeks under the eyes, nose, ears, then the neck, abdomen and flanks, and especially the groin. In some cases the whole body may be affected and accom-

Control and prevention

panying the eczema there is hyperaesthesia, discharge Effective control of the ticks responsible with acaricides and loss of the surface epithelium. Many badly affected

is helpful. Eradication is usually not feasible and so acaricides show total hair loss.

acaricide regimens should be designed to clear short-term infestation with the ticks, insufficient to produce dis-

Subacute: The signs are a sudden rise in temperature, distress but of long enough duration to allow immunity to develop but to a lesser extent and lasting only two or three days, develop. As the tick needs to be attached to the animal and the mucous membranes show slight hyperaemia, as for at least 7–10 days to produce disease, weekly dipping can the skin.

dipping normally produces reasonable control.

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Mhlosinge

dromes in dogs and cattle resulting from severe tissue

damage. *Contributions to Microbiology and Immunology*, 7, This is a less important toxicosis that is transmitted by

103–19.

certain strains of *Hyalomma truncatum*. It affects cattle, Murray, M. (1978) *Anaemia of bovine African trypanosomiasis-sheep and pigs*. The signs are usually milder than sweat-

sis: an overview. In *Pathogenicity of Trypanosomes*. Progressive sickness. Cattle do not die of the disease but show

*ceedings of a workshop held at Nairobi, Kenya, November
pyrexia and anorexia. There is no cross-immunity
1978.*

between sweating sickness and Mhlosimge.

*Mwongela, G.N., Kovatch, R.M. & Fazil, M.A. (1981) Acute T.
vivax infection in dairy cattle in Coast Province, Kenya.
Tropical Animal Health and Production, 13, 63–9.*

Magudu

*Nantulya, V.M., Musoke, A.J., Rurangirwa, F.R., Saigar, N. & Minja, S.H. (1987)
Monoclonal antibodies that distinguish*

This is also a less important toxicosis transmitted by

*T. congolense, T. vivax and T. brucei. Parasite Immunology, some strains of H.
truncatum. Again the disease is*

9, 421–31.

non-fatal and can affect cattle, sheep and pigs. The signs

*Njogu, A.R., Dolan, R.B., Wilson, A.L. & Sayer, P.D. (1985) are milder than
those of sweating sickness, including*

*Trypanotolerance in E. African Orma Boran cattle. Veteri-
anorexia and pyrexia. Cattle previously infected with
nary Record, 117, 632–6.*

sweating sickness are immune to Magudu.

Paris, J., Murray, M. & McOdimba, F. (1982) A comparative

evaluation of the parasitological techniques currently available for the diagnosis of African trypanosomiasis in cattle.

Stomatitis–nephrosis syndrome

Acta Tropica, **39**, 307–16.

Pilson, R.D. & Leggate, B.M. (1961) A diurnal and sea-

This condition is reported in cattle in Zimbabwe. It is a

sonal study of the feeding activity of Glossina palli-

toxicosis but it is not known whether it is of the sweat-

dipes. Australian Bulletin of Entomological Research, **53**, ing sickness type or a different *Hyalomma toxicosis*.

541–9.

Rae, P.F. & Luckins, A.G. (1984) Detection of circulating

trypanosomal antigens by enzyme immunoassay. Annals of

References

Tropical Medicine and Parasitology, **78**, 587–96.

Stephen, L.E. (1986) Trypanosomiasis – A Veterinary Prospec-

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tive. Pergamon Press, Oxford.

Trail, J.C.M., Sones, K., Jibbo, J.M.C., Durkin, J., Light, D.E. & Urquhart, G.M., Armour, J., Duncan, J.L., Dunn, A.N. &

Murray, M. (1985) Productivity of Boran cattle maintained

Jennings, F.W. (1987) Veterinary Parasitology, pp. 1–286.

by chemoprophylaxis under trypanosomiasis risk. ILCA

Longman Scientific, Harlow.

Research Report No. 9, Addis Ababa.

Whiteside, E.F. (1962) The control of cattle trypanosomiasis

Trypanosomosis

with drugs in Kenya: methods and costs. East African Agri-

cultural and Forestry Journal, 28, 67–73.

Banks, K.L. (1978) Binding of Trypanosoma congolense to the walls of small blood vessels. Journal of Protozoology, 25, 241–5.

Other problems

Clarkson, M.J. (1976) Trypanosomiasis of domesticated

*animals of S. America. Transactions of the Royal Society of Van Amstel, S.R.,
Reyers, F., Oberem, P.T. & Mathee, O. (1987) Tropical Medicine and Hygiene,
70, 125–6.*

Further pathological studies of the clinical pathology of

Hoste, C. (1987) Trypanotolerant livestock and African animal

*sweating sickness in cattle. Onderstepoort Journal of Veteri-trypanosomiasis.
World Animal Review, 62, 41–50.*

nary Research, 54, 45–8.

*Losos, G.J. (1986) Trypanosomiasis. In Infectious Tropical Diseases of Domestic
Animals, pp. 183–318. Longman, Harlow.*

Masake, R.A., Nantulya, V.M., Akol, G.W.O. & Musoke, A.J.

(1984) Cerebral trypanosomiasis in cattle with mixed T.

Further reading

congolense and *T. brucei* infections. *Acta Tropica*, **41**, 237–46.

Sewell, M.M.H. & Brocklesby, D.W. (1990) *Handbook on*

Morrison, W.I., Murray, M., Whitelaw, D.D. & Sayer, P.D.

Animal Diseases in the Tropics, 47th edn, pp. 1–385, Baillière (1983) *Pathology of infection with T. brucei: disease syn-Tindall*, London.

Adult Cattle

Metabolic Problems

Chapter 46

Major Metabolic Disorders

R.G. Eddy

Milk fever (parturient paresis, hypocalcaemia, eclampsia)

781

Breed: The Jersey and, to a lesser extent, the Guernsey Hypomagnesaemia (grass tetany, grass staggers, lactation

are particularly susceptible to milk fever. This would

tetany, Hereford disease)

787

indicate a genetic predilection for this disease and is

Hypophosphataemia

791

probably related to the relatively high production level

Postparturient haemoglobinuria

792

for a small breed.

Acetonaemia (ketosis, slow fever)

793

Pregnancy toxaemia

796

Age: It is rare for milk fever to occur at the first calving The downer cow

797

Fatty liver syndrome

801

and relatively uncommon at the second. The incidence does appear to increase with age and incidence levels of 20 per cent or more are common at the sixth calving

Milk fever (parturient paresis,

and beyond. The reason is thought to be that the re-

hypocalcaemia, eclampsia)

quirement for calcium at parturition increases as milk yield rises with each lactation and the ability to mobilize calcium quickly from the body reserves, i.e. bone,

Milk fever or hypocalcaemia is probably the most

decreases with age.

common metabolic disorder affecting cattle. It is normally associated with parturition occurring just before,

Seasonal factors: The incidence in the UK is highest in during or immediately after calving although it has

the months of August to October and at its lowest in

been reported in dry cows and, increasingly, during mid

the winter months of December, January and February.

lactation. The incidence of milk fever is higher in dairy

However, this is more likely to be a result of differing

cows than beef cows and increases with age and yield.

feeding regimens of dry cows than a seasonal effect. In

Milk fever undoubtedly increased in incidence during

countries where there is little change in dry-cow nutri-

the 1970s and 1980s when levels of around 9 per cent

tion during the year, e.g. Israel, there appears to be no

per annum were being reported. More recent studies

seasonal differences in the occurrence of milk fever.

would suggest that the incidence has fallen to around

5–6 per cent, in spite of increased milk yields, and prob-

Nutritional factors: The wide range of incidence of milk fever is probably the result of improved nutrition of dry cows. The

fever observed between seasons, within a season and incidence does vary considerably between seasons as between farms in the UK is due to the variation of well as between farms. In some years the incidence in nutritional input given to dry cows. Dry cows that are September and October in the UK can be as high as fed a diet of hay or silage only, which is now common- 60 per cent on some farms, whereas during the winter place in the UK, will have a low incidence of milk fever months when dry cows are housed 0–6 per cent would at calving. However, if feeds containing high calcium be frequently reported. The disease does appear to be levels are included in the diet, e.g. sugar beet pulp or more common when dry cows are fed grass rather than high calcium minerals, the incidence will increase. conserved fodder. Milk fever is a common cause of The incidence of milk fever in cows at grass varies death and is probably the most common cause of appar- considerably with the season. In dry weather the inci- ent sudden death in dairy cows. It is also a common dence is low, but during long wet spells the level is high.

cause of dystokia and hence stillborn calves.

A diet of grass with a low dry matter (DM), whether in the spring or autumn, can predispose to high incidences

Predisposing factors

of milk fever. There is more than one reason for this.

There are several important predisposing factors that

One is that such a diet leads to diarrhoea, which probably influence the occurrence of milk fever and these

ably reduces the calcium available for absorption. The

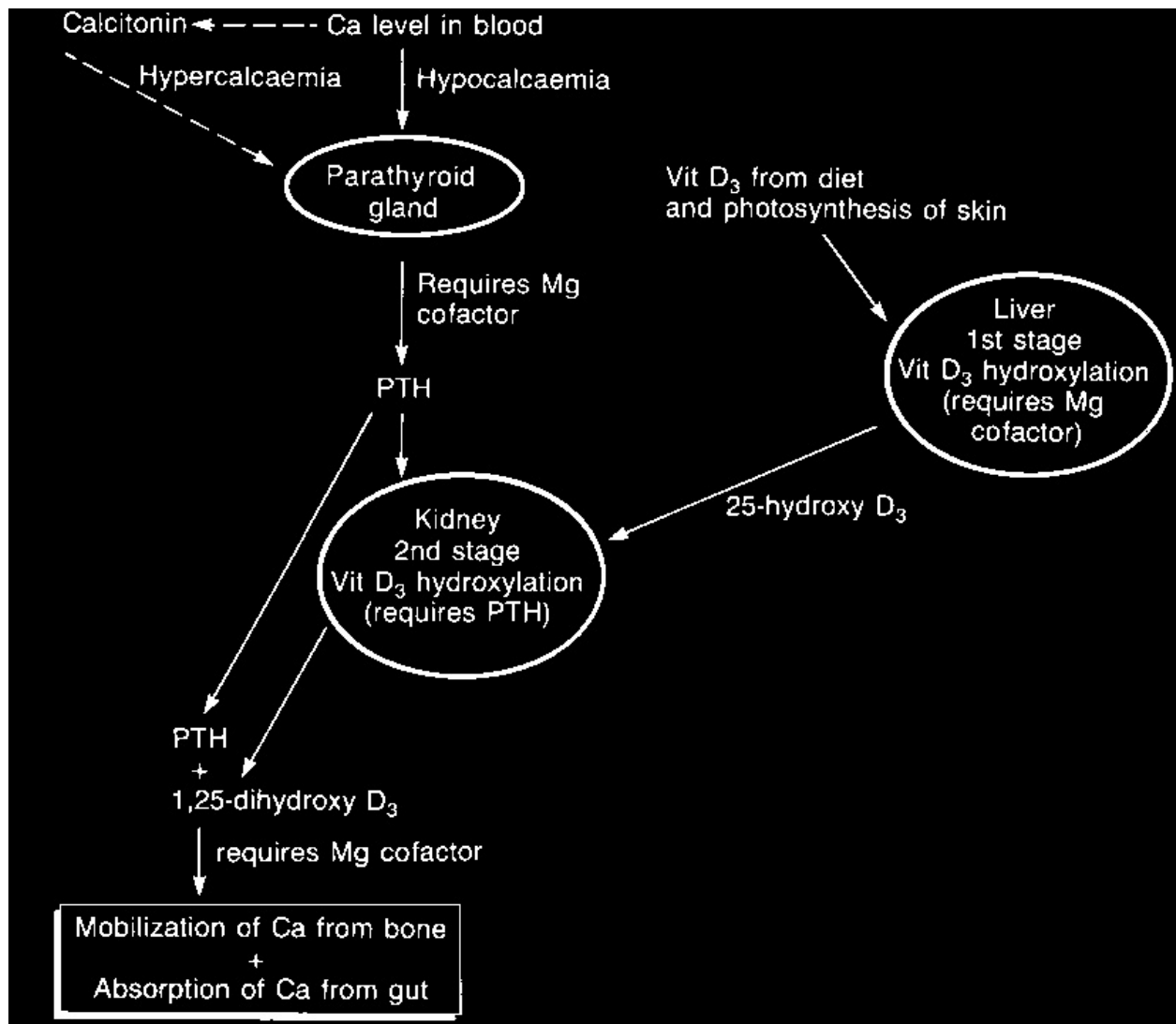
account for the wide variation of incidence observed in

calcium level of wet grass, particularly in the autumn,

the UK and other countries worldwide.

can be excessively high, often as high as 1 per cent of

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DM. Potassium levels may also be high. It has been shown that low magnesium levels in the diet restrict the ability of the cow to absorb calcium (Sansom et al. , 1983) and fold in a very short period. In order to meet this requirement is increased two- or three-fold in a very short period. In order to meet this the high levels of milk fever found in the spring and

enlarged demand and to avoid hypocalcaemia, calcium
autumn in the UK are often due to low magnesium
absorption from the gut is increased and further
levels in the grass.

calcium is available from mobilization of the calcium
reserves in bone. This explains why mild hypocalcaemia
occurs in all cows at parturition. If the cow does not

Aetiology

respond quickly to the sudden increase in calcium
requirement the hypocalcaemia deepens and signs of
Some degree of hypocalcaemia occurs in all cows at
milk fever become apparent.

parturition, but only when this becomes severe do

In the normal cow this adaptation process at parturi-
clinical signs develop. Frequently, hypophosphataemia
tion is under the hormonal control of the parathyroid
and hypermagnesaemia accompany the hypocalcaemia,
hormone (PTH). Hypocalcaemia stimulates the secre-
although when milk fever is due to low magnesium
tion of PTH, which in turn stimulates the production of
intake the blood levels of magnesium will also be

a hydroxylase enzyme in the kidney that is able to synthesize 1,25-dihydroxycholecalciferol (1,25(OH)₂D₃),

which is produced from vitamin D

10.4 mg/100 ml), but will fall to 1.5 mmol/l (6.0 mg/

3. The 1,25(OH)₂D₃

stimulates increased gut absorption of calcium and probably the mobilization of calcium from bone (Fig. 46.1). signs occurring. Usually, when milk fever is present the plasma level will be in the range 0.75–1.5 mmol/l adaptation process:

(3.0–6.0 mg/100 ml).

The predisposing factor in the aetiology of hypocal-

- Age of the cow, as already mentioned. Older cows are less able to mobilize calcium from the requirement of calcium for the production of

skeleton.

colostrum. The daily calcium requirement of a 600 kg

- Oestrogens also inhibit calcium mobilization and as cow in late pregnancy is approximately 28–30 g; this oestrogen levels rise at parturition this will have a comprises 13–15 g required for endogenous loss in negative effect on the adaptation process to maintain calcium levels. Milk fever does occasionally At the onset of lactation, approximately 1–1.5 g of occur during lactation, usually in association with*

Fig. 46.1

System for mobilizing calcium in cattle.

PTH, parathyroid hormone (Kelly, 1988).

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oestrus. This again would be due to the inhibitory tympany and total absence of the pupillary light reflex effect of oestrogens.

until the cow dies. Death can be due to paralysis of the

- Food intake is often depressed at or around partu-respiratory muscles, but more often death occurs from*

rition so the total available calcium in the diet will bloat. Many cows with milk fever are found in ditches be reduced. This is particularly the case if diets low or streams, having fallen in during the incoordination in calcium, e.g. straw or cereal-based diets, are fed phase of the disease. In these situations death can be over the parturition period.

due to drowning.

- The calcium intake during the dry period. If high The length of time for the disease to progress from levels of calcium are present in the diet the reduced first signs of inappetance to death varies from 10 to 24 PTH output that occurs reduces the rate of absorption. Many cows are found dead or near to death by tion from the gut, so that when the demand for calcium suddenly increases and the cow's appetite is been quite normal the previous evening at 7.00 pm. generally reduced absorption does not satisfy the If hypocalcaemia occurs at the onset of calving the body requirement (Fig. 46.1).

parturition process will cease due to lack of myometrial

- *A low magnesium intake in the diet reduces by contractions. A considerable number of cases of dysvarious mechanisms the ability of the gut to allow tokia due to uterine inertia are the result of hypocalcalcium absorption. Therefore, diets that are caemia and, if not treated, will result in a stillborn calf deficient in magnesium will predispose to hypo- or even death of the cow before the calf is born. It is calcaemia by reducing calcium absorption.*

common for the stockworker to find a cow in lateral Hypomagnesaemia also inhibits mobilization of recumbency in the early morning with the calf precalcium from bone (Fig. 46.1).

sented in the birth canal or even with the head present

- *Problems associated with digestion, e.g. acidosis and at the vulva. Frequently, the calf will be dead and occa- profuse diarrhoea, will reduce the amount of sionally even partially eaten by foxes or other wildlife. calcium in the gut available for absorption. This could also explain cases of hypocalcaemia that occur*

Clinical pathology

at times other than parturition or oestrus.

The concentration of calcium in plasma is usually below 1.5 mmol/l (6 mg/100 ml) in cows with clinical hypocal-

Signs

caemia and will fall to as low as 0.25 mmol/l (1.0 mg/

The clinical signs of milk fever are progressive. In the 100 ml). Phosphorus levels also fall to 1.0 mmol/l from first stage there is a loss of appetite, lethargy and the the normal range of 1.4–2.5 mmol/l (4.3–7.8 mg/100 ml).

rectal temperature is reduced by 0.5°C (1°F). This pro-

Magnesium levels usually increase to around 1.25

gresses to a stage where the cow stands with the hocks mmol/l (3.0 mg/100 ml), except where the cause of milk straight and sways laterally, particularly when moving.

fever is related to low magnesium diets when hypo-

Constipation is normally seen at this stage. Muscle magnesaemia may be present. Hyperglycaemia is also tremors may be present about the head and limbs.

usual during milk fever, but this is frequently seen in

Hyperaesthesia is also often evident at this stage and

normal cows at parturition.

the cow becomes apprehensive. The lateral swaying

In fatal cases of milk fever there are no gross or his-

develops into incoordination and ataxia and the cow

tological lesions characteristic of the disease. Bruising

will fall over sideways and rise with increasing difficulty

of subcutaneous tissue and muscles due to localized

until she becomes permanently recumbent.

trauma may be apparent. The liver is occasionally dis-

The recumbent stage is the one most commonly seen

tended and infiltrated with fat, resulting in a yellow

by veterinarians in practice. The cow will be sitting in

discoloration. Cows with such a fatty liver are thought

sternal recumbency, often with a noticeable S bend

to be more prone to milk fever, but fatty liver is not

in the neck, progressing to the stage where the head

pathognomonic of milk fever.

will be resting on her shoulder. The heart rate will be

slightly raised but rarely exceeds 90/minute. The pupils

Diagnosis

will be dilated and the pupillary light reflex will be

reduced or even absent. The gut stasis that characterizes this stage of the disease further reduces the availability of calcium for absorption and the disease will progress to the comatose stage. The rumen ceases to function and often becomes tympanic and the cow is unable to obtain a result. A cowside test for calcium appears severely depressed. The comatose stage is now available in some countries but its value is probably limited to differential diagnosis of the downer cow

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syndrome or cows failing to respond to treatment for slipping on ice or slippery concrete or even a collision with a vehicle such as a tractor.

is, once the differential diagnoses have been eliminated, the response to treatment. Intravenous treatment with

Hypomagnesaemia: This can occur at any time of lactation during the spring or autumn but is sometimes within minutes. The cow will become more alert, defecate and eructate, often before the full dose of calcium has been administered.

autumn. Hyperaesthesia is the main differentiating sign of hypomagnesaemia.

Differential diagnosis

Downer cow syndrome: Initially, this may be difficult to differentiate from milk fever but response to therapy

Although milk fever is an extremely common disease it and clinical biochemistry will be helpful. This will be essential that a full clinical examination be made for discussed in full later in the chapter (see p. 797).

every case. It is common for the new veterinary graduate to diagnose and treat a recumbent cow for milk

Inanition: Any condition producing severe weight loss fever when the cause of the recumbency is toxic masti-

will result in recumbency, particularly in pregnant cows, tis. The clinical examination must therefore include the most common of which is probably starvation. In examination of milk from all four teats, the heart rate seasons where insufficient conserved food is available, and the mucous membranes. Misdiagnosis is now more recumbency due to starvation is common. Body condition common as farmers treat their own cases of milk fever tion is the obvious differentiating feature. However, any and frequently confuse the disease with other causes of disease that causes considerable weight loss will have recumbency.

the same effect, e.g. liver fluke (p. 276). In seasons when

The differential diagnoses of milk fever are:

liver fluke prevalence is high, this disease can result in

- Acute toxic mastitis (Chapter 23);

extreme weight loss and anaemia and recumbency prior

- Calving paralysis (p. 438);

to parturition is common. Weight loss, red cell count

- Physical injury (p. 446);

and presence of fluke ova in faeces would be the dif-

- *Hypomagnesaemia* (p. 787);

ferentiating features.

- *Downer cow syndrome* (pp. 439, 789);

- *Inanition and other disease* (p. 150);

Pregnancy toxaemia: This can occur, particularly in

- *Pregnancy toxaemia* (p. 796);

beef cows, in the last two or three months of pregnancy.

- *Acidosis* (p. 829);

The sweet smell of acetone on the breath, poor condi-

- *Hypothermia* (p. 930);

tion or excessive fat and a history of unavailability of

- *Bovine spongiform encephalopathy (BSE)* (p. 909).

food should distinguish this problem. The rumen will also be functioning in the early stages of this condition.

Acute toxic mastitis: In acute toxic mastitis from

whatever cause the temperature may be raised but is

Acidosis: Acute acidosis as a result of the sudden inges-sometimes subnormal, the pulse may be in excess

tion of large amounts of carbohydrate material, usually

of 120/minute, the eyes are often sunken, the mucous

cereal-based concentrates, will result in recumbency,

membranes injected and, in acute coliform mastitis, although hypocalcaemia is often a feature of acidosis. they are frequently a purple colour. Abnormal milk Here the history will help and usually acidosis is accom- secretion will be found in one or more teats, although panied by diarrhoea.

in acute coliform mastitis this change in milk character may be less than obvious.

Hypothermia: Mild hypothermia (reduction of 0.5°C or 1°F) is a normal feature of milk fever. However, severe

Calving paralysis: The history of dystokia due to fetal hypothermia (reduction of $3\text{--}4^{\circ}\text{C}$ or $5\text{--}7^{\circ}\text{F}$) occurs in

oversize and recumbency since calving help in the dif- cows that have been recumbent all night in winter out ferential diagnosis, but many cases are not clear-cut. of doors. Although these cows have some other primary Intravenous calcium together with the response noted disease, e.g. milk fever or mastitis, treatment will be un- is probably the best aid to differential diagnosis. successful unless steps are taken to raise body temper- ature as rapidly as possible.

Other physical injury: This can occur at any time of lactation. In summary, there are many diseases and conditions

tation or the dry period and can include fractured limbs, that result in recumbency of the cow. However, careful pelvic damage and severe muscle damage. These can history taking and a thorough clinical examination will be the result of excess riding behaviour during oestrus, eliminate most differential diagnoses and if one is still

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not sure of the diagnosis, response to intravenous of the downer cow syndrome in recent years. Following calcium will always be a useful indicator.

intravenous therapy plasma calcium rises rapidly and falls to around 2 mmol/l (8 mg/100 ml) 5–6 hours after treatment. Following subcutaneous therapy, the plasma Treatment

calcium levels may take 3–4 hours to reach 2 mmol/l

The treatment for milk fever is the slow intravenous (8 mg/100 ml). Furthermore, in severe cases of milk infusion of 8–12 g of calcium as soon as possible after fever the peripheral blood circulation will be impaired,

the onset of clinical signs.

which will inhibit the absorption of any fluid material

A number of licensed preparations are available

administered subcutaneously. In practice one is often

for the treatment of milk fever and most are based on

called to attend cases of milk fever that have not

calcium borogluconate (CBG) at 20 per cent, 30 per

responded to subcutaneous treatment administered by

cent or 40 per cent strength (see Table 46.1). It has been

the farmer several hours before and the whole of the

shown that 400 ml of 30 per cent CBG is adequate to

treatment solution is still present at the injection site.

treat milk fever in average size cattle and will provide

As already stated approximately 85 per cent of cows

9 g of calcium. However, the most commonly used

will respond to one treatment. If response is not evident

product in the UK is 400 ml of 40 per cent CBG, which

by 5–6 hours, then the cow should be re-examined,

will provide 12 g of calcium. During cold weather the

the diagnosis reassessed and if necessary a further in-

CBG solution should be warmed to body temperature.

travenous infusion of 8–12 g of calcium administered.

Approximately 85 per cent of cases will respond to one

Some practitioners advocate 400 ml of CBG intra-

treatment. In many cases cows recumbent from milk

venously plus 400 ml subcutaneously to prevent

fever will rise within 10 minutes of treatment and others

relapses. Relapse of milk fever occurs in 25 per cent of

will get up 2–4 hours later.

cases treated and this figure is not affected by additional

Following the intravenous infusion, which itself

subcutaneous administration. Blood levels of calcium

should take five minutes, it is essential to sit the cow in

six hours after intravenous or subcutaneous infusions

a sternal recumbency position and turn her so that

are similar and by 12 hours after administration all the

she is lying on the side opposite to the one on which

calcium administered, whether by the iv or sc route, has

she was found. Many cases will eructate and defaecate

been eliminated from the body.

during the treatment. If the cow does not rise immedi-

Treatment of milk fever should also be accompanied

ately she should be turned to lie on the opposite side by removal of the calf and advice to the farmer not to milk the cow for 24 hours except to check for the presence of mastitis. Following intravenous treatment, milk the cow for 24 hours except to check for the presence of mastitis. As already stated, relapses occur in sternal recumbent position without the aid of support such as bales of straw. If the cow keeps returning to relapse will be reduced if milk is not drawn from the udder during this 24-hour period. The 8–12 g of calcium should be reassessed as it is unlikely to be milk fever. given is only a small proportion of the daily calcium requirement, so the treatment is only a holding operation until the normal adaptation process is in full operation. Cases of relapse usually occur at 18–24-hour complications resulting in the downer cow syndrome.

intervals and should be treated in the same way, i.e. by Farmers treating their own cases of milk fever commonly use subcutaneous injections of CBG. This is occasionally cows, particularly Jerseys, have been known to undoubtedly a factor that has increased the incidence relapse on up to seven occasions.

There is a tendency amongst farmers to give two bottles of 40 per cent CBG, which would amount to

Table 46.1

Licensed products available for the treatment of 24 g of calcium. Such a procedure is probably counter-milk fever in the UK.

productive, as there is some evidence to suggest that too high levels of calcium administration will slow up

Product

Pack size Available

Dose required for

the adaptation process and actually increase the

(ml)

calcium (g)

600 kg cow (ml)

number of cows that relapse.

Some proprietary CBG preparations also contain

CBG 20%

400

6

600–800

magnesium (1.0 g) and phosphorus (2.6 g) in addition to

CBG 30%

400

9

400

calcium. If hypomagnesaemia is a complicating factor

CBG 40%

400

12

400

Maxacala

100

4.17

200

of milk fever then the addition of the magnesium may be helpful. However, in cases of clinical hypomagnesaemia C-Vet Veterinary Products.

saemia more than 1.0 g of magnesium will be required.

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The presence of the phosphorus has no doubt been magnesium chloride to the drinking water is a practical added because of the finding that the blood levels of and effective way to supply magnesium to cows; 40 g phosphorus in cases of milk fever are also depressed. daily of magnesium chloride crystals can be added to However, clinical evidence would suggest that the drinking water if large enough troughs are available. addition of phosphorus has no effect on the percentage Trough size should be 20 l/cow. If trough size is limited, of cases that recover or relapse. In fact, it has been magnesium acetate can be added to the water supply shown that plasma phosphorus levels return to normal using a water proportioner plumbed into the water within a few hours after successful treatment with CBG supply pipe.

without the addition of phosphorus.

If the plasma levels of the dry cows are normal then Historically, the treatment of milk fever was by udder diets restricting calcium intake may be considered, par-insufflation. This has the effect of slowing down milk ticularly if the plasma calcium levels are at the high end production. Plasma calcium levels do rise following of the normal range. To be certain to prevent milk fever, udder insufflation and will reach 2.5 mmol/l (10.0 mg/dry-cow diets should contain less than 30 g/day of 100 ml) by 4–5 hours after treatment. However, the efficiency of CBG intravenously and the danger of mastitis 50 g/day of calcium will prevent most cases of milk following insufflation have now rendered this mode of fever. Autumn grass in the UK often contains 8–10 g/kg treatment almost extinct except that some clinicians DM calcium. Thus with daily intakes of 12–14 kg DM, have been known to use the technique in cows that 90–140 g/day of calcium will be available in the diet. It persistently relapse.

is impossible appreciably to reduce this level of intake without removing the cows from grass and substituting a diet based on maize silage, hay or straw. However,

Prevention

some farmers are prepared to do this to reduce the risk

Much milk fever can and should be prevented (see

of milk fever and incidence levels of 5 per cent or less

Table 46.2). If an outbreak occurs in any season where

are possible. It should be appreciated that if low

more than 10 per cent of cows are needing milk fever

calcium diets are advocated for dry-cow use, just before

treatment the first action the clinician should consider

parturition a diet containing more calcium should be

is to blood test a group of six or seven dry cows to

administered to ensure adequate calcium being avail-

measure the concentration of calcium and magnesium.

able over the risk period. This can be achieved by

If the magnesium levels are low, i.e. below 0.85 mmol/l

feeding cattle concentrate or feeds that are high in

(1.8 mg/100 ml), this should be seriously considered as

calcium, e.g. sugar beet pulp (see Table 46.3).

interfering with calcium absorption, in which case sup-

Other methods of milk fever prevention involve

plemental magnesium should be administered to all

dealing with individual cows. Maintenance of appetite

dry cows within three weeks of calving. Approximately

is essential and, in the past, appetite stimulation by the

10–12 g of magnesium administered daily to dry cows

use of anabolic steroids has been suggested, but the use

will be sufficient to produce normal plasma levels of

of these products is now illegal in Europe. Maintaining

magnesium and to allow normal calcium absorption.

adequate calcium intake by drenching cows daily with

However, this is often difficult to achieve because of the

150 g/day of calcium chloride on the day before calving

problem of giving supplemental feeds to grazing cows.

and for four days thereafter has had some success in

A supplement of 25 g daily of calcined magnesite mixed

with cereal (1 kg/cow) or silage will produce the desired

effect, but many farmers are reluctant to give supple-

Table 46.3

Approximate calcium, phosphorus and magnesium

mental feeds to dry cows. Recent experience in the UK
contents (g/kg DM) of some common feedstuffs.

has shown that the addition of magnesium acetate or

Ca

P

Mg

Barley

0.6

3.8

1.4

Table 46.2

Approximate requirements of 600 kg cows for

Wheat

0.5

3.5

0.6

dietary calcium, phosphorus and magnesium (g/day). Source:

Maize

0.2

2.7

1.0

ARC (1980), reproduced with permission of CABI International.

Sugar beet pulp

10.4

0.9

1.4

Brewers grains

5.0

6.0

1.8

Ca

P

Mg

Maize silage

3.7

3.2

3.0

Grass and grass silage

3.0–10.0

1.5–4.5

1.0–3.0

Maintenance (non-pregnant)

15

13

9

(range)

Maintenance + late pregnancy

28

22

12

Grass and grass silage

5.9

3.9

1.5

Lactation (g/kg milk)

1.65

1.55

0.74

(average)

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*preventing milk fever and might be considered in cows
tating or dry cows in the spring or autumn and is usually
with a known history of milk fever.*

not accompanied by clinical signs. It usually affects the

The use of vitamin D3 and its metabolites given by whole herd. Lactating cows will suffer a slight reduction in milk yield and may have a slight nervous disposition, injection has been advocated in preventing milk fever. The administration of 10 million iu of vitamin D3 given e.g. reluctance to enter the milking parlour. Dry cows eight to two days before calving will considerably will be more prone to milk fever at parturition. If the reduce the incidence of milk fever but the practical herd is affected with subclinical hypomagnesaemia problem remains of accurately predicting the time of some individuals will develop grass staggers, particularly if stressed. Milk tetany occurs in calves fed pre-expected. More recently, the analogue of the vitamin D3 dominantly milk, particularly calves suckling cows that metabolite 1 α -hydroxycholecalciferol (1 α (OH)D3) has are subclinically hypomagnesaemic (see p. 253). The been used in trials to prevent milk fever. Doses of incidence of hypomagnesaemia varies considerably

350 mg given at least 24 hours and not more than five from region to region. Some areas of the UK are particularly high in the incidence of the disease in both beef and dairy cows. Overall, the annual incidence in predicting the time of calving. Recently, workers in UK dairy herds is approaching 1 per cent.

Israel (Sachs, 1988) have suggested that a single dose of 700 mg of $1\alpha(\text{OH})\text{D}_3$ given eight to seven days before expected calving is more effective and that the accuracy of calving date prediction is less important.

death. In 1984 in the UK it was estimated that 0.8 per cent of the dairy cow population died from the disease diet is deficient in magnesium. Before recommending

(Whitaker et al. , 1984). This was following the introduction of the use of the magnesium status of the herd should be

duction of milk quotas in Europe and a dramatic
assessed by blood testing six or seven dry cows.
reduction in the use of concentrate feeds for dairy
It has been found that there is less milk fever when
cows, particularly during the summer months.
diets are acidic. This is the basis of the dietary cationic–
anionic balance (DCAB). It is the balance between

Aetiology

cations (Na^+ and K^+) and the anions (Cl^- and S^{2-})
present in the diet. Their levels affect the acid–base
As there are no readily available body stores for mag-
balance and thus calcium metabolism. Some diets in the
nesium, the main factors involved in the aetiology of
dry period are anionic and the reduction of the balance
hypomagnesaemia are the reduction of the amount of
in favour of a negative balance assists. This is achieved
magnesium available in the food, and hence available
by increasing the feeding of Cl^- and/or S^{2-} , by reducing
for absorption from the gut, accompanied by a high
the amount of Na^+ and/or K^+ present or by feeding
physiological demand for magnesium. Magnesium is

maize silage. The target is to achieve in the last two lost from the body in milk, urine and faeces. The weeks before calving -200 mEq/kg DM. This can be endogenous loss in faeces has been calculated to be done by using 200–250 g of anionic salts. Practically this approximately 1.8 g/day (ARC, 1980). Milk contains is often achieved by the addition of 100 g NH_4Cl and 0.12 g/l of magnesium so a cow producing 30 kg of milk 100 mg of MgCl_2 . However, such diets should only be would lose 3.6 g daily in the milk. Any excess magnesium used where there is adequate calcium provision. The sium absorbed will be excreted via the urine, this being system is complex and should only be used on farms the mechanism for stabilizing plasma magnesium with good quality management.

levels. If the plasma magnesium levels rise much above 0.8 mmol/l (2.0 mg/100 ml) the excess will be excreted.

If magnesium intake levels are excessive, up to 5.0 g/day

Hypomagnesaemia (grass tetany,

may be excreted in the urine. However, if magnesium

grass staggers, lactation tetany,

intake falls to the level to maintain homeostasis or

Hereford disease)

below there will no magnesium identifiable in the urine.

To maintain magnesium homeostasis the absorption

Hypomagnesaemia is a common feature of a group of

of magnesium must be continuous so a constant supply

syndromes dominated by hyperaesthesia, incoordina-

is necessary in the diet. Feeds vary considerably in both

tion, tetany and convulsions that can occur in all rumi-

the content and the availability of magnesium so the

nants of all ages. Grass tetany or grass staggers is the

choice of pasture is important. Clovers have higher

name given to the syndrome affecting lactating cattle

magnesium content than grasses and grasses themselves

(beef or dairy cows) when grazing grass in the spring or

vary. Fast-growing Italian ryegrasses have lower levels

autumn. Subclinical hypomagnesaemia can occur in lac-

than perennial grasses. Many broad-leaf plants, such as

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buttercups (*Ranunculus* spp.), plantains and nettles,

two to four-month-old calf is denied feed supplemen-

have considerably higher magnesium levels than
tation other than cow's milk, magnesium absorption
grasses, which probably accounts to some extent for
will decline to less than requirements and clinical signs
the increasing incidence of hypomagnesaemia seen in
will appear. This is seen in suckler calves or veal calves
recent years in the UK as old permanent pastures have
reared on milk or milk substitute that has not been sup-
been replaced with ryegrass leys.

plemented with magnesium (see p. 253).

Some soils are known to be deficient in magnesium
but also the uptake of magnesium by plants may be

Signs

influenced by cations such as calcium and potassium in
the soil. It has been shown that fertilizers containing

The signs of grass staggers may be classified as sub-
potassium applied to grazing areas in late winter or
acute, acute or peracute. In the subacute form cows will
early spring will reduce the absorption of magnesium
be apprehensive and hyperaesthetic. The head will be
by plants, and soils containing high levels of potassium

held high and tremors may be seen around the head are particularly prone to producing hypomagnesaemia (particularly the eyelids), over the shoulder and on the in grazing ruminants.

flank. These tremors will be exaggerated if the animal

Although historically this disease has been one

is touched or the skin pinched. The legs may become

related to spring grass, in recent years it has become

stiff and a staggering gait may be evident. Cows can

increasingly common throughout the whole grazing

remain in the subacute phase for several hours or

season from April to October. The occurrence during

progress to the acute or peracute form, particularly in

winter has also increased in the UK; this can be attrib-

response to noise or some other stimulus such as

uted to the increasing use of grass silage made from

attempting to herd them. The peracute cases will

young leafy grass low in magnesium during May.

stagger for a few steps and fall over with tetanic spasms

Another important factor in the aetiology of the

of the head, neck and legs followed by clonic convul-

disease is the energy intake of the animals. This is particularly the case in beef suckler cows grazing inadequate pastures, particularly during times of inclement and furious weather. Experiments have shown that reduced energy intake will interfere with the magnesium available for to noise from a vehicle or other stimulus, to stagger, fall absorption. The availability of magnesium for absorption from the gut is thought to range from 4 to 35 per cent. The signs in the acute case will be similar but may last for up to an hour or more. In these cases a period of convulsions will be followed by a quiescent period in magnesium.

which the cow may attempt to rise, only to walk a few It is possible to find animals with low plasma magne-

steps and fall over again followed by convulsions. The sium levels but yet clinically normal. It would appear rectal temperature, if taken, will be elevated by 1 or 2°C that the critical factor influencing the onset of clinical (2– 4°F). Cows are often found dead with obvious signs disease is the level of magnesium in the cerebrospinal that the limbs had been paddling prior to death, thus fluid (CSF). The speed at which magnesium levels fall disturbing the soil around the feet.

also influences the onset of clinical signs. In the spring, In the subclinical form the majority of the herd are magnesium levels fall rapidly and clinical disease may usually affected even if acute cases have not been diagnosed. become apparent at blood levels at which in the nosed. If acute cases are present it can be assumed that autumn, when the fall is more gradual, the cows remain the majority of the herd will be affected subclinically. clinically normal.

The cows may be slightly nervous, reluctant to enter the The role of calcium in the onset of clinical hypomag- milking parlour or be unwilling to be herded. The milk

nesaemia is also unclear. Approximately 80 per cent of yield will be depressed slightly.

cows with grass staggers have low plasma calcium

The signs of milk tetany in calves (p. 255) are much levels. It has been shown that cows will develop clinical the same as for cows with subacute, acute and peracute disease within 24 hours after a fall in plasma calcium cases occurring. Often in peracute cases the animals are levels although they have been hypomagnesaemic for found dead.

several days.

Milk tetany occurs in calves two to four months old

Clinical pathology

reared on whole milk diets without the addition of magnesium supplementation. In young calves absorption of Healthy normal cows should possess plasma magnesium is good. However, as the calf grows older sium levels over 0.85 mmol/l (2.0 mg/100 ml). Any levels the ability to absorb magnesium declines. Thus, if the below this must be considered a risk and indicative of

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subclinical hypomagnesaemia, although in acute cases cow group and testing for magnesium is the most useful the plasma levels will generally be below 0.4 mmol/l indicator. Testing urine for magnesium levels has been (1.0 mg/100 ml). Magnesium levels in the CSF in acute advocated, absence of magnesium indicating the sub-tetany will generally be below 0.6 mmol/l (1.4 mg/ clinical state. Urine test strips are available in some 100 ml). Hypocalcaemia is present in at least 80 per cent countries. However, there are real practical problems in of acute tetany cases and hyperkalaemia is common. getting a number of cows to micturate on demand.

Following tetany or in recovered cases aspartate amino-transferase (AST) and creatine kinase (CK) levels will

Differential diagnosis

rise to relatively high levels but return to normal quite soon after recovery.

Acute lead poisoning: In lead poisoning (see pp. 906,

At post mortem there are no pathognomonic signs.

944) there may be excitement and occasional convul-

Haemorrhages may be present on the heart muscle and

sions. Hyperaesthesia, as measured by observing muscle occasionally along the aorta. Regurgitation and aspiration tremors in response to pinching the skin, will be absent. Regurgitation of rumen contents may sometimes be seen. The absence of vision and blindness is usually a feature of lead poisoning. CSF levels of magnesium will be low and magnesium will be absent from the urine, although the bladder is empty. Milk fever (p. 783): Some cows in the early stage of milk fever are nearly always empty at post mortem.

Milk fever may exhibit hyperaesthetic signs. The history of being close to parturition is the most helpful aid to this differential diagnosis. Many cows are hypocal-

Diagnosis

caemic as well as hypomagnesaemic and so treatment is with calcium. The diagnosis of grass staggers is made on the signs described above. The time of year and type of grazing may help in forming a diagnosis. As there is little or no time in acute cases to conduct blood biochemistry,

time in acute cases to conduct blood biochemistry,

response to treatment will also confirm a diagnosis. It

Acetonaemia (p. 740): Some cows with acute acetonaemia may be useful for the clinician to take a blood sample. A cow with acetonaemia will have hyperaesthetic signs and appear nervous and apprehensive. However, the depraved actions of these cows, such as licking the walls and floor found dead then diagnosis must be differentiated from other causes of death. Diagnosis at post mortem is difficult because of the absence of obvious lesions. If a sample of milk.

traces of soil have been gouged out of the ground by each of the four feet during the paddling phase this is

Listeriosis (p. 904): Acute listeriosis may be confused with acute grass staggers. Absence of hyperaesthesia with acute grass staggers, but the high rectal temperature and absence of true hyperaesthesia in listeriosis

the bladder is generally empty. Blood and tissues are of cases should be enough to distinguish this disease.

no value at post mortem because magnesium levels rise rapidly after death. Levels in CSF may be helpful and

Bovine spongiform encephalopathy (p. 909): The emer- recently it has been suggested that magnesium levels in

gence of BSE in the UK in 1986 has sometimes made

aqueous humour taken after death will be depressed in

the differential diagnosis of the subacute form of hypo-

cows that have died from grass staggers. If a cow is

magnesaemia more difficult. The ataxia, apprehension

found dead and grass staggers suspected from the

and mild hyperaesthesia found in BSE are similar to

history and absence of other lesions at post mortem,

that found in subacute hypomagnesaemia. Diagnosis is

the wise clinician will blood sample six to seven cows in

usually confirmed by response to treatment and blood

the group and test for magnesium levels. It is important

biochemistry, a sample being taken before treatment. If

to make a diagnosis to be able to offer preventive

there is any doubt in the clinician's mind it would be

advice for the remainder of the herd.

wise, in cases of suspected BSE, to take a blood sample

Diagnosis of death due to milk tetany in calves is possible by measuring the calcium and magnesium levels in bone. A rib or coccygeal bone is usually used for this purpose. A calcium/magnesium ratio in bone of 70 : 1 is

Others: Lightning strike (p. 930) as a cause of sudden death, some plant poisonings, and in particular of severe magnesium depletion.

Paspalum staggers (Claviceps paspali poisoning), are To diagnose subclinical hypomagnesaemia blood differential diagnoses but can usually be distinguished sampling seven cows each from a lactating and a dry-

on grounds of history alone. Rabies (p. 908; Chapter 70)

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may cause problems in countries where it occurs, but raised into sternal recumbency and left for a further 30 such animals usually are hyperactive, bellowing and

minutes before attempting to stimulate the animal to riding other cattle.

rise.

Treatment is successful, if early, in 80 per cent of acute

Suboptimal production: If production is below expectant cases and nearly 100 per cent in acute stand-tations, particularly in seasons associated with hypomagnesaemia. If the recumbent case is not able to rise within

then subclinical hypomagnesaemia

two hours of treatment the likelihood of success is could be present. Blood biochemistry in the form of a extremely poor and slaughter should be considered.

metabolic profile that includes magnesium should be

Subacute grass staggers should be treated in the same considered (see p. 813).

way and success is usually near to 100 per cent.

Relapses are considered normal unless preventative measures are taken. Blood magnesium falls to pre-

Treatment

treatment levels within six hours of intravenous

Acute cases of grass staggers must be treated promptly. administration, although the subcutaneous injection has During the course of treatment the operators should be a more prolonged effect. Daily subcutaneous injections as quiet and gentle as possible as any sudden stimulus of 200–400 ml of 25 per cent magnesium sulphate for will initiate a bout of convulsions. Hence, actually five days following treatment have been recommended restraining a staggering acute case will often be difficult or the oral administration of four magnesium bullets, and when attempting to place a rope or halter on which are composed of an alloy of 86 per cent magne- the animal it will sometimes collapse into a fit of sium, 12 per cent aluminium and 2 per cent copper convulsions and may even die before treatment has weighted with iron shot (Rumbul bullets, Agrimin Ltd), been administered. The success of treatment is immediately following the intravenous and subcuta- also related to the length of time signs have been neous treatment. present in the recumbent acute case. The longer the cow

An individual cow suffering from grass staggers is has been showing convulsions, the less likely it will be one of a herd and many cows in the herd could be at to recover.

risk, of or at least suffering from, subclinical hypomag-

Intravenous infusions of magnesium salts, e.g. mag-

nesaemia. It is important therefore that a group of six

nesium sulphate, are sometimes recommended for

to seven cows be blood tested to ascertain the herd

treatment but this procedure is not without its dangers.

magnesium status, and if the blood levels are below

Intravenous magnesium sulphate may cause cardiac

0.8 mmol/l (2.0 mg/100 ml) herd supplementation with

embarrassment or even respiratory failure. If magne-

magnesium must be instituted.

sium sulphate is administered intravenously the con-

The treatment of milk tetany in calves is generally

centration should be no more than 6 per cent and

academic, as most cases are found dead. However, very

must be administered very slowly with the heart being

slow intravenous infusion of 100 ml of 20 per cent CBG

auscultated. However, it is also essential to administer with the addition of 20 ml of 25 per cent magnesium calcium in the form of CBG because most cases of grass sulphate followed by 60 ml of 25 per cent magnesium staggers are also hypocalcaemic.

sulphate subcutaneously would be the regimen of choice. The treatment protocol favoured by the author is to discard 100 ml of fluid from a 400 ml bottle of 40 per cent CBG, which also contains magnesium 0.2 per cent magnesium oxide would be 1, 2 and 3 g for calves up to five weeks, five to ten weeks and ten to fifteen weeks 100 ml of 25 per cent magnesium sulphate solution. The old, respectively.

mixture, which then contains 9 g of calcium and approximately 6 g of magnesium, is infused intravenously very

Prevention

slowly, taking 8–10 minutes. The remaining 300 ml of the original 400 ml 25 per cent magnesium sulphate is

The simplest method of prevention of hypomagnesaemia is to add calcined magnesite (magnesium oxide) injected subcutaneously. If during the infusion, or saemia is to add calcined magnesite (magnesium oxide) immediately after, the convulsions get worse 10 ml of to cattle concentrate that is being fed to the cows.

pentobarbitone sodium 200 mg/ml (euthanasia solu-

However, the main risk periods in northern Europe for tion) can be administered intravenously and this will

hypomagnesaemia are times when concentrate food is often reduce or even eliminate the convulsions. Fol-

not being fed, e.g. spring grazing and autumn grazing

lowing this treatment regimen, if the cow is recumbent,

for dry cows. In Australia, New Zealand and Ireland

it is important to remain quiet for a further 10 minutes

very little concentrates are fed, and in many beef

as stimulation even immediately after treatment may

suckler herds other ingenious methods must be devised.

initiate convulsions. The recumbent cow should then be

The magnesium bullets mentioned above will give

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protection for four to six weeks. However, although two

growing in the pasture may not only be aesthetically
bullets are recommended, in severe hypomagnesaemic
acceptable but may reduce the incidence of hypomag-
areas experience has shown that four bullets are
nesaemia in grazing animals.

required to prevent problems.

The prevention of milk tetany in calves can be

Dusting the pastures with magnesium oxide has been
achieved by ensuring magnesium supplementation.

attempted using 50 g/cow per day of magnesium oxide,

Proprietary milk substitute powders are adequately

or 0.5 kg/week applied in the early morning when the

supplied with magnesium. If whole milk is used mag-

dew is on the grass. This works best when cows are strip

nesium oxide at the rates given above should be added

grazed and the magnesium oxide is applied every

to the daily diet (see p. 790).

morning. Weekly applications are effective in dry

weather, but must be reapplied following rain.

However, grass staggers is more common in wet than

Hypophosphataemia

dry weather.

The increasing practice in recent years in the UK of Hypophosphataemia is the result of a primary deficiency of phosphorus in the diet (see also p. 792).

during the grazing season, has meant that magnesium oxide at the rate of 50 g/cow per day can be mixed with

Occurrence

or sprinkled on top of the silage and increases feed passage time.

Dietary deficiencies of phosphorus are widespread
Supplementation of water supplies using magnesium
under natural conditions. There is a distinct geographical distribution where large land masses are identified
expensive. The proportioner has to be adjusted daily
as being deficient in phosphorus and livestock cannot
to allow for the variation in water intake that occurs
be supported without phosphorus supplementation.
under different weather conditions. The expense and
These areas will be deficient because of the underlying

management input required have been a disincentive rock formation. Large areas of southern Africa and for this method of prevention to become widespread. Australia are well identified as being deficient in phosphorus. The addition of magnesium chloride crystals to phosphorus. Such areas, however, are unknown or rare in the drinking water has increased in popularity in the northern Europe and, if they exist at all, will be local-UK in recent years. It is relatively inexpensive but, to be effective, the trough sizes must be large enough underlying rock formation contains no phosphorus, for all cows to drink the medicated water. The addition continuous application of fertilizers containing phosphorus of 40 g/cow per day of magnesium chloride to the phosphorus and cultivation techniques have improved the drinking water has been shown to give reasonable soil structure and nutritive value. In consequence, protection. Addition of magnesium salts to the drinking water primary phosphorus deficiency is probably a rare occurring water will depress water intake if the concentration

rence in Europe.

is too high. Troughs with a volume of 20 l/cow are required for this method of control to work effectively.

Aetiology

Mineral licks and powders containing high levels of magnesium are relatively useless in preventing hypophosphorus deficiency is usually primary, although a magnesaemia because of the uncertainty of all cows severe deficiency of vitamin D (p. 253) may exacerbate consuming enough material to give them protection the problem. It was once considered that excess calcium when it is required.

would also reduce the availability of phosphorus in the When discussing the long-term strategies for con- diet and cause deficiencies of phosphorus. Although the trolling hypomagnesaemia, fertilizer policy should be calcium : phosphorus ratio in the diet is important in included. All potassium-containing fertilizers should monogastric animals, it is probably of less significance be avoided on soils with high potassium levels and on in cattle. The maintenance requirements for adult and

other soils, if potassium-containing fertilizers are used, growing cattle would be approximately 15 g of phosphorus daily. The lactating cow requires approximately 0.75 g/kg of milk produced, so a cow yielding 30 kg of milk will require 40 g of phosphorus daily. This level of

Perhaps plant species should also be considered in requirement is considerably less than that currently long-term control strategies. Clover/grass mixtures being recommended in the UK by the Agricultural Development and Advisory Service, but nevertheless is above all the use of selective weedkillers should be supported by experimental evidence from the Agricultural Research Council (ARC, 1980).

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Signs

Control

In young cattle phosphorus deficiency results in slow growth and rickets. In adult cattle the principal signs are reduced milk yield, weight loss and depraved appetite. Supplementation of the diet with phosphorus is often impractical. Bone meal, dicalcium phosphate or disodium phosphate may be provided in deficiency of phosphorus. The depraved appetite or pica results in cows eating earth, licking rocks and, where bones are available, osteophagia. The osteophagia frequently results in a high incidence of botulism (p. 721) Top dressing pastures with superphosphate fertilizers will correct any underlying deficiency and have the added advantage of increasing the yield and protein content of the pasture. However, this is often impractical of phosphorus deficiency. Reduced fertility has been

cal in areas where the problem exists.

considered a feature of severe phosphorus deficiency,

In acute cases where phosphorus therapy is urgent,

but experimental work has demonstrated that fertil-

e.g. postparturient haemoglobinuria, the intravenous

ity is independent of either calcium or phosphorus

administration of sodium acid phosphate (30 g in 200 ml

intakes. Reduced fertility is a feature of malnutrition

distilled water) is advocated (see below).

and frequently animals that are deficient in phospho-

rus are also suffering from malnutrition. Low energy

Postparturient haemoglobinuria

intake in such animals is much more likely to be the

cause of reduced fertility than any specific mineral

One specific syndrome associated with phosphorus

deficiency.

deficiency is postparturient haemoglobinuria. This is

Cows in late pregnancy often become recumbent,

a disease of cows one to four weeks after calving.

particularly in drought seasons. This recumbency is

Haemolytic anaemia and hypophosphataemia are con-

*probably a result of general malnutrition rather than a
sistent features.*

specific phosphorus deficiency.

*Hypophosphataemia nearly always accompanies
hypocalcaemia in cows with milk fever, but this is not*

Occurrence

thought to be of any significance. Phosphorus levels

*Postparturient haemoglobinuria was first described in
rarely fall to the levels of 0.3 mmol/l (1 mg/100 ml) seen*

Scotland in 1853 and has been reported from many

in severe clinical cases and in any case calcium therapy

countries including Australia, USA and most of

will result in the blood phosphorus levels quickly

Europe. The occurrence is sporadic and when it does

reverting to normal.

arise it usually only affects one or two cows within a

herd. In recent years its occurrence in the UK has been

extremely rare. In Scotland it has been associated with

Diagnosis

the feeding of beets and turnips, in Holland with the

The presence of rickets or osteomalacia and/or pica will

feeding of lush spring grass and occasionally it has been indicate a dietary deficiency of phosphorus, which can reported to accompany the feeding of sugar beet be confirmed by analysis of serum and the diet for the byproducts and alfalfa.

presence of phosphorus. The normal blood level is 1.3–1.6 mmol/l (4–5 mg/100 ml). Levels of 0.5–1.1 mmol/l

Aetiology

(1.5–3.5 mg/100 ml), falling as low as 0.3 mmol/l (1 mg/100 ml) of serum in severe clinical cases, will be found

Diets low in phosphorus are incriminated in the cause in hypophosphataemic animals.

of postparturient haemoglobinuria. However, it is

Most pastures in northern Europe contain 1.5–

probable that there is some additional factor that pre-

4.5 g/kg DM of phosphorus and at these levels phospho-

cipitates the problem in hypophosphataemic cattle. This

rus deficiency will not occur. Osteophagia will occur with

is likely to be a haemolytic factor present in sugar beet

pasture levels of 0.2 g/kg DM of phosphorus and rickets

leaves, alfalfa, kale and other Brassica spp. plants.

and osteomalacia at pasture levels of 0.1 g/kg DM.

The only commonly used feedstuffs in the UK that

Signs

are deficient in phosphorus are sugar beet pulp at

0.9 g/kg DM, kale and other Brassica spp. crops. These The principal clinical signs are those associated with

are rarely used at a proportion of the diet high enough

anaemia. The cow may be weak and staggering, with

to significantly reduce the phosphorus intake to dan-

mucous membranes pale and heart rate raised. Haemo-

gerous levels.

globinuria will be a consistent feature. The faeces are

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firm and dry. If left untreated the cow will finally

ally, nervous signs. Ketone bodies, e.g. acetoacetate, b-

become recumbent and may die within two to five days.

hydroxybutyrate or acetone, are present in all body

Less severely affected cases may recover, albeit slowly,

fluids. Hypoglycaemia together with increased plasma

in three or four weeks. Pica is frequently observed

free fatty acids and liver fat and decreased liver glyco-

during the recovery period.

gen are also a feature of this disease. These changes are associated with an inadequate supply of the energy that is necessary to sustain high levels of milk production in

Clinical pathology

early lactation. Pregnancy toxaemia, a common disease

Serum phosphorus levels are low, usually 0.15–

of pregnant sheep and characterized by hypoglycaemia

1.0 mmol/l (0.5–3.0 mg/100 ml). Low phosphorus levels

and hyperketonaemia, can also occasionally affect

will be encountered in other cows in the herd. Red cell

pregnant cows particularly when carrying twins (see p.

counts, packed cell volume and haemoglobin are all

796).

dramatically reduced. Serum bilirubin will increase in

Historically clinical acetonaemia was more common

the later stages of the disease.

in the winter when cows were housed and being fed con-

At post mortem the liver will be swollen and infil-

served forage of dubious quality. The annual incidence

trated with fat. The carcass is jaundiced and anaemic

in dairy herds is around 0.5 per cent and recent reports and red or red/brown coloured urine will be found in have indicated an all year round occurrence with a the bladder.

higher incidence in June, July and August than in the winter months. Cows of any age can be affected and Channel Island breeds, particularly the Jersey, appear to

Diagnosis

be more susceptible than the Friesian or Holstein. Ac- Postparturient haemoglobinuria must be suspected in tonaemia is less common in the UK now than it was 20 any cow that is weak, anaemic and exhibiting haemo- to 30 years ago. This is probably due to higher quality globinuria within four weeks of parturition. The haemo- feeds being available although the preponderance of the globinuria must be distinguished from babesiosis (p. Friesian breed may have some effect on the disease inci- 748) and copper poisoning (p. 948). A history of feeding dence. Outbreaks are usually restricted to one or two large quantities of kale, beet tops or alfalfa will also be cows but varying numbers of cows in the herd may be

a helpful aid to diagnosis. The diagnosis should be confirmed by demonstrating hypophosphataemia and become a severe economic problem due to depressed haemoglobinuria.

milk production. The disease usually occurs three to six weeks after calving, when the cow is at her peak milk production but her appetite or DM intake has not yet

Treatment

reached its peak. During early lactation the dairy cow is in negative energy balance. The energy intake in feed is insufficient to meet the energy output in milk. This calcium phosphate in the diet is the first line of treatment. In extremely anaemic cases blood transfusion results in the mobilization of fat reserves to meet the energy deficit and a consequent loss in body weight. This with 5–10 l of blood should be considered. Supportive therapy with large doses of vitamin C intravenously and

high-yielding dairy cows. Such cows will have slightly iron dextran injections will aid the recovery.

raised blood ketone levels and may even excrete ketones in urine and possibly in milk. The cow in early lactation is therefore in a delicate metabolic balance and any

Prevention

stress that causes a reduction of feed intake can disturb

If brassicas have to be fed to cattle in early lactation

this balance and result in the onset of clinical ketosis.

their intake should be limited to a maximum of 20 kg

Factors that can influence the occurrence of the disease

wet matter/day. This limitation and an adequate phos-

include excessive feeding of silage that has a high

phorus intake will prevent the occurrence of postpar-

content of butyric acid, a deterioration in forage quality,

turient haemoglobinuria.

sudden changes in types of food on offer and excessive

fatness at calving. In the UK the butyric acid content of

grass silage is of considerable importance in the aetiol-

Acetonaemia (ketosis, slow fever)

ogy of this disease because wet conditions, so frequently

encountered during silage making, predispose to butyric Acetonaemia or ketosis is a metabolic disorder of high-fermentation of the silage. The role of such silage in the yielding lactating cows characterized by reduced milk aetiology of acetonaemia is probably two-fold. Firstly, yield, loss of body weight, inappetance and, occasion- there is the direct effect of the presence of butyric acid

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and secondly there is the reduced dietary intake that acetyl-CoA is converted into the ketone bodies acetoaccompanies such silage. Cows that are too fat at calving toacetate and β -hydroxybutyrate and, to a small extent, have lower DM intakes in early lactation and are there- acetone. Tissues other than liver can utilize ketone fore more likely to suffer acetonaemia.

bodies but, if their production exceeds the rate they are Acetonaemia is diagnosed in grazing cattle if the used by muscle and other tissues, they accumulate and grass has a high moisture content and/or when the ketosis is the result. Ketone bodies are excreted in milk energy intake is insufficient. Cobalt is required for

and urine.

rumen microbial synthesis of vitamin B12 and is also

The reduction of propionic acid production by the essential for adequate utilization of propionic acid. In rumen is usually a feature of underfeeding or a reduced areas of cobalt deficiency acetonaemia will be com-feed intake caused by inappetance. Cobalt deficiency, as monly diagnosed in grazing cows (p. 295).

mentioned above, will also have the effect of reducing

Secondary ketosis is common, if not more common, propionic acid production. Butyrate is a precursor of than primary ketosis and can result from any disease acetyl-CoA and is therefore ketogenic. An increase in that causes a reduction in appetite in early lactation.

butyrate uptake from the rumen will therefore be keto- Displaced abomasum (see p. 839) and traumatic reticulitis. This explains why silage high in butyric acid will ulitis (p. 837) are two common problems frequently induce ketosis in apparently normal cows.

associated with secondary ketosis.

Signs

Aetiology

Hypoglycaemia is the major factor involved in the onset

*To understand the aetiology of acetonaemia one must
and development of the clinical signs of acetonaemia.*

realize the precarious metabolic balance that exists in

There will have been a gradual loss of body condition

all cows in early lactation. To satisfy the requirements

over several days or even weeks. There is also a mod-

of milk production the cow can draw on two sources of

erate decline in milk yield over two to four days before

nutrients, food intake and her body reserves. In the first

the onset of the obvious clinical signs, which are refusal

two months of lactation a cow producing up to 45 kg of

to eat grain and concentrate feeds and a more sudden

milk daily will use up to 2 kg of body fat and up to 350

drop in milk output. At this stage a sweet smell (as in

g of body protein per day. As far as the dietary supply

pear drops) of acetone is apparent on the breath and

of nutrients is concerned 80 per cent of the ingested car-

the discerning stockworker will even detect the same

bohydrates are fermented by the rumen microflora into

acetone smell in the milk. Once appetite is decreased the volatile fatty acids, acetic, propionic and butyric weight loss is accelerated due to utilization of body acids, which are themselves absorbed. Acetate may be stores. Rectal temperature, pulse rates and respiratory oxidized by various tissues or incorporated into milk fat rates are normal in the early stages of the disease, as are by the mammary gland.

ruminal movements. Faeces will usually be firm with a Glucose is synthesized in the liver and renal cortex dark 'waxy' appearance.

by the gluconeogenic pathway. Approximately half of A small number of cows with acute acetonaemia the cow's glucose requirement is derived from dietary exhibit nervous signs, which include excessive saliva-propionic acid, which is incorporated into the tricar-tion, abnormal chewing movements and licking walls, boxylic acid (TCA) cycle and converted to glucose by gates or metal bars. Incoordination with apparent gluconeogenesis. Glucogenic amino acids, lactic acid blindness will also be a feature. Some cows will even

and glycerol can be converted into glucose by this show a degree of aggression and will sometimes charge process. Reduced production of propionic acid in the into walls, occasionally injuring themselves. The other rumen will result in inadequate glucose production and signs observed above are also present. The nervous a consequent hypoglycaemia. Hypoglycaemia leads signs often only last for a few hours with the animals to a mobilization of free fatty acids and glycerol from showing more normal behaviour in between.

the fat stores. Hormones such as adrenaline, glucagon, adrenocorticotrophic hormone, glucocorticoids and

Clinical pathology

thyroid hormones all influence this mobilization from the body fat stores. Skeletal muscle and heart can utilize Hypoglycaemia, hyperketonaemia and the presence of fatty acids for energy production when glucose is short.

ketones in the urine and milk are the features of this However, the liver has a limited ability to oxidize fatty disease. Cowside diagnosis is obtained by the detection acids because acetyl-CoA, which is the end product of

of ketones in milk and urine using the Rothera's test
fatty acid oxidation, cannot be adequately incorporated
reaction. A drop of milk or urine is added to a small
into the TCA cycle when levels of oxaloacetate, the
quantity (which consists of sodium nitroprusside 3 g,
result of active gluconeogenesis, are low. The excess
sodium carbonate 3 g and ammonium sulphate 100 g) of

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Rothera's reagent on a white tile or piece of white card.

Treatment

A pink to purple coloration of the reagent confirms the
There are three main components of successful
presence of ketones. Urine normally contains low levels
treatment:

of ketones so a diagnosis is only positive when the milk
is also positive.

(1)

To restore blood glucose levels as quickly as

Blood glucose levels are reduced to below 1.4 mmol/l
possible.

(25 mg/100 ml). Total blood ketone levels are raised

(2)

To replenish oxaloacetate, an essential intermediate to over 5 mmol/l (30 mg/100 ml). The plasma glycerolate in the TCA cycle in the liver, so that fatty acids and free fatty acid levels (non-esterified fatty acid, mobilized from the fat deposits are completely NEFA) are also elevated. Subclinical ketosis has oxidized. This will reduce the rate of production become more important in recent years with the introduction of ketone bodies.

duction of the laboratory test for b-hydroxybutyrate

(3)

To increase the availability of dietary glucogenic (bHB). The level of bHB is frequently used on a herd precursors, notably propionic acid.

basis as a measure of energy balance in both lactating and dry cows. Herds with subclinical ketosis have been

An intravenous infusion of 500 ml of 40 per cent identified using this test. Serum levels of bHB in excess glucose will cause a transient rise in blood glucose levels of 1.75 mmol/l (10 mg/100 ml) will indicate a severe

that lasts approximately two hours. This should be energy deficit in the diet (see p. 807).

accompanied by oral administration of glucose precur-

Although mortality is not normally a feature of acetonaemia, affected cows do possess fatty infiltration and sors such as propylene glycol (150 ml, twice daily).

Propylene glycol is preferred to propionate or glycerol degeneration of the liver.

because propionate is fermented in the rumen and may cause digestive disturbances and glycerol is converted to ketogenic acids as well as propionic acid in the

Diagnosis

rumen. Cobalt salts are frequently added to the pro-

The diagnosis is made on the history of a cow in early pylene glycol and in cobalt-deficient areas at least lactation with a sudden fall in milk yield, some weight loss, refusing to eat concentrates, with normal temperature, pulse and respiratory rates and normal rumen therapy for acetonaemia, either used alone or in com-

100 mg/day of cobalt should be administered.

Glucocorticoid drugs are the most commonly used

movements. Many astute stockworkers will recognize
bination with glucose therapy or when followed by oral
the acetone odour on the breath or in the milk and
administration of glucose precursors. Glucocorticoid
report this to the attending veterinarian. The diagnosis
therapy results in a reduction of ketone body formation
is confirmed by a positive Rothera's reaction on milk
due to utilization of the acetyl-CoA derived from fatty
and urine and, if this is not conclusive, a blood sample
acid oxidation and raises blood glucose levels due to a
can be analysed for glucose and ketone levels. It is
greater availability of glucose precursors in the liver.
important to differentiate between primary and sec-
The commonly used glucocorticoids are dexametha-
ondary ketosis so a complete clinical examination must
sone, betamethasone and flumethasone and all are
be performed. Many cases presented by the farmer as
effective. Frequently, a single dose is administered but
acetonaemia are in fact suffering from displaced abo-
this does often result in relapses two to three days after
masum (p. 839). Some cows with hypocalcaemia (p.

the treatment, when the injection can be repeated.

783) may also show acetonaemia.

There is one disadvantage of repeated glucocorticoid

The differential diagnosis of the nervous form of ace-

therapy and that is that appetite and milk yield are

*tonaemia can be sometimes confusing. The behavioural
reduced.*

changes are similar to listeriosis (p. 904), but usually

For successful treatment in most cases of aceton-

with listeriosis pyrexia will be present. Hypomagne-

aemia the following regimen is to be recommended:

saemia (p. 788) should be distinguishable by the pres-

• 500ml of 40 per cent glucose intravenously, fol-

*ence of hyperaesthesia, particularly the tremors of the
lowed by*

eye-lids and muscle tremors over the shoulders and the

• One dose of glucocorticoid, followed by

presence of tetanic convulsions. Bovine spongiform

• Oral treatment twice daily with 150g of propylene

*encephalopathy (p. 909) may also be confused with ace-
glycol containing cobalt for three to four days.*

tonaemia because of weight loss. However, the apprehension, kicking and progressive nature of BSE should be distinguishing features, besides which blood glucose, tonaemia and were used in Europe before their use was magnesium and ketone levels will be normal in BSE. Anabolic steroids are also a useful treatment for aceto- prohibited under the EC hormone ban. They are effective by increasing the levels of the intermediates of the Rabies (p. 908; Chapter 70) is characterized by mania, ascending paralysis and is always fatal.

TCA cycle in the liver. They also stimulate appetite,

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which ensures an increased supply of the glucogenic cattle in late pregnancy. The problem is best described precursors. They do not directly raise blood glucose as starvation, but the aetiology and pathogenesis are levels. It is important that the cow's appetite returns to similar to acetonaemia in that an energy deficit in normal as soon as possible after treatment so access to the diet leads to massive mobilization of fat reserves, good quality fodder is a prerequisite to successful treat-

resulting in hypoglycaemia and hyperketonaemia. The problem is most common in beef cattle grazing marginal land, but has been seen in dairy cattle in late winter in seasons where there has been a shortage of well-fermented silage or good quality hay offered. If acetonaemia is affecting a high proportion of the conserved forage. Cows of all ages are affected, but herd it would be wise to obtain a supply of ground maize, as it has been shown that ground maize is readily digested in the small intestine and results in a rapid rise in blood glucose levels. Beef cows often have access to good pasture in the summer months and can get overfat. If the same cows do not have access to good quality forage during the winter months, when they are in late pregnancy they will succumb to ketosis because of the deficit in energy intake. In dairy cows the problem can occur

Prevention

nancy they will succumb to ketosis because of the deficit in energy intake. In dairy cows the problem can occur

The prevention of acetoanaemia starts before calving. at or around calving and is again the result of insufficient energy intake in excessively fat animals. Cows should not be too fat at calving, a condition score of 2.5–3.0 would be optimum and anything higher would be considered too fat. Access to a plentiful supply of long coarse fibre to promote good rumen

Signs

digestion is also important during the dry period. Concentrates used during lactation should be introduced in small quantities (1–2 kg/day) two weeks before calving onset are associated with the stage of pregnancy and the to allow adjustments in the rumen microflora. Changes degree of nutritional stress. Affected cows are usually to diet in early lactation should be made gradually. seven to nine months pregnant and show the same clinical signs as cows with acetoanaemia. They become butyric acid should be avoided in early lactation. increasingly dull and depressed and the smell of

two to five days after sternal recumbency. Cows
ing blood glucose and BHB levels in groups of dry cows
affected close to parturition often die during
and cows in early lactation can be useful in the hands
parturition.

of the experienced veterinarian. This will often indicate
an energy-deficient diet and one that could predispose
to subclinical if not clinical acetonaemia.

Clinical pathology

As already stated, acetonaemia is less common now
Hypoglycaemia, hyperketonaemia and ketonuria are
than in previous years. This is due mainly to improve-
consistent findings. In recumbent cases the blood levels
ments in forage conservation techniques and the use of
of bHB are much higher than in acetonaemia; levels
mixed forages, especially maize silage. Thus cows are
up to 22 mmol/l (125 mg/100 ml) may be found. Cows
fed better quality feeds and there is increased aware-
affected close to parturition have hypocalcaemia and
ness that optimum output comes as a result of optimum
occasionally hypomagnesaemia. Recumbent cows in

input.

the terminal stages have hyperphosphataemia (up to 6.5 mmol/l; 20 mg/100 ml), hyperglycaemia (up to

Pregnancy toxaemia

9.0 mmol/l; 160 mg/100 ml) and raised AST levels. At

post mortem the most consistent findings are an

Pregnancy toxaemia, although primarily considered a

enlarged, yellow, fatty liver with fatty changes in the

disease of sheep, does also affect cattle, particularly beef

kidney and adrenal cortex.

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Diagnosis

(1)

How long has the cow been recumbent?

(2)

When did she calve and was calving difficult?

Often several cows are affected before veterinary atten-

(3)

Did she rise after calving?

tion is sought. The usual stimulus to seek veterinary

(4)

*Has she been treated for milk fever, and if so how
help is when one or two cows are dead or close to death.
often and how much CBG has been used?*

*The history, stage of pregnancy and the nutritional
(5)*

*Has the cow moved recently, either spontaneously
status will usually be enough to enable a tentative diag-
or with help from the farmer?*

*nosis. Raised blood or urine ketone levels and low
(6)*

*Where did the cow go down, e.g. concrete, ice, in
blood glucose (plus low calcium in cows close to
field, in a ditch, and was this likely to affect the
calving) will usually confirm the diagnosis.
pathogenesis?*

(7)

Is there adequate bedding?

Treatment

Examination

*Treatment as described under acetonaemia (see p. 795)
would normally be indicated. However, so severely*

The first superficial examination will be the position of affected are the majority of these cows that medical the animal, position of the legs and degree of alertness. treatments almost invariably fail to succeed. Immediate Lateral recumbency, if not due to hypocalcaemia, hypomagnesaemia or bloat, is indicative of a terminal state save a valuable cow. This should be followed by the full and slaughter should be advised as soon as possible. course of treatment described under acetonaemia.

This includes cows which, although they will sit in the sternal recumbency position for short periods with the aid of supports such as hay bales, revert to the lateral

Prevention

recumbent position when they struggle free of the

Although the problem is more common in fat cows it is supports.

essentially the result of starvation and is predominant

The position of the legs is a useful aid to prognosis.

in years when insufficient conserved fodder has been

If the hindlimbs are in the normal position and the cow

made. To prevent further cases developing and becoming attempts to rise the prognosis is guarded to hopeful. If the hindlimbs are rigidly extended forwards so the feet are touching the elbows of the front legs this indicates a severe sciatic nerve damage, upper hindlimb muscle degeneration or hip problems, e.g. fracture or dislocation, and the prognosis is hopeless. If both the hindlimbs are spread laterally, with lateral flexion at the stifle, the

The downer cow

cow has probably 'done the splits' and again the prognosis is hopeless. If one hindlimb is in this position then attending the recumbent cow is one of the more challenging problems encountered by the bovine practitioner (see also p. 439). Often, accurate diagnosis is not possible but prognosis is extremely important and prob-

to the original position when the cow attempts to move, ably more important than accurate diagnosis. It must be again the prognosis is hopeless as this indicates severe remembered that recumbency is the normal course in muscle degeneration.

the terminal stages of any disease so a full clinical exam-

The full clinical examination will then be conducted ination is essential in every case plus a thorough history, (see Fig. 46.2) and may well reveal other diseases

often supported by biochemical examination of blood.

present, e.g. mastitis, metritis (vaginal examination is

Many workers have offered definitions of the downer essential), torn vagina, ruptured uterus, pneumonia,

cow, but the most useful in practice and the one to be septicaemia, hypothermia or abdominal catastrophe. It

used here is: a cow that has been recumbent for 24

is essential that a rectal examination be performed.

hours or more, is in sternal recumbency and is not suf-

Assuming there is no abnormality in rectal tempera-

fering from hypocalcaemia or hypomagnesaemia, mature, respiratory rate, mucous membranes and the heart

titis or any obvious injury to the limbs or spine.

rate is below 80/minute, then ischaemic necrosis of the hindlimb muscles or, as it is more commonly referred to, pressure syndrome should be considered.

History

It is important that the clinician obtains an accurate

Pressure syndrome (ischaemic necrosis): Experimental history of the case and the following questions must be work (Cox, 1982) has demonstrated that if a cow is lying asked:

in the same position for six hours or more there will be

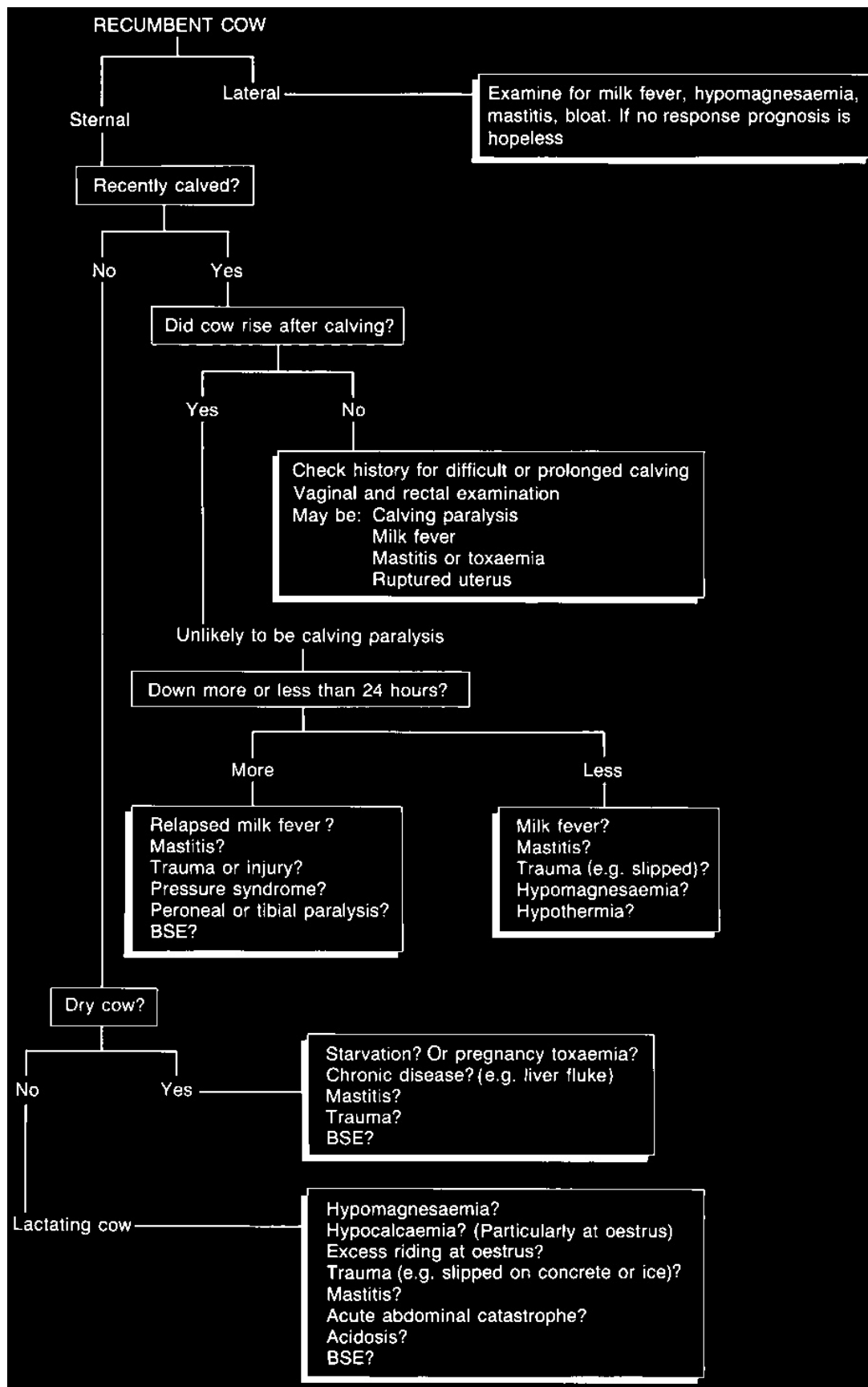


Fig. 46.2

Aid to diagnosis and

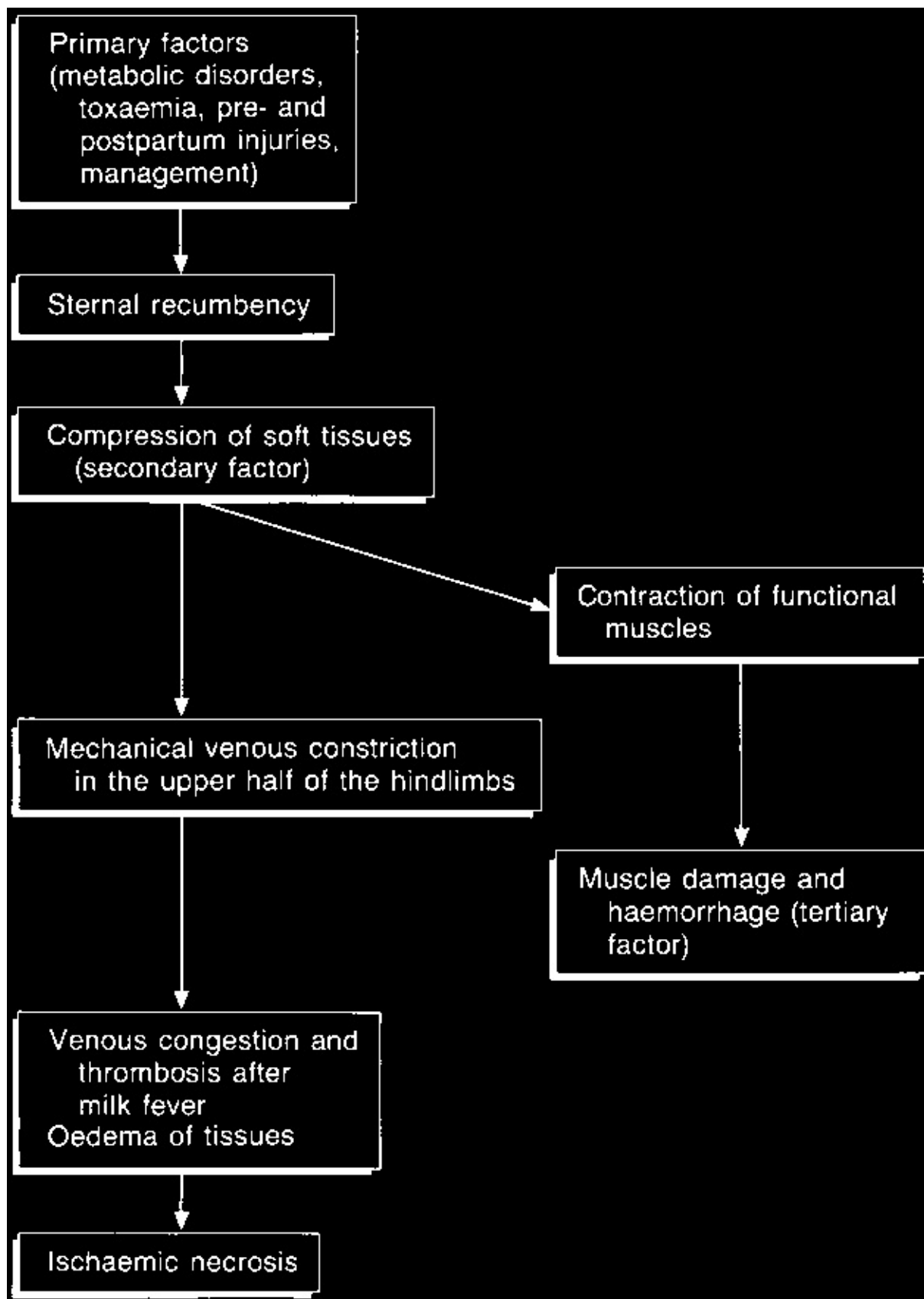
prognosis of the downer cow.

*damage to the musculature of the leg on which the cow
cows first seen with milk fever at morning milking will
is lying. Cox describes this as ischaemic necrosis as a
have been recumbent for six hours, and some for almost
result of pressure. If the cow is in the same position for
12 hours. Many will be suffering some muscle damage,
12 hours continuously then the damage to the muscle is
particularly if they have been lying on unbedded con-
irreversible and the prognosis is therefore hopeless.
crete. This is why prompt intravenous treatment for
This situation is most common following milk fever,
milk fever is essential.*

particularly where treatment has been delayed or inef-

Figure 46.3 shows the pathogenesis of ischaemic

*fective subcutaneous treatment has been given. Many
necrosis.*



Diagnosis and prognosis

Having completed a thorough examination of the recumbent animal that has recently calved and eliminated disease, obvious injury or starvation as a cause of the recumbency, the two most likely problems to be affecting the cow are (i) pressure syndrome (ischaemic necrosis) or (ii) calving paralysis. These cows will be alert, in sternal recumbency, and some will be attempting to rise or crawling along the ground. Appetite for food and water will be good. At this stage it is essential for the clinician to attempt a more exact diagnosis and offer a prognosis. One of the most important inputs that will aid recovery is tender loving care (TLC). The ability and willingness of the stockworker to nurse and attend these cases will be the most important element in successful treatment. So knowledge of the farm and its staff is essential in forming a prognosis, as is the ability to provide soft bedding such as deep litter or a nearby paddock. Diagnosis will be helped if the animal is raised using a Bagshawe hoist and it can be observed

if the cow can take weight on one or both hindlimbs and if either or both hindlimbs are abnormal. Abnormalities to observe include the following:

- *Flexion of fetlock and extension of the hock, which will indicate paralysis of the tibial, peroneal or sciatic nerve (pp. 438, 439).*

Fig. 46.3

Pathogenesis of ischaemic necrosis (from Andrews,

- *Abduction of one or both hindlimbs will indicate 1986).*

obturator nerve paralysis (p. 438).

- *Swelling of the upper hindlimb musculature of one leg would indicate severe pressure syndrome. If the pressure syndrome is so severe that muscle swelling is obvious and one upper hindlimb is larger than the other, prognosis for that limb is hopeless.*

Calving paralysis: If the cow has not risen since calving

- *Inability to extend or flex the stifle, hock and fetlock and has not responded to milk fever therapy, and is not joints.*

suffering from any toxic or septicaemic condition such

- The willingness of the cow to take weight on the forelegs. If the cow is so weak she cannot take weight on the forelegs the prognosis is hopeless. The history should indicate whether calving has been difficult but occasionally a cow will deliver, with difficulty, a large calf but second stage labour may last 3–6 hours. In such cases, calving paralysis may occur. Calving paralysis as extension of stifle and hocks and flexion of the fetlock, a term is preferable to obturator paralysis, sciatic paralysis or other defined nerve paralyses. The damage affected and as long as TLC will be available, the prognosis would be guarded but hopeful. It is surprising how many cows suffering from calving paralysis affecting one or both hindlimbs are showing signs of nerve paralysis, e.g. With calving paralysis one can generalize that if both hindlimbs are showing signs of nerve paralysis, e.g. then the prognosis must be hopeless. If only one leg is affected and as long as TLC will be available, the prognosis would be guarded but hopeful. It is surprising how many cows suffering from calving paralysis affecting one or both hindlimbs are showing signs of nerve paralysis, e.g. result in a variety of lesions in the pelvic cavity. Generally, the bruising and swelling of the soft tissues of the

one hindlimb only will rise seven to ten days after pelvic cavity will damage the sciatic nerve and occasionally the obturator nerve. Obturator paralysis (p.

As an aid to prognosis, several workers have

438), on its own, whether it is affecting one or both

attempted to use blood biochemistry. Serum CK rises

hindlimbs, will not cause recumbency. Such cows can

to astronomically high levels following muscle damage rise, with difficulty, but will show abduction of one or

but its half-life is short and on its own has not proved

both hindlimbs. Paralysis of the sciatic nerve (p. 439) is a reliable indicator of success. Raised serum AST levels

more serious and will prevent the cow rising.

will also indicate muscle damage and this parameter is

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probably more useful than CK levels. As a prognostic

continuous access to food and water. It is surprising

indicator in cows that have been recumbent four days

how many stockworkers will forget to provide water to

or more AST levels can be quite useful. Interest in

a cow recumbent for 24 hours or more.

serum myoglobin levels has been reported from

If the hindlimbs are continuously abducted either

Sweden (Holmgren, 1988), where early work would

due to bilateral obturator paralysis or injury from slip-

suggest that in the first three days of recumbency serum

ping on ice or concrete the application of hobbles (Save

myoglobin levels could be a valuable prognostic

A Cow, Arnolds Ltd) or a soft rope to tie the hindlimbs

indicator. Levels below 3 mg/ml would indicate likely

approximately 50 cm apart will prevent further muscle

recovery.

damage. This will sometimes result in the cow rising

It is unlikely that any one parameter will be shown

immediately, the only factor causing the recumbency

to be of value for accurate prognosis. However, a com-

being the persistent hindlimb abduction.

bination of biochemical parameters and clinical signs,

If there is any doubt as to whether the cow may still

particularly attempting to rise, should be useful. Cows

be suffering from milk fever, 8–12 g of calcium should

recumbent for three days or more must be attempting to rise, as well as bright, alert and eating well, for there to be any likelihood of eventual recovery. The quality of nursing available on the farm must also be considered in making a prognosis. Without good nursing, calcium has not been absorbed completely and a state of hypocalcaemia still exists. Some cows with milk fever that recover will relapse so, if in doubt, intravenous calcium should be administered. A blood sample analysed for calcium levels would provide valuable information, if provided quickly, either as a cowside test or in the practice. Unfortunately, on many large dairy farms where labour is in short supply, there is often an unwillingness to break from the normal farm routine to provide extra attention to a recumbent cow. This information will be available to the regular attending vet-

tice laboratory.

erinarian to the farm and will influence the prognosis.

The most important element of treatment, once the

On such farms, in the interests of the welfare of affected

cow is on a soft bed or in the field, is frequent turning

cows, one will advise immediate slaughter of many

so that she does not spend more than three hours in one

cows, while on other farms where TLC is available, they

position or on one side. Cows that are attempting to rise

would be likely to recover.

will frequently change their position and will move

from side to side. Cows with an injury, whether it is a

nerve paralysis or pressure syndrome, can often be

Treatment

turned to lie on the side opposite to which they are

Where the prognosis is considered hopeless or where it

found, but at the next attempt to rise they fall back on

is known that TLC is absent, all cows should be slaugh-

to the side that is paralysed or injured.

tered humanely as soon as possible. In some countries,

The Bagshaw hoist, which attaches to the pin bones

particularly the UK, such animals can be fit for human and is lifted using a fore-end loader or pulley blocks, is consumption if they are not suffering from septicaemia, often advocated for treatment. This equipment will excessive bruising or any other infectious disease.

cause muscle damage and its continuous use on the However, in the interests of welfare, slaughter must same cow must be questioned on welfare grounds.

take place on the farm. This is perfectly legal in the UK

However, it can be a useful instrument to aid diagnosis.

as long as the cow is bled on the farm and transported

Occasionally, a cow raised by the hoist will take weight

as soon as possible to the nearest slaughterhouse and

on her hindlimbs, remain standing and slowly walk

the carcass is accompanied by a veterinary certificate

away.

that complies with the Fresh Meat (Hygiene and

In recent years interest has increased in various

Inspection) Regulations 1995. If the slaughterhouse is

inflatable rubber bags (Bovijac, Alfred Cox; Henshaw

EC approved, the carcass must reach it within 30

Airlift, J.M. Henshaw; Downer Cow Cushion, Hamco minutes of being bled.

Products). The reasoning behind the use of these air Treatment will of course depend on the diagnosis, but cushions is to allow improved blood circulation to the for the cow that has been recumbent for 24 hours or affected legs. Unfortunately, with most of the inflatable more a soft bed is essential. A box or barn with an earth bags available, once inflated and the cow raised, she frequently struggles and falls off the side of the bag. Cow weather is reasonable the best place for recumbent nets are available and a supportive harness (Downacow cows is in a field. Cows can be transported to the field Harness, Alfred Murray) can be quite useful.

on a gate, on a buck rake or with a cattle net on a fore-

In Denmark, water flotation is being used. The end loader. Wherever the cow is moved it must have recumbent cow is hauled into a drop-sided water tank

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and the tank filled with warm water. The cow will then

in dairy cows at or around calving. Reports of its occurrence stand, aided by the water, and is left in the same position for up to seven hours a day. The system is called an Aqua Lift (Rasmussen, 1988).

appears to occur in high-producing dairy cattle where The use of the Bagshaw hoist (apart from aiding diagnosis) and the various slings and inflatable bags should overfeeding in the dry period results in overfat cows at calving. Depressed appetite after calving and the condition be questioned. If a cow cannot stand unaided there is sequent energy deficit result in a rapid weight loss and little point in raising her to the standing position.

an accumulation of intracellular fat in the liver. The syndrome Repeated use of the Bagshaw hoist will cause extensive drome is associated with an increased incidence of muscle damage and must be discouraged. Until an metabolic, infectious and reproductive disorders such inflatable bag has been designed that will prevent a as milk fever (p. 781), ketosis (p. 793), mastitis (Chapter

raised cow falling off the side, these air bags have only 23) and retained placenta (Chapter 34). Fat cow syndrome has limited use.

drome as described by Morrow (1976) is clinically the same. The Danish Aqua-lift would appear to be the most practical aid to the recumbent cow, but the capital cost of making the lift and providing transport for it may be the most extreme manifestation of the syndrome but is probably only the 'tip of the iceberg' and a much larger number of cows were affected by subclinical FLS, as prevent its widespread adoption.

described by Reid and Roberts (1982).

The only therapeutic agents now licensed for use in cattle which may be of benefit in treating downer cows

Occurrence

are the corticosteroids and the NSAIDs with intravenous glucose as supporting therapy. However, there

The FLS was thought to be widespread in UK dairy cows in the late 1970s and was probably related to gross is little or no evidence that would suggest any therapeutic agent may have a beneficial effect and TLC

overfeeding of cows during late pregnancy, particularly remains the most likely effective treatment.

with the use of high levels of concentrate foods during the late dry period. However, 'steaming up' with concentrates is now out of favour and is rarely practised in

Prevention

the UK, which is probably why the clinical incidence of

Selecting bulls with known shorter gestation lengths

FLS has apparently declined in recent years. However,

and ease of calving scores can, in part, prevent calving

in seasons where grass is in abundance there is still a

paralysis. Feeding of dry cows should also be monitored

danger of cows becoming excessively fat if their diet is

and frequently rationed, particularly in seasons where

not restricted during the dry period and hence FLS can

grass is plentiful. Overfat cows (condition score 3.5 or

and still does occur. At worst 50–90 per cent of freshly

over) more frequently develop dystokia due to relative

calved cows can be affected with FLS, although on some

fetal oversize. If a particular bull has been identified as

farms the incidence was very low or even non-existent.

causing dystokia, then all cows remaining that are pregnant to him should be considered for induced parturition where severe FCS exists (see also p. 796).

Mortality up to 25 per cent has been reported in herds where severe FCS exists (see also p. 796).

tion using long-acting corticosteroids (see p. 687).

The pressure syndrome is a preventable problem.

Pathogenesis

The majority of cases are a sequel to milk fever where treatment has been delayed or is inadequate. Intra-

As most dairy cows in early lactation are in negative energy balance they mobilize energy reserves of fat and venous therapy with 8–12 g of calcium as soon as possible after the cow becomes recumbent, accompanied by muscle and consequently lose body weight and condition. moving the cow into sternal recumbency and turning her so that she is lying on the side opposite to that in the release into the blood of free fatty acids from fat depots and glucogenic amino acids from protein stores. drome occurring. If the cow is down on concrete she

The fatty acids are transported via the blood to various organs, e.g. the kidney, liver and muscle, where they are deposited as intracellular droplets of triglyceride. Thus calcium therapy that fails to be absorbed completely. at one to four weeks after calving there is an increased level of fat in the liver. There is a rise in liver fat levels occurring two to three weeks before calving. This fat

Fatty liver syndrome

mobilization before calving is probably brought about by the changes in hormonal status as the cow

In the 1970s and early 1980s the syndrome of fatty liver approaches calving. The extent of fat deposition in (FLS) or fat cow syndrome (FCS) was widely reported the liver and other organs after calving is probably

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determined by a number of factors including high milk biopsy samples can then be assessed for fat level per-yield potential, body fatness or condition at calving and centage. The technique of liver biopsy in cattle is rela-

loss of body condition after calving. Excessively fat
tively easy to perform and should be used by all cattle
cows tend to have depressed appetites so the fat mobi-
practitioners. Levels of fat, as assessed by staining sec-
lization is exacerbated, resulting in even higher liver fat
tions of liver with toluidine blue or oil red O, in excess of
levels and increased weight loss.

20 per cent would indicate the presence of FLS. Levels
in excess of 50 per cent fat would indicate severe FLS.

Blood biochemistry has been widely explored as a

Signs

measure of fatty liver and various parameters have

The most common indication of the existence of FLS is

been explored, e.g. non-esterified fatty acids (NEFA),

a high incidence of peri- or postparturient disease, e.g.

glucose, AST and glycerol, but these cannot, as yet, be

retained placenta, milk fever, mastitis and ketosis. Cows

interpreted with great confidence. In the USA another

with FLS will subsequently prove to be less fertile.

method of estimating liver fat levels is based on the

However, if FLS is suspected it should be noticed that

*buoyancy of needle biopsy samples in water or copper
a high proportion of the dry cows are excessively fat (in
sulphate solutions.*

*excess of body condition score 3.5) and many of the
cows four weeks after calving are thin (body condition*

Treatment

score less than 2).

There is no proven treatment for either FCS or FLS.

Various empirical treatments have been suggested but

Clinical pathology

there is little evidence that any are of value. The logical

Cows with FLS one week after calving will have signif-

icant alterations in their blood constituents. Free fatty

used for acetonemia. Increasing the glucose supply by

acids, bilirubin and AST will all be increased and

the administration of glucose, glycerol or propionate

glucose, cholesterol, albumin, magnesium, insulin and

and a glucocorticoid followed by the stimulation of

white blood cell count (WBCC) will all be decreased.

protein synthesis by the administration of an anabolic

In cows with severe FCS the WBCC may fall to as low as 3 × 10⁹/l.

to treatment.

Prevention

Pathology

Fatty liver is a sign of cows too fat at calving and losing weight. At post mortem, cows with FCS will have large deposits of fat around the heart, kidney, pelvis and in the mesentery. The main element of a prevention programme is to restrict the feeding of dry cows so that they calve in a body condition score of 2.5–3.0. The liver will be enlarged, with rounded edges and a pale yellow colour. Intracellular droplets of triglycerides will be found in liver cells, kidney, adrenal glands, skeletal muscle fibres and cardiac muscle. In the reduced number of cows suffering from FLS at calving the triglyceride globules are deposited within the liver. Cows in condition score 2.5–3.0 at calving cer-

hepatocytic cytoplasm. The extent of deposition may be
tainly have appetites greater than fat cows and hence
as high as 70 per cent of total hepatocyte volume.

lose less weight in early lactation. However, dry cows
It is important to realize that the findings described
can become fat on grass diets alone in northern Europe
above may be seen in animals that have been deprived
during the spring and early summer, when grass quality
of food 24–48 hours before death. Therefore, post-
is at its best. At such times, therefore, grazing for dry
mortem findings can only assist in the diagnosis of FCS
cows should be restricted or supplemented with low
or FLS when used in conjunction with the herd history
quality fibre such as straw or hay. The rules for preven-
and clinical pathology.

tion of FLS are therefore the same as for acetonaemia
(p. 796).

Diagnosis

The history of increased disease incidence just after

References

calving followed by examination of body condition of

dry cows and cows three to four weeks after calving will

Andrews, A.H. (1986) The downer cow. In Practice, 8, 187–9.

often give a strong indication of the presence of FLS.

ARC (1980) The Nutrient Requirements of Ruminant Live-

Confirmatory evidence is best obtained by liver biopsy

stock. A technical review by an ARC Working Party. Com-of five or six cows immediately after calving. The liver

monwealth Agricultural Bureau.

Major Metabolic Disorders • 803

Cox, V.S. (1982) Pathogenesis of the downer cow syndrome.

Sachs, M. (1988) The prevention of the periparturient hypocal-

Veterinary Record, 111, 67–9.

caemia syndrome by the use of 1 α -hydroxy vitamin D₃. In

Holmgren, N. (1988) Immunological determination of myo-

Proceedings of the XV World Conference on Cattle Diseases, globin as a diagnostic and prognostic aid in downers. In

11–14 October 1988, Palma de Mallorca, Spain, pp. 286–91.

Proceedings of the XV World Conference on Cattle Diseases, Sansom, B.F., Manston, R. & Vagg, M.J. (1983) Magnesium and 11–14 October 1988, Palma de Mallorca, Spain, pp. 276–

milk fever. Veterinary Record, 112, 447–9.

7.

Whitaker, D.A., Kelly, J.M. & Smith, E.J. (1984) Hypomagne Kelly, J. (1988)

Magnesium and milk fever. In Practice, 10, saemia increase. Veterinary Record, 116, 451–2.

168–70.

Morrow, D.A. (1976) Fat cow syndrome. Journal of Dairy

Science, 59, 1625–9.

Further reading

Rasmussen, J. (1988) The downer cow. In Proceedings of the XV World Conference on Cattle Diseases, 11–14 October

Hibbitt, K.G. (1979) Bovine ketosis and its prevention.

1988, Palma de Mallorca, Spain, pp. 282–5.

Veterinary Record, 105, 13–15.

Reid, I.M. & Roberts, C.J. (1982) Fatty liver in dairy cows. In Spence, A.B. (1978) Pregnancy toxaemia in beef cows in

Practice, 4, 164–8.

Orkney. Veterinary Record, 102, 459–61.

Chapter 47

Metabolic Profiles

D.A. Whitaker

Introduction

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Introduction

Timing of blood tests

805

In relation to feed changes

805

The use of metabolic profiles in dairy cattle was pio-

In relation to feeding

805

*neered by Payne et al. (1970) at the Compton Institute In relation to calving
pattern and seasonal feeding*

changes

805

of Animal Health. The approach has been adapted and

Selection of cows

805

modified since then (Blowey, 1975; Kelly et al., 1988; Early lactation group

806

Whitaker, 2000) so that it is now widely used in many

Mid lactation group

806

countries in European-type cows (for example in Italy,

Dry cow group

806

Bertoni & Cappa, 1984; in Chile, Wittwer et al., 1987; Background information

806

*in Germany, Mansfeld et al., 1996; in Turkey, Sevinc & Interpretation of results
at the farm*

806

Aslan, 1998; in Belgium, Opsomer et al., 2000). The

Written advice

807

approach has also produced useful information in low

Metabolites measured

807

yielding cows in developing countries (Whitaker et al., Optimum values

807

1999) by drawing attention to potential constraints and

Energy balance

807

b

by confirming that some were not present.

-Hydroxybutyrate (BHB)

807

Non-esterified fatty acid (NEFA or free fatty acid)

807

In Britain the system has been developed as a man-

Glucose

808

agement aid for dairy farmers and their veterinary sur-

Bile acids

808

geons (Ward et al., 1995; Kelly & Whitaker, 2000). It Interpretation of results for energy balance: cows

contributes information for decision making about

in milk

808

nutrition in a more precise and detailed way and more

Expected forage ME

809

quickly than by other, more conventional approaches.

Interpretation of results for energy balance;

Traditionally forages are analysed, stocks of them meas-

dry cows

810

ured, targets for productivity set, financial constraints

Protein

811

considered – and a ration plan is produced from one of

Urea-nitrogen (ureaN)

811

many perfectly satisfactory sets of software. Inevitably

Digestible undegradable protein (DUP)

813

such a ration has limitations, starting with those of the

Albumin

813

Total protein

813

reliability of forage analysis and of assessment of in-

Globulin

813

takes. Furthermore, the ration may not be delivered

Minerals and trace elements

813

in practice according to the theory of the plan. It may

Magnesium

813

well be that the ration plan is for a cow producing 35

Inorganic phosphate

814

litres of milk at 4.0% butterfat and 3.3% protein, but it

Calcium

814

may actually be the only one like that in a group of

Sodium and potassium

814

cows, some of which may have just calved and others

Thyroxine (T4)

814

of which may be 100 days in to their lactations. If the

Copper

814

ration is not adequate, a farmer and his advisers can

Glutathione peroxidase (GSHPx)

814

detect this from individual milk yields, from milk con-

Manganese

815

stituents, from body weight and condition changes, from

Zinc

815

Cobalt

815

weak oestrous signs and from poor conception rates.

Metabolic profiles on milk

815

All of these, with the exception of butterfat percentage,

Metabolic profiles in maiden heifers

815

even if measured regularly and carefully, take weeks or

Metabolic profiles in suckler cows

815

months to alter enough for the presence of a problem

804

Metabolic Profiles • 805

to be clear. And once it is clear, what actually is it? If

with the first cows checked so that the majority can

the metabolic profile approach is used ‘properly’, then

benefit from the information derived.

it can, within two weeks of a ration being started, iden-

Of equal importance is the need to test as soon as

tify that there is a problem, what it is and what is the

possible (see previous section) after the introduction of

best and/or the most economic solution.

a new ration, so that the judgement of the cows' bio-

The 'proper' use of metabolic profiles depends on

chemistry can be made available as quickly as possible,

care with the timing of blood tests, the selection of cows

i.e. what the cows, the end users, think of the ration.

to be included and the collection and use of background

Therefore planning of metabolic profile tests needs

information about the farm, feeding and feeding system

to be done in advance and should take into account

and physical state and performance of the cows.

both expected calving pattern and feed changes.

Without planning along these lines, time may be lost

and productivity with it.

Timing of blood tests

In relation to feed changes

Selection of cows

As changes in the diet of ruminants require changes in the character of rumen activity, blood samples for metabolic profiles should not be done until two weeks after a major change and activity has had time to adapt. This is because some of the metabolites should not be done until two weeks after looked at, particularly those relating to energy balance, can quickly return to the optimum range as cows adapt themselves, including their productivity, to a nutritional existing component or in access to the same ration do constraint. It is possible for cows to experience a significant energy deficit in the first 2–3 weeks of lactation forage type, such as turnout to grass, housing or the because of intake problems, lose excessive body condition, perhaps modify their milk yield and have their does the introduction of concentrates or of a new type subsequent fertility efficiency suppressed but yet still

of concentrates.

arrive at four weeks calved with all biochemical measurements within the optimum ranges. This is because

In relation to feeding

the common appetite constraint of the new calved has worked its way out and there is plenty of food avail-

There can be changes in biochemical values in blood able for lower performance than anticipated. If blood associated with feeding. These are most marked in cows were sampled at four weeks calved or longer, a farmer receiving all their concentrate ration at milking time. In could see thin, underproducing cows with poor fertility such cases two hours should be allowed to elapse after but with nothing abnormal about their biochemistry.

milking before blood sampling. In circumstances where

Thus the farmer would be entitled to feel the metabolic the major part of the concentrate input is mixed

profile test was of no value. However, if those cows with the forages and is available for most of each 24 had been blood sampled at 14 days calved instead of 27, hours, the timing of tests in relation to feeding is less

the blood results would have been quite different and critical. If lower yielding mid lactation cows are would have identified the nutritional constraint on included (see later), their results can be used as a check productivity.

to see whether there is an effect of feeding on the Individual variations in biochemical values are such biochemical values in the blood samples. Cows should that single cows should not be tested. Groups of no less not be separated at milking time and confined for hours than five should be sampled. They should not be picked without access to food when waiting for blood sampling at random but rather should be typical, average cows as this can also affect the results.

of their stage of lactation. Cows with extremes of performance – either very high or very low – should not be selected. Cows with problems should also not be

In relation to calving pattern and

included because the type of analysis carried out is not seasonal feeding changes

designed to clarify individual problems. It is important

The cow in early lactation is the most important to make all this clear to farmers in advance because because what happens to her in the first few weeks after they cannot be expected to appreciate the limitations calving has the major influence on her subsequent productivity, including her future fertility efficiency. There-Health and Productivity Service (DHHPS) (Kelly & Whitaker, 2000) suggests that selecting cows for metabolic profiles should be carried out at the beginning of each new calving season, metabolic profiles may be best done by the veterinary

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surgeon himself in advance of the test after looking at sampled. Cows tested with longer to go than that tend the calving and production records. If there is a specific to have normal measurements of energy balance even concern, such as a poor conception rate, farmers may though they can still get into difficulty. This is because expect only cows which have failed to conceive to be the period of greatest risk is when the volume of the

sampled. This hardly ever delivers helpful information pregnant uterus increases to the point that it can seriously as any nutritional constraints have been compensated ously inhibit food intake. It follows that, in a seasonal for by then and blood biochemical values are usually calving herd, the first dry cows which come in to these within optimum ranges. The best approach may be to last 7–10 days ought to be blood sampled, so that the include such cows as the mid lactation group (see later). information can be used for the benefit of the others still to come in to the maximum risk period.

Blood sampling a group of dry cows with a month or

Early lactation group

longer to go to calving at the same time can sometimes The definition used for this group is most critical for the provide a useful within-herd comparison with respect reasons given in the previous paragraph. Since the orig- to energy balance. It may also identify the presence of inal Compton metabolic profile (Payne et al., 1970), dietary protein inadequacy – specifically rumen degrad- where a high yielding cow was used as the definition,

able – in the early part of the dry period.

the importance of this group has become increasingly apparent. The definition also has had to be changed to take into account changes in farm practice. The way

Background information

cows are fed now – total mixed rations, increased out-of-parlour concentrate feeding – has reduced the time So that full value can be obtained by the farmer from after calving by when they can adapt themselves to an the metabolic profile approach, information about the unsatisfactory diet. To be sure of detecting the presence cows and the farm should accompany the blood of an energy constraint in particular, blood sampling samples to the laboratory. This should include cow should be carried out between 10 and 20 days calved – identification; last calving date for milkers/expected for less than 10 days and yield is still too far below peak for dry cows; bodyweight – by calculation from heart girth the test to be a realistic one for early lactation per-measurement with a weighband pulled to a constant formance; more than 20 days and some cows will be

5 kg tension is the best, because it is not affected by thin, unproductive and subfertile but have compensated gutfill, and usually most practical, because no mechanical weighing device/crush is required; body condition metabolite values.

score by a palpation method; current daily milk yield; expected current daily milk yield; lactation number; daily supplementary feed intakes; daily estimated

Mid lactation group

forage intakes; analytical description of feeds and Some cows which are past the period of peak yield, current herd milk solids percentages. It is useful to have and so past the greatest period of potential nutritional information on herd size, breed, feeding systems and stress, should always be included. They should be health and fertility. A note of what concerns the farmer between 80 and 150 days calved so that they are still has, if any, should also be made.

relatively high yielding. This group provides a within-herd comparison with the early lactation cows. Without

this it is very difficult to distinguish between problems

Interpretation of results at the farm

caused by constraints on intake of food or protein and

energy content, to identify changes in biochemical

Circumstances where the diagnosis of a nutritional con-

values caused by mistiming of tests in relation to

straint from blood samples is clearly correct, but the

feeding or by oddities in the diet such as silage with a

cause is unclear from a distance and there could be

high butyric acid content and to make judgements on

many, are common. Therefore it is very important that

concentrate/forage usage within the herd.

a final interpretation of what is not working and what

are the best and most economic solutions ought to be

made at the farm with the information from the labo-

Dry cow group

ratory to hand. Farm advisory visits should be made as

As the dry period is so important to the success of the

soon as the results are available and discussions held,

following lactation, blood sampling to make sure nutri-

including farm staff and any other advisers involved.

tion is adequate is essential. However, the nature of the Experience in the DHHPs suggests that such a team measurements which can be made means that primarily approach produces a more balanced strategy and is cows in the last 7–10 days of pregnancy should be more beneficial than each party working in isolation.

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Written advice

important, depending on the metabolite. Group means should be looked at and compared firstly and then the Any advice given should be recorded concisely in individual variations and the number of them within writing and copies given to all participants on the farm. each group. Variations which might relate to performance. This ensures that the agreed path is followed, keeps a record of it and ensures that the fee is for something particular attention because they can provide important tangible.

information on the practical causes of a constraint.

Values within the optimum ranges can also be used, in

Metabolites measured

conjunction with the background information, to give advice of economic importance on concentrate/forage

The number of metabolites which could be measured usage (see following sections on interpretation).

is great. For the sake of cost, not overcomplicating the picture and speed of turn round of results, the number is usually restricted. Metabolites measured

Energy balance

should also be confined to those from which reliable, useful and practical information can be derived.

b -Hydroxybutyrate (BHB)

Consequent actions will therefore be beneficial. It In cows in milk the optimum level for b-hydroxybutyrate (BHB) in blood is below 1.0 mmol/litre. BHB protein nutrition are the most important and constitute, reflects fat mobilization as an energy source and so between them, at least 90 per cent of identified higher values are associated with greater degrees of constraints (Kelly & Whitaker, 2000).

negative energy balance. Values below 0.6 mmol/litre
Chosen metabolites need to be stable in the blood
represent a situation where cows are unlikely to be
samples after collection for two to three days while in
losing body condition. Between 0.6 and 1.0 a low and
transit to the laboratory. Methods of analysis need to
acceptable rate of fat mobilization may be taking place,
be quick, accurate and automated. Each laboratory
depending on the stage of lactation. Above 1.0 mmol/
must use an outside agency as a continual check on
litre health and productivity will be affected. Cows with
quality control and so reliability of results.

ketosis will probably have values over 2.0 mmol/litre. In
a small proportion of cows BHB results, if very high in

Optimum values

early lactation, remain outside the optimum range after
What constitutes a 'normal', or rather optimum, value
energy balance has been restored and body condition
or range of values for a metabolite in blood is crucial.
is being regained.

In the DHHPS (Kelly & Whitaker, 2000) we used orig-

Dry cows in the last month of pregnancy should have initially the values proposed by Payne et al. (1970). These BHB values below 0.6 mmol/litre because they should were based on population means from 2400 cows plus be restoring and maintaining energy resources. or minus two standard deviations. The DHHPS laboratory has been operating since 1978, with thousands of days. Butyric acid from grass silage, directly absorbed blood samples passing through every year, accompanied across the rumen wall, has been blamed for increased blood values. In our experience since 1978 this is a rare problem. If it is present, all cows with access to that silage will have high BHB levels in blood. Including modified. Account must also be taken of the fact that lower yielding mid lactation cows receiving the same variation in some metabolites is skewed. For these we basic diet, as a within-herd comparison, provides the

have carried out a lognormal transformation (Whitaker
most reliable means of ruling it out – if those cows have
et al., 1983) before arriving at an optimum range. For lower results than the
early lactation animals which are
some metabolites, such as urea, the interest primarily is
eating the same silage.

in low values and so in variation only one way from the
optimum. Some controlled trials, where detailed meas-

Non-esterified fatty acid (NEFA or

urements of weight and condition change have been
free fatty acid)

made with blood sampling (e.g. Whitaker et al., 1989,
1993), have confirmed the otherwise largely anecdotal

The optimum level of non-esterified fatty acid (NEFA)

evidence for what is optimum. These ranges are the

in cows in milk is below 0.7 mmol/litre and for dry cows

ones quoted in the following pages.

within the last four weeks of pregnancy 0.4 or below.

Optimum values should nevertheless not be used too

The reasons for the difference are the same as for BHB.

precisely. Small variations from optimum may not be

NEFA is a more direct measure of fat mobilization than

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BHB. It may rise more quickly and it often returns to

Bile acids

within the optimum range quite quickly as well,

Some laboratories include bile acid measurement in

even when negative energy balance is still present.

metabolic profiles as a means of assessing subclinical ill

Cows with ketosis have values in their blood above

health, with reference in particular to heavy fat infil-

1.5 mmol/litre.

tration (lipidosis) into the liver in the peri-parturient

NEFA is less stable in the blood sample after collec-

period as a consequence of severe negative energy

tion than BHB. It is reputed to start to rise after 48

hours. There is not firm agreement on the value of

this. West (1991) found bile acids to be significantly

in samples analysed four to five days after collection.

correlated with the degree of a variety of liver diseases

Severe stress prior to blood sampling is also reputed to

including lipidosis, whereas Garry et al. (1994) found no cause the level to rise, but this also has not apparently

correlation between bile acid level and liver fat content.

happened in the ten years that we have used this

Schrotter et al. (2000) found bile acid levels to be within metabolite. Sampling groups of no fewer than five cows

the normal range in cows with subclinical disorders

and including those in early and mid lactation as within-

and chronic hepatopathy. In the DHHPS (Kelly &

herd comparisons enable these potential problems to

Whitaker, 2000) we have carried out this analysis on

be set aside.

several hundred occasions alongside those for BHB,

plasma glucose and NEFA and have found that it did

Glucose

not appear to add sufficiently to the information

obtained to justify its regular inclusion.

Glucose was the first metabolite used in the Compton

Metabolic Profile (Payne et al., 1970). Because of the fundamental requirement for glucose in the tissues,

Interpretation of results for energy

homeostatic mechanisms are very strong and variations

balance: cows in milk

were not found to be great. Whole blood or serum
Raised BHB and/or NEFA values with or without low
tended to be used, neither of which are as accurate with
plasma glucose results indicate that cows are experi-
respect to energy balance as the plasma content. Added
encing a dietary energy problem and not necessarily a
to the difficulties was the fact that glycolytic enzymes
dietary energy deficiency. The easy response is to rec-
start to break down glucose after sampling. So either
ommend an increase in energy supply through more
analysis needed to be carried out very quickly or blood
concentrates. This can be completely the wrong thing
needed to be collected into a tube containing oxalate
to do, which illustrates that identifying that there is a
fluoride as an anticoagulant. This does not mix as easily
problem is the easy bit and only the start. Identifying
as other anticoagulants and sample tubes have expiry
the cause(s) and the practical solution(s) is the diffi-
dates which are easily overlooked. Glucose is also the
cult bit. Using the type of background information

most likely metabolite to change with delay in transit described earlier and seeing what is actually happening and it goes down. All this has meant that glucose at the farm are essential parts of the process.

acquired a reputation as not being very useful in assess-

There are a number of possible reasons for dietary ing energy balance with certainty.

energy problems in milking cows, all of which should be

Our experience suggests that if blood is collected considered:

into tubes within their dates, mixing is thorough but gentle and analysis is carried out within four to five days

- The forage component of the diet is being expected of collection, plasma glucose is a sensitive measure of to contribute more energy than is reasonable, i.e.

energy balance – even within the optimum range which not enough concentrates are being fed.

is above 3.0 mmol/litre. Sampling groups within a herd,

- Less concentrate is being fed than supposed as for BHB and NEFA, allows a check for abnormal- because of misunderstanding or feeders are in need

ities not associated with nutrition.

of recalibration.

There are some nutritional circumstances where

- *Too much concentrate is being fed, resulting in a the predominant finding in a metabolic profile is that concentrate to forage ratio in the rumen which is plasma glucose levels are low but BHB and NEFA are in excess of 3 : 1 on a dry matter basis. This causes within their optimum ranges. These appear to be associated often with a dietary shortage of fermentable*

For this calculation grazing, silages, hay and straw metabolizable energy (FME), judging by the responses only count as forages. Often the only way to arrive obtained to dietary adjustments.

at a ratio is to add up the amount of dry matter

Plasma glucose does increase sharply to over 5.0 mmol/ being supplied as non-forages, calculate the theoretical total dry matter intake of the cow and prior to sampling.

Plasma glucose does increase sharply to over 5.0 mmol/

being supplied as non-forages, calculate the theoretical total dry matter intake of the cow and prior to sampling.

assume the difference is the amount of forage dry

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matter being eaten. Another way of looking for this

• Grass silage can be very low in fibre – uncommon problem is from an expected forage ME calculation but can occur if of high quality, having been harvested (see later). Rationing, which is expecting the forage vested very early in the spring. Mixing 0.5 kg of component to provide less than the equivalent of chopped straw with it may be enough.

the energy needs of maintenance (about 65 mega-

• Access to grazing is restricted – strip grazing joules), is likely to be threatening this ratio. Loose systems or paddocks not changed often enough; dung provides supporting evidence.

set stocking systems allowing the development of

• The concentrate amount offered each milking is fibrous, less nutritious grass.

too high – it is either not eaten or, if it is, causes

• Buffer feeding to grazing may be inappropriate – transient rumen acidosis; 4 kg per milking is a

poor quality forage used; timing of access; substitute instead of supplements grazing; site of access

reasonable top limit.

- *Concentrates supplied contain too much readily inappropriate.*

soluble carbohydrate, causing rumen acidosis.

- *Too much digestible undegradable protein and not*

- *Single feeds of concentrates, unmixed with forages, enough energy in a ration can occur.*

supplied at times other than milking, may be too

- *Salt deficiency is common in some winters.*

great to ensure even intakes between cows – 2 kg

- *Water deprivation is easily overlooked – trough per cow per feed is a reasonable limit.*

size, site, filling rate and cleanliness can all be

- *Not enough conserved forage is being offered – constraining.*

ideally fresh forages should always be readily avail-

- *Inadequate dry cow nutrition and management*

able to all cows at all times. Empty troughs militate

can be unnoticed – changes in diet components at

against the cows who are most at risk. Between 5 calving; failure to allow dry cows to realize their and 10 per cent more food than the cows are going potential appetites in late pregnancy; fat mobilization and body condition loss before calving.

the surplus removed at least once daily.

- *Lack of reasonable cow comfort and space can contribute considerably to energy problems for new calved cows and especially heifers.*
- *Silage or silage concentrate mixtures can become*

enough.

- *Loss of nutrients from silage due to secondary fermentation may occur – from exposure to air or water troughs, to be there when food is put out to water at the clamp face by the way it is handled or see what happens, both with reference to men and cows, because rations are mixed too infrequently. Silages and to check troughs and self-feed faces at other times*

of high dry matter content are more at risk. The when at the farm. What is said or believed to be happening warm to the hand or with steam rising from pening is not always so!

it on a still day shows this as a problem.

There are rare circumstances where BHB levels are

- Conserved forages may be unpalatable, in UK circumstances grass silages which are very wet or very raised and glucose levels are low which do not reflect an energy problem. Observation of the cows' body condition and their performance can support this, but give some indications.

looking at the results in the different groups should

- Access to conserved forages is restricted, e.g. by confirm it. If cows in the mid lactation group, producing inadequate trough space and design, troughs not filling much less milk, have got the same scale of out-of-filled evenly, self-feed faces tightly compressed, range results as cows in early lactation producing a lot severe electrified barriers, not enough ring feeders

more, then some aberration of the timing of sampling or the feeding facilities or the route to them are in relation to feeding or of a direct effect of a diet con-
exposed to bad weather.

stituent, such as butyric acid in silage, is likely to be

- Different forages fed unmixed – grass silage available as an alternative to maize or whole crop, but not mixed with them, does not deliver the same

Expected forage ME

potential nutritional benefits as a mixture.

- Conserved forage is of low energy content –

More specifically, it is useful to look at the way the provided it is palatable this can usually be balanced rationing is set up for the individual cows and what contributed by other components of the diet, if the problem is being sought from the forage component – is recognized. Laboratory analysis of forages the expected forage metabolizable energy (ME) in can exaggerate the energy content, particularly megajoules. The energy the cow needs, the required

the fermentable metabolizable energy in maize

ME (AFRC, 1993), is calculated from the cow's body silage.

weight, milk yield and milk quality. The energy supplied

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as concentrates, the fed concentrate ME, i.e. not

their expected forage ME figures are at least the same

forages, is calculated from the information obtained

as those for cows 1–5 or perhaps even greater. So blood

from the farm on fed amounts and analysis, actual

tests showing the presence of no nutritional constraint

analysis if available or book values if not. The differ-

– often looked at by farmers as a waste of money – can

ence is the energy that the rationing assumes is supplied

be used to guide the important economic management

by the forages. Similar calculations can be made to

of concentrate usage.

assess the energy contribution of forages where systems

Some further points need to be considered, however.

not based on metabolizable energy are used.

The cost of concentrates and forages may not, in every

country, be such that lower use of concentrates is more

$\text{Expected forage ME} = \text{required ME} -$

economic. In addition, a reduction in concentrate input

fed concentrate ME

will mean that cows will look to increase forage intakes.

So there has to be forage offered/available to them to

The amounts of concentrates eaten per cow per day

do this or milk yields will suffer. There may be impor-

are usually the most accurate information available on

tant implications for forage stocks too because they will

feeding – certainly more accurate than the amount of

be used more quickly. Also it is necessary to consider

forage eaten. Where concentrates are mixed in with

the protein content of the ration. A reduction in con-

silage offered ad lib, individual concentrate intakes are concentrates in this context
is of energy supplied, but it may

less accurate and group means should be considered

mean that effectively protein input is reduced as well

first, not least because it is these which will have been

and that could also reduce milk yield. The protein

used to design the ration.

content of the reduced level of concentrate fed may
Where metabolite values have been disturbed
need to be increased to avoid this. Blood urea levels
because cows have been blood sampled too close to a
may give some guidance. If they are relatively high, no
feed, cows will have similar out-of-range levels, even
increase in protein content may be necessary. This is
though expected forage ME figures vary widely.
likely to be so where this assessment is being made in
This calculation is of further use when assessing the
cows at grass.

economic aspects of concentrate/forage usage. Table
It is worth repeating that optimum levels of BHB,
47.1 shows an example of a blood test in which all the
glucose and NEFA can be found in thin, underproduc-
energy measurements were within the optimum ranges,
ing cows in modern feeding systems after four weeks
indicating that all these cows were having their energy
calved, who have adapted their performance and their
needs met. Cows numbered 1–5 are within the second
metabolic state after experiencing serious energy con-

and third weeks of lactation, the period of greatest strains earlier in lactation.

potential nutritional stress. Because they are in energy balance, they must be achieving from forages the figures

Interpretation of results for energy

shown in the expected forage ME column. If they can,

balance: dry cows

then the rest of the herd can too. This means that cows

6–10, who are being asked to get less from forages,

The dry period needs to be one of maintenance and

should have their concentrate input reduced so that

restoration of resources for cows, especially high yield-

Table 47.1

Examples of individual cow results for assessing concentrate usage/forage contribution to energy needs.

Cow

Days calved

BHB mmol/l

Glucose mmol/l

NEFA mmol/l

Expected forage ME (MJ)

1

10

0.4

3.4

0.5

169

2

0.6

3.2

0.2

175

3

to

0.3

3.5

0.3

135

4

0.4

3.2

0.4

181

5

20

0.4

3.2

0.4

150

6

80

0.6

3.4

0.3

152

7

0.7

3.3

0.4

101

8

to

0.3

3.4

0.2

131

9

0.3

3.2

0.3

95

10

110

0.4

3.3

0.3

121

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*ing ones, to be able to realize their productivity and
matter intakes will be reduced. Expecting cows
longevity potentials. Body condition change during the
to 'fill up' even to 12 kg dry matter by providing
dry period and body condition score at calving – aspects
free access to straw does not work. Failure to*

of energy balance primarily – influence productivity provide enough fresh, quality forage, in cows and fertility in the following lactation (e.g. Domecq receiving concentrates, is the commonest cause et al., 1997; Markusfeld et al., 1997). So identifying the of negative energy balance in late pregnancy.

presence of undesirable situations during the dry period

(9)

Feed 2–3 kg of good quality concentrates/cow per is important. Body condition scoring, if done once a day. This increases the energy density of the total week, is sensitive enough until the last two weeks of food intake, compensating for the dropping pregnancy. Then blood sampling may be the only way appetite. It allows the rumen environment and to confirm that there is a problem.

lining to adapt fully to concentrates by the time

The following is a dry cow management ‘best practice’ list against which current practice on a farm can be two weeks.

checked where a problem has been found.

(10)

Ensure that these concentrates contain appropriate digestible undegradable protein (DUP) if enhanced milk protein production is desired.

From drying off

(11)

Ensure that the forages contain adequate ERDP

(1)

Look upon the dry period as a unique opportunity for rest and recreation for cows. and if not compensate through the concentrates.

Specialist dry cow concentrates are usually

(2)

Provide a clean, comfortable environment in the formulated assuming that cows, if getting grass first month of the dry period.

silage, will be getting enough ERDP from that.

(3)

Manage cows so that they go dry at body condi-

This is not always the case. A ration with maize

tion score 2.5–3.0 on a scale of 1–5 and maintain or whole crop cereal silage included is more at it until calving (p. 10). This may mean reducing risk.

concentrate inputs in late pregnancy, which is (12)

Ensure that appropriate minerals are incorporated where much overfatness develops. It may mean restricting the quality of forage available in the ration minerals are satisfactory for most first month of the dry period to stop excessive farms for most of the time and only need change increase in condition.

ing if cases of hypocalcaemia start. Free access at (4)

Confine overfat cows at drying off on a diet of this stage is not satisfactory.

straw balanced with adequate effective rumen (13)

Include pregnant heifers in this close to calving

degradable protein (ERDP) to maintain rumen group, unless to be kept separately in lactation – function. The restriction should be of quality and but even then provide the same ration.

not quantity of forage so that rumen fill is main- (14)

Oversize calves come from overfeeding in the tained as far as possible. Only 10–14 days on this seventh or eighth month of pregnancy, extended regime is advisable and it must be stopped gestation length (a function of the genetic make- absolutely by the time of one month to go to up of the sire) or an underdeveloped heifer with calving.

a genetic potential to be a lot bigger. The rate of (5)

Feed overthin cows at drying off generously growth of the calf in the last month of pregnancy enough for some condition to be restored.

is at its greatest but it cannot be influenced by (6)

Provide free access minerals during the first feeding in that month – either to increase or month of the dry period if they cannot be mixed decrease the eventual size of the calf. in with the forage on offer, which is preferable.

(15)

Provide the same fresh concentrates and forages in calving boxes/pens. Also ensure fresh water For the last month of the dry period supplies.

(7)

Provide the best environment and forages on the farm.

(8)

Offer 24 hour access to fresh, quality forages in

Protein

the last month of pregnancy. All of the forage types which are going to be fed in production

Urea-nitrogen (ureaN)

should be included. Cows should be challenged to eat between 12 and 15 kg of dry matter each

If rumen function is satisfactory blood ureaN level is day so that they can eat more and get to above 1.7 mmol/litre. In some laboratories urea rather maximum intakes more quickly after calving. If than ureaN is measured. This makes no difference more than 0.5 kg of straw is included, total dry except that the values are higher by a multiplication

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RUMEN

ABOMASUM

SMALL

INTESTINE

Dietary

Degradation

Multiplication

Microbes

FME

and

of

digested

ERDP

fermentation

microbes

as

protein

amino

source

acids

NH₄

Heat

Liver

Fig. 47.1

*Diagram of degradation and
digestion of ERDP and FME by ruminants.*

Blood urea

*factor of 2.14. So the optimum for urea is above
rumen function and so will look for more forage to eat.
3.6 mmol/litre.*

*Therefore for the approach to work, more silage must
Blood ureaN reflects the rate of arrival in the rumen
be made available to them. However, account must be
of effective rumen degradable protein (ERDP) and the*

*taken of the fact that forage stocks may be depleted
balance there with fermentable metabolizable energy
more quickly than previously anticipated.*

(FME), as illustrated in Fig. 47.1. Low values are the

*There is a further practical risk arising from the fact
most important from a practical, nutritional point of
that the theoretical ration plan being followed*

view but care needs to be exercised in distinguishing

often indicates that ERDP is adequate – the reason is

between values which are low because of low total food

probably a shortcoming in the laboratory analysis of

intake or because the diet does not contain adequate

forages. Feed advisers may therefore be reluctant to

ERDP. This is one of the reasons for including mid lac-

agree to more ERDP – partly because it is usually

tation cows in a metabolic profile test as a within-herd

cheaper than the existing ration – and often suggest

comparison. If the new calved cows, which are the most

‘higher quality’ protein, which is usually digestible

likely to have appetite constraints, have low ureaN

rumen-undegradable protein (DUP).

That,

by

results and so do the mids, who should be able to eat definition, passes through the rumen, plays no part in readily, then the diet will be short of ERDP. If the mids rumen function and does not work where ERDP is have optimum ureaN results though, the diet should be short.

adequate in ERDP but the new calved are not eating Low ureaN results in dry cows are quite common for as much as they should. If diet intake is the predomi- the reasons indicated in the dry cow/energy section. As nant problem, some of the energy metabolites may be a dietary shortage of ERDP inhibits rumen function, out of their ranges too. Obviously the approach to this reduces potential appetite and enforces a change in is different – and importantly so – from a diagnosis of rumen function at calving, it is important to identify it. a dietary shortage of ERDP.

The same high level of ureaN can reflect (i) an excess

On farms where maize and grass silages are not

of ERDP and adequate FME, (ii) an excess of ERDP mixed but are offered so that cows can make a choice, and inadequate FME or (iii) adequate ERDP but low or where one silage is fed for 12 hours and the other for FME. Only (ii) and (iii) need action and some other the next 12 hours, low blood ureaN may not always evidence of nutritional imbalance is desirable to make relate to an overall diet shortage of ERDP. The best the diagnosis more certain – some energy metabolites solution then may be to promote mixing of the forages outside their reference ranges for example. Where rather than a change in concentrates.

ureaN results are above 3.3 mol/litre and there is evidence of an energy constraint, it is worth increasing the to be supplied, cows will respond with increased milk level of FME in the diet. This may mean substituting yield within 24–48 hours. They will have improved energy sources not available in the rumen, such as pro-

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tected fats, with wheat or barley for example, which are

liver health or a poor supply of amino acids from the high in FME.

diet. Any effect from diet is long term. Low levels will

It is not advisable to try to correct an excess of ERDP

only appear after prolonged and severe underfeeding

in relation to FME (examples ii and iii) by reducing the

of protein. If the cause is disease, the effect can be more

amount of ERDP in the diet. This is because it may be

immediate and may involve a raised globulin level as

impossible not to reduce the DUP supplied as well and

well.

so precipitate an unwelcome fall in milk yield. The sup-

Serum samples as opposed to plasma produce

position that high blood urea levels directly affect fer-

albumin results between 2 and 5 g/litre lower.

fertility remains controversial and unproven. Certainly

many cows retain good fertility, or even have improve-

ments, when turned onto grass in spring with high blood

Total protein

urea levels. It is possible that observed subfertility in

Measurement of total protein is required to deduce

cows with such high levels occurs primarily because of globulin level. This is not directly measured and is the negative energy balance. Therefore care should be difference between albumin and total protein. As total taken, if looking at metabolic profile results in herds protein consists of something based on diet and some- with fertility problems, before drawing definitive thing based on disease and inflammation, it should not conclusions.

be shown on result sheets. If it is, people who are not aware of its content frequently draw exclusively dietary conclusions, which are incorrect.

Digestible undegradable protein (DUP)

Higher yielding cows cannot meet their total protein requirements from ERDP and the production in the

Globulin

rumen of microbial protein alone; DUP is required as Globulins are produced in response to inflammation of well. This passes through the rumen unaffected and is the chronic type. The optimum level is below 50 g/litre. digested in the abomasum and small intestine as an

If it is higher, the individual cow concerned has a recent, additional source to microbial protein. There is no way acute or chronic inflammation problem. The globulin of directly assessing the dietary adequacy of DUP measurement is crude and not specific, consequently a through metabolic profiles. However, if milk yield is raised value does not indicate with any accuracy the below expectation and cows are maintaining good body severity of the problem or how recently it took place. In condition in early lactation – and if ERDP supplies are some cases a cow may have some disease process clinically adequate – increased dietary DUP should be tried. As cally obvious, but the globulin has not risen at the time with ERDP, the milk yield response should be within of blood sampling. The whole value of the measurement 24–48 hours and also as with ERDP cows will be is to draw attention to the possibility of individual or looking for greater forage intakes. More must be made herd disease problems and so stimulate discussion and available and account taken of a greater rate of depletion investigation. The commonest individual complaints are

tion of conserved forage stocks.

mastitis, metritis or lameness. The most likely herd

Before embarking on this approach a metabolic

problem in the UK is infection with Fasciola hepatica, profile test to confirm satisfactory energy balance and

when albumin levels will be low as well (p. 276).

adequate dietary ERDP is strongly advisable. As part

of a dietary shortage of DUP is often a complaint that

milk protein production is low and as energy con-

straints are the commonest cause of this, the check

Minerals and trace elements

metabolic profile test is the first step in such a com-

plaint. Farmers need to be advised as well that feeding

Magnesium

more DUP, where there has been a dietary shortage,

Blood magnesium levels should be between 0.8 and

should result in an increase in milk and milk protein

1.3 mmol/litre. This reflects current dietary intake rather

yield but not necessarily an increase in milk protein

than body tissue resources from which magnesium is

percentage.

not readily available. Low blood levels can therefore be a matter requiring urgent action to avoid clinical hypomagnesaemia. A number of slightly low magne-

Albumin

sium results may indicate subclinical hypomagne-

Albumin is a protein that is synthesized in the liver. The saemia, which can affect appetite, performance and optimum level is over 30 g/litre. Low levels reflect poor behaviour.

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Because of the complex nature of the dietary factors to identify an iodine deficiency or the presence of which interfere with the absorption of magnesium from goitrogens, although the iodine content of the gland the diet, a check test 10–14 days after increased supplementation is advisable to make sure that it has worked. provide confirmation. Iodine deficiency has been associated with poor production as well.

Low dietary magnesium in the dry period may be involved in the incidence of hypocalcaemia, but low

Measurement of inorganic iodine in plasma is also blood levels may not be detected until a few days before widely practised. Caution is required over interpretation as the level is very sensitive to variation in daily but it can occur and, if it does, is usually fatal. Under-iodine intake. A cow which has not eaten will have a feeding and exposure to severe adverse weather conditions result primarily because of appetite.

tions are the predisposing factors.

Copper (see also p. 298)

Inorganic phosphate

Blood copper levels are not an accurate guide to body Plasma values also reflect current dietary intake and liver status where most of it is found. So, while the primarily. Optimum levels are between 1.4 and optimum level is over 9.3 mmol/litre, clinical copper deficiency is 2.4 mmol/litre.

Unlike magnesium and calcium, deficiency can be found with results above or below that. however, low levels in diet and blood can be tolerated

A number of individual results below that in a meta-

without harm to health and fertility for months

bolic profile merit further, clinical enquiry because defi-

(Brodison et al., 1989). A check blood test, a few weeks ciency, if present, will affect health and productivity.

after some low results occur, is advisable before

Recent reports of copper toxicity in cattle, perhaps

embarking on potentially expensive and probably

associated with higher absorption rates from the use

useless supplementation. Measurement of this mineral

of chelates or proteinates in minerals or overzealous

is usually carried out because of the widespread mis-

supplementation, suggest that blood levels over

conception that dietary deficiency is a common cause of

20 mmol/litre should be followed up (p. 948).

poor fertility because of the aggressive marketing of

Much more difficult is assessing the possible primary

supplements to meet this perceived need.

role of excessive molybdenum on fertility and second-

Hypophosphataemia can be a contributory cause of

arily on copper status (Phillippo et al., 1987). Suttle the downer cow syndrome (pp. 439, 797). However, mis-

(1993) suggested that this problem would not be diagnosis can take place because a blood sample taken present unless the forage offered contained more than after a cow has been recumbent and not eating for 24 5 mg/kg molybdenum on a dry matter basis. Telfer et al. hours may be low because of that only. Firm diagnosis (1996) believe that forage values can be lower than this can only be on response to treatment with phosphate. and the problem may still be present because of molybdenum in concentrates. They have developed a complex

Calcium

system of testing blood involving a ratio between plasma copper and serum caeruloplasmin, trichloroacetic Homeostatic control of the level of calcium in the blood acid precipitated copper and superoxide dismutase. is so strong that variations are small and do not reflect This may be too arduous for many laboratories. If this dietary intake at all. Measurement of calcium should not problem is suspected – and forage analysis frequently therefore be carried out as part of a metabolic profile encourages farmers to think it is – the most practical

*and should only be done as part of clinical diagnosis.
solution may be to administer for six months an
intrareticular copper bolus to alternate cows as they go*

Sodium and potassium

dry or calve and see what happens.

*Homeostatic control of sodium and potassium is also
too strong to allow measurement in blood to be a reli-*

Glutathione peroxidase (GSHPx) (see p. 302)

able indicator of dietary intake.

*The optimum level is over 50 units/g of haemoglobin or
over 15 units/ml of red blood cells. As this is an enzyme*

Thyroxine (T4)

*containing selenium and as it has a half-life of some
Thyroxine provides an indication of the iodine status
weeks, GSHPx values reflect historical selenium intake.*

of the cow. The optimum level is above 22 nmol/litre

*Low values in early lactation cows, satisfactory ones in
(Whitaker, 1999). Low values have been associated with
mid lactation and lower but still satisfactory ones in dry
abortion and stillbirth. Weighing the thyroid gland of
cows describe a dietary deficiency in the dry period,*

stillborn calves does not appear to be sensitive enough with production rations taking care of it. GSHPx values

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can be dropping while selenium intake is rising and vice measured and is virtually the same as the level in blood.

versa. Isolated groups of cattle sampled can therefore Methods of assessing energy balance through measure-produce a deceptive picture. Clinical assessment of pos-

ments in milk are available – b-hydroxybutyrate, acetosibly related problems is necessary – such as muscular acetate, ketones. There are technical difficulties, for dystrophy, poor fertility in maiden heifers, retained placenta because of volatility. The practical value is less clear at the time of writing and measurements may not the relationship with vitamin E, measurement of this in be as sensitive as for metabolites in blood. Minerals and blood may be advisable as well if a problem is suspected. Blood selenium itself can be measured, but there is no reliable information on reference values. Further-analysis is too expensive for routine use.

more, as milk is manufactured for the specific purpose of feeding a young animal, it contains concentrated amounts of many elements, bearing little or no relation **Manganese** (see p. 305)

to levels in blood, body or dietary intake.

Estimation of blood levels is practised but the relevance

Bulk milk measurements are generally unhelpful.

of results is questionable. Since Wilson (1966) found no

Analysis for urea is widely practised and frequently

differences in conception rates between supposedly

misunderstood by farmers causing unnecessary con-

manganese deficient supplemented and unsupple-

cern. A number of milk purchasers have now given this

mented herds no published evidence has appeared that

up. The problem is largely because the result is an

this trace element is limiting on fertility. More recently,

average for the herd and because of variations in urea

Gelfert and Staufenbiel (2000) found no evidence of

in cattle which relate to intake rather than to diet

effects on fertility in 70 dairy herds in Germany where,

content. Only if bulk milk urea is very low should there

*according to published values for the manganese
be any response and that should be a proper metabolic
content of hair and serum, deficiency should have
profile to determine the nature of the constraint.
existed.*

Metabolic profiles in maiden heifers

Zinc (see p. 305)

*In response to complaints of poor conception rates in
Optimum values for zinc concentrations in blood are
heifers, blood samplings in housed animals often show
equivocal. In UK circumstances the likelihood of
diets to be inadequate in ERDP. Sometimes minerals
primary zinc deficiency is low. Furthermore, specially
and trace elements do not all fall into optimum ranges
prepared sample tubes need to be used to avoid con-
either. Anecdotal responses are achieved by correct-
tamination from the glass or the cork.*

*ing the ERDP query. So investigation of this type of
complaint should not be confined to minerals and trace*

Cobalt (see p. 295)

elements, which is where feed advisers and farmers

often expect the problem to lie.

Levels of vitamin B12 in blood below the reference value of 90 ng/litre are commonly found in productive, healthy and fertile cattle. It is probable therefore that

Metabolic profiles in suckler cows

what is usually measured is not biologically active or cattle can exist with lower levels than this without

In the DHHPS metabolic profile samplings are often coming to harm. It may therefore be preferable to avoid carried out in suckler herds on cows in late pregnancy altogether the expense of a laboratory analysis of and when new calved. While these sometimes suggest doubtful practical value.

the presence of some mineral or trace element shortcomings, it is common to find evidence of considerable negative energy balance at these stages, using the same

Metabolic profiles on milk

optimum blood values as in dairy cows, which may inhibit subsequent fertility. As with dairy cows, waiting If the same rigorous 'rules' are applied to the selection to blood sample until inseminations are about to start

of cows, timing of tests and the use of background information is likely to miss identifying the presence of the most probable nutritional constraint. Equally, if sampling is not done until a problem of low pregnancy rates balance and protein intakes. Failure to follow the 'rules' is apparent, the true cause may not be revealed by it. and failure always to check energy and protein together Then it may be better to wait until pregnant cows are lead to erroneous conclusions. Milk urea can be readily available before the start of the next calving season.

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Dietary shortages of ERDP are also common in Kelly, J.M. & Whitaker, D.A. (2000) Multidisciplinary housed suckler cows fed on low quality conserved approach to dairy herd health and productivity management. British Society of Animal Science Occasional Publication, 26: Vol. 1, 209–22.

A dangerous degree of hypomagnesaemia present in

Kelly, J.M., Whitaker, D.A. & Smith, E.J. (1988) A dairy herd health and productivity service. British Veterinary Journal, autumn and winter suckling cows is quite commonly

144, 470–81.

revealed. Follow-up tests can assess the success of

Mansfeld, R., Gruneberg, W., Thiemann, E. & Grunert, E.

supplementation.

(1996) Statistical analysis of metabolic profiles of blood and saliva used as a tool for herd diagnostic procedures.

Züchtungskunde, 68, 325–45.

*Markusfeld, O., Galon, N. & Ezra, E. (1997) Body condition **Summary***

score, health, yield and fertility in dairy cows. Veterinary Record, 141, 67–72.

The metabolic profile approach in dairy cattle should

Opsomer, G., Grohn, Y.T., Hertl, J., Coryn, M. & de Kruif, A.

be used as a way of ‘asking the cows what they think of

(2000) Protein metabolism and the resumption of ovarian

their nutrition’. If the timing of this question is planned

cyclicity post partum in high yielding dairy cows. Repro-

for as soon as possible in each new feeding regime, any

duction in Domestic Animals, Suppl. 6, 54–7.

shortcomings can be quickly identified and corrected

Payne, J.M., Dew, Sally M. & Manston, R. (1970) The use of before major losses in productivity, including fertility,

a metabolic test in dairy herds. *Veterinary Record*, **87**, are inevitable. While the approach may well help in

150–8.

Phillippo, M., Humphries, W.R., Atkinson, T., Henderson, G.D.

problem solving, continued use as a preplanned

& Garthwaite, P.H. (1987) The effect of dietary molybde-

exercise in the DHHPs since 1978 in Britain (Kelly &

num and iron on copper status, puberty, fertility and

Whitaker, 2000) shows that its most valuable asset is as

oestrous cycles in cattle. *Journal of Agricultural Science*, a means of providing more information to cow man-Cambridge, **109**, 321–36.

agers, more quickly and more precisely, than is possible

Schrotter, G., Mostl, E. & Baumgartner, W. (2000) Determi-

by any other means.

nation of bile acid in serum for diagnosis of liver function

in dairy cows. *Wiener Tierärztliche Monatsschrift*, **87**, 94–100.

Sevinc, M. & Aslan, V. (1998) The relationship between par-References

turient paresis and fat cow syndrome in dairy cows. *Turkish Journal of Veterinary & Animal Sciences*, **22**, 23–8.

AFRC (1993) In: *Energy and Protein Requirements of Rumi-*

Suttle, N. (1993) Overestimation of copper deficiency. *Veteri-nants*, pp. 21–31.

An advisory manual prepared by the Agri-nary Record, 133, 123–4.

cultural and Food Research Council Technical Committee

Telfer, S.B., Mackenzie, A.M., Illingworth, D.V. & Jackson, on Responses to Nutrients. CAB International, Wallingford.

D.W. (1996) The use of caerulosplasmin activities and

Bertoni, G. & Cappa, V. (1984) Il profilo metabolico nella plasma copper concentrations as indicators of copper status

vacca produzione di latte. Biochemica Clinica, 8, 131–

in cattle. In: Proceedings of XIX World Buiatrics Congress, 3.

2, 402–4.

Blowey, R. (1975) A practical application of metabolic pro-

Ward, W.R., Murray, R.D., White, R. & Rees, M. (1995) The files. Veterinary Record, 97, 324–7.

use of blood biochemistry for determining the nutritional

Brodison, J.A., Goodall, E.A., Armstrong, J.D., Givens, D.I.,

status of dairy cows. In: Recent Advances in Animal Nutri-

*Gordon, F.J., McMaughey, W.J. & Todd, J.R. (1989) Influen-
tion. (ed. by P.C. Garnsworthy & D.J.A. Cole), pp. 29–51.*

ence of dietary phosphorus on the performance of lactating

Nottingham University Press.

*dairy cattle. Journal of Agricultural Science, Cambridge, West, H.J. (1991)
Evaluation of total serum bile-acid concen-112, 303–11.*

trations for the diagnosis of hepatobiliary disease in cattle.

Domecq, J.J., Skidmore, A.L., Lloyd, J.W. & Kaneene, J.B.

Research in Veterinary Science, 51, 133–40.

(1997) Relationship between body condition scores and

Whitaker, D.A. (1999) Trace elements – the real role in dairy

conception rates at first artificial insemination in a large

cow fertility. Cattle Practice, 7, 239–41.

dairy herd of high yielding Holstein cows. Journal of Dairy Whitaker, D.A.

(2000) Use and interpretation of metabolic

Science, 80, 113–20.

profiles. In: The Health of Dairy Cattle (ed. by A.H.

Garry, F.B., Fettman, M.J., Curtis, C.R. & Smith, J.A. (1994) Andrews), pp. 89–107. Blackwell Science, Oxford.

Serum bile-acid concentrations in dairy cattle with hepatic

Whitaker, D.A., Goodger, W.J., Garcia, M., Perera, B.M.A.O.

lipidosis. Journal of Veterinary Internal Medicine, 8, 432–

& Wittwer, F. (1999) Use of metabolic profiles in dairy cattle 8.

in tropical and subtropical countries on smallholder dairy

Gelfert. C.C. & Staufienbiel, R. (2000) Problems in diagnosis farms. Preventive Veterinary Medicine, 38, 119–31.

of manganese status in dairy herds by herd supervision.

Whitaker, D.A., Kelly, J.M. & Smith, E.J. (1983) Subclinical Tierartzliche Praxis Ausgabe Grosstiere Nutztier, 28, 69–

ketosis and serum beta-hydroxybutyrate levels in dairy

73.

cattle. British Veterinary Journal, 139, 462–3.

Metabolic Profiles • 817

Whitaker, D.A., Smith, E.J. & Kelly, J.M. (1989) Milk produc-
(1966) Bovine functional infertility in Devon

tion, weight changes and blood biochemical measurements

and Cornwall: reponse to manganese therapy. Veterinary

in dairy cattle receiving recombinant bovine somatotrophin.

Record, 76, 562–6.

Veterinary Record, 124, 83–6.

Wittwer, F., Bohmwald, H., Contreras, P., Phil, M. & Filoza, J.

Whitaker, D.A., Smith, E.J., da Rosa, G.O. & Kelly, J.M. (1993) (1987) Análisis
de los resultados de perfiles metabólicos

Some effects of nutrition and management on the fertility

obtenidos en rebaños lecheros en Chile. Archiva Medicina

of dairy cattle. Veterinary Record, 133, 61–4.

Veterinaria, 19, 35–45.

Adult Cattle

System and Miscellaneous Conditions

Chapter 48

Alimentary Conditions

R.G. Eddy

The mouth and associated structures

821

Winter dysentery

852

Salivation

822

Bovine virus diarrhoea/mucosal disease

853

Simple stomatitis

822

Johne's disease (paratuberculosis)

857

Necrotic stomatitis (calf diphtheria)

822

Tenesmus

858

Papular stomatitis

822

Diseases of the rectum and anus

859

Mucosal disease

822

Rectal prolapse

859

Mycotic stomatitis

823

Rectal tears

859

Phlegmonous stomatitis

823

Recto-vaginal fistula

859

Wooden tongue (actinobacillosis)

823

The jaw

824

Fractures

824

The mouth and associated structures

Abscesses of the jaw

824

Retropharyngeal abscess

824

Examination of the mouth, muzzle, mucous mem-

Lumpy jaw (actinomycosis)

824

branes, tongue and teeth should form a part of the

Teeth

825

normal clinical examination. Mouth lesions can be signs

The oesophagus

825

of systemic diseases such as foot-and-mouth disease,

Emesis or vomiting

825

mucosal disease, bluetongue and various poisonings.

Disease of the oesophagus

826

Lesions of the muzzle and lips and occasional conges-

Oesophageal trauma

826

tion of the oral mucosa can be a feature of photosensi-

Oesophageal stenosis

826

Choke

826

tization. Injuries occasionally occur and there are a

Upper alimentary squamous cell carcinoma

828

number of infections and allergic diseases that produce

Diseases of the rumen

828

lesions of the lips, tongue and jaws. Diseases of the

Indigestion

829

gums and teeth are rare in cattle, although chronic flu-

Rumen acidosis

829

orosis (see p. 949) where it occurs will cause mottling,

Rumen parakeratosis

832

discoloration or hypoplasia of the teeth, particularly in

Bloat (rumen tympany)

832

growing animals. Even problems associated with excess

Vagal indigestion

835

tooth wear are uncommon, presumably because the

Cold cow syndrome

836

majority of cattle are slaughtered well before they

The oesophageal groove

837

reach 'old age'.

Traumatic reticulitis

837

The main signs associated with mouth lesions are sali-

Abomasum

839

Displacement of the abomasum to the left

839

vation, excess chewing movements, frequent protrusion

Right-sided abomasal dilatation and torsion

842

of the tongue and licking of the lips and muzzle.

Abomasal ulceration

844

Swellings of either jaw will indicate lesions of the jaw,

Abomasal impaction

844

the most common of which are abscesses, infections of

Abomasal impaction in calves

845

the buccal mucosa, e.g. calf diphtheria, or lumpy jaw

Colic and acute intestinal obstruction

845

(actinomycosis). Submandibular swelling may indicate

Tympanic intestinal colic

846

mouth lesions, e.g. wooden tongue (actinobacillosis), or

Torsion of intestines (red gut in calves)

846

be a sign of cardiac or liver dysfunction, e.g. liver fluke

Prolapse of intestines through mesentery

846

(p. 276) or severe hepatitis (p. 147).

Caecal dilatation and torsion

847

Swelling of the submandibular and retropharyngeal

Intussusception

847

lymph nodes may be the result of infections such

Diaphragmatic hernia

848

Fat necrosis

849

as tuberculosis, actinobacillosis or lymphosarcomata.

Peritonitis

849

Swollen salivary glands do also occasionally occur due

Diarrhoea

850

to tuberculosis infection or infections resulting from

Salmonellosis

850

penetrating wounds.

Salivation

wedged between the mucosa and the teeth; this produces a swelling on the side of the face. Removal of the Ruminants normally produce large quantities of saliva, offending material and treatment with a course of par- which acts as a buffer in the rumen to maintain the enteral antibiotics (penicillin and streptomycin) results normal ruminal pH. Adult cattle normally produce in recovery, although further accumulation of cud 5–10 ml/100 kg body weight/minute. A 600 kg cow can material may occur and this has to be removed daily therefore produce 60 ml/minute of saliva. until the lesion heals.

Excess salivation occurs as a result of many diseases:

The treatment of these conditions is generally

- Foot-and-mouth disease (p. 700) and infectious symptomatic. The foreign bodies are removed, anti-bovine rhinotracheitis (p. 289).

biotics may be administered and corticosteroids or non-

- The various causes of stomatitis, e.g. wooden tongue steroidal anti-inflammatory agents may help to reduce (p. 823), calf diphtheria (p. 250), and teeth-related the painful effects of the inflammation. Most conditions problems (p. 825).

recover quickly when removal of the offending

- Pharyngeal paralysis, although rare in cattle, is a material has occurred.

sign of rabies (p. 908), Aujeszky's disease (p. 907), botulism (p. 721) and occasionally tetanus (p. 733).

Necrotic stomatitis (calf diphtheria)

- Obstruction of the oesophagus, the most common of which is choke (p. 826).

Fusobacterium necrophorus will be associated with

- Eating plants or chemicals that are themselves necrotic lesions of the mouth, tongue and larynx. In irritant and cause inflammation in the mouth calves the condition is extremely common, but it does (Chapter 54).

occasionally occur in growing or adult cattle. A fre-

- Certain chemicals such as copper, lead, mercury and

quent site is the buccal mucosa and this may be a cause
arsenic also stimulate excess saliva production
of decaying cud accumulating between the teeth and
(Chapter 54).

buccal mucosa as described above. The lesions are
necrotic and often contain caseous material. Necrotic
Salivation can be controlled using atropine at a dose
glossitis has been reported in feedlot cattle with the
of 30 mg for adult cattle and can be used 20 minutes
necrotic lesions present on the tongue. The aetiology is
prior to anaesthesia. However, the use of atropine to
unknown but is probably infectious and may be viral.
control excess salivation is not to be recommended or
The signs are of excessive salivation, swollen cheeks
considered necessary. The primary cause of the problem
when the buccal mucosa is affected and a foul smel-
should be diagnosed and corrected.
ling breath. Treatment with parenteral antibiotics or
sulphonamides is generally very effective if given over
a period of three to five days (see p. 250).

Simple stomatitis

Inflammation of the oral mucosa is characterized by

Papular stomatitis

redness and excess salivation. There are a variety of causes, which include the following:

Papular stomatitis is a virus condition producing vesicles that rupture and produce ulcerative lesions. It is a

- *Traumatic injuries from the use of balling and common condition in calves (see p. 252) but is of little drenching guns, mouth gags and stomach tubes may economic significance. It is, however, a differential diagnosis occasionally be encountered.*

nosis of foot-and-mouth disease and lesions caused by

- *Foreign bodies, such as sticks and vegetable roots, mucosal disease, but there are not usually any signs of may damage the roof of the mouth or be wedged systemic disease and there is no excess salivation. between the teeth and the buccal mucosa.*

- *Injuries to the tongue may also occur, for example it may be accidentally amputated by sharp metal.*

Mucosal disease

Stomatitis caused by chemicals is more common.

Erosions or shallow ulcerative lesions of the dental pad, Creosote, discarded engine oil, formalin, acids or caustic the mucosa below the tongue, the roof of the mouth and soda used for forage preservation, chemical dips or occasionally the tongue occur as a sign of mucosal sprays are all found on most livestock farms and, if left disease, which sporadically affects growing cattle unprotected from animals, the inquisitive cow may well around nine to 18 months of age. The lesions are similar consume small quantities, resulting in inflammation to to papular stomatitis, except that vesicles do not the oral mucosa.

precede them. Diarrhoea and wasting are also present.

Inflammation or infection of the buccal mucosa is Mucosal disease and bovine viral diarrhoea (BVD) are occasionally seen. Also, decaying cud may be found described in detail on p. 853.

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Mycotic stomatitis

The organism does not normally invade healthy skin or mucosa but trauma to the mucosal surface from

Mycotic stomatitis caused by Monilia spp. is thought to sharp objects such as sticks, dried thistles, straw or

be a specific disease entity and is characterized by

barley awns will allow entry of the organism. Although

yellow necrotic lesions on the buccal mucosa, which

wooden tongue is the most common clinical manifesta-

erode, coalesce and become covered in a fibrinous

tion of A. lignieresii, the organism will produce lesions necrotic membrane. It is likely that the fungal infection

elsewhere, e.g. the oesophageal groove, the rumen wall,

is secondary and it has been suggested that bluetongue

and other soft tissues of the head and neck. It is fre-

virus (see p. 691) may cause the primary disease.

quently isolated from cervical and pharyngeal lymph

Muzzle lesions similar to mycotic stomatitis also occur

nodes. The organism, once it has gained entry into

occasionally.

tissues, produces small multiple swellings that develop

into small abscesses 2–5 mm in diameter. The sur-

Phlegmonous stomatitis

rounding tissues swell as a result of the inflammation

and the tongue may increase in size by 50 per cent.

Phlegmonous stomatitis and deep-seated cellulitis occur sporadically in adult cattle. It takes the form of an acute, deep-seated, diffuse, rapidly spreading inflammation of the oral mucosa, pharynx and surrounding structures.

Signs

*Wooden tongue has a sudden onset, with excess salivary secretions. It may often follow injury to the mucosa by a foreign body. *Fusobacterium necrophorus*, streptococci and *Escherichia coli* have all been isolated from lesions.*

There is considerable submandibular swelling and frequently swollen submandibular and retropharyngeal lymph nodes. The tongue will frequently protrude from the mouth. The tongue (particularly the dorsum) will be swollen, hard to the touch and on the surface will be seen round, discrete, yellow lesions 2–3 mm in diameter. The onset of signs is sudden, commencing with excessive watery salivation and lacrimation. The rectal temperature is raised to 40.5–41.5°C (105–107°F) and the heart rate and respiratory rate are increased. There is marked swelling of the face, mouth, muzzle and the sub-

mandibular region. The tongue (particularly the dorsum) will be swollen, hard to the touch and on the surface will be seen round, discrete, yellow lesions 2–3 mm in diameter. The onset of signs is sudden, commencing with excessive watery salivation and lacrimation. The rectal temperature is raised to 40.5–41.5°C (105–107°F) and the heart rate and respiratory rate are increased. There is marked swelling of the face, mouth, muzzle and the sub-

5 mm in diameter. These are abscesses situated just mandibular area. There is a foul or fetid smell to the below the tongue epithelium. If left untreated the breath and the oral epithelium frequently peels off. In disease progresses so that eating becomes impossible severe cases death may ensue within 24 hours. and weight loss follows. Eventually death will occur due Treatment is successful if commenced early and to starvation.

the condition usually responds to sulphonamides or a course of injections with penicillin and streptomycin.

Treatment

Wooden tongue (actinobacillosis)

If initiated early in the course of the disease, treatment is generally successful. However, if treatment is delayed

Wooden tongue is a well-defined disease producing beyond two weeks it is less likely to be so. Traditionally, stomatitis and glossitis, mainly in adult cattle. The causal treatment consisted of intravenous sodium iodide 7

agent is *Actinobacillus lignieresii*. The disease is found g/100 kg body weight administered as a 10 per cent solu-worldwide but the incidence varies considerably

tion and repeated in 10–14 days. Oral treatment with
between countries. In the UK the incidence of the
sodium or potassium iodide is also effective.

disease has declined considerably in recent years;

Iodine treatment should continue until signs of
whereas 25 years ago the annual incidence was of the
iodism occur, such as dry scaly skin rather similar to
order of 20–30 per 10 000 cattle it has declined to less
dandruff. Sodium iodide should not be administered
than 5 per 10 000.

to heavily pregnant animals because of the risk of
abortion (see pp. 261, 302).

Antibiotics are effective. Streptomycin at a dose rate

Aetiology and pathogenesis

of 10–15 mg/kg daily given intramuscularly for 10 days

The aerobic, Gram-negative coccobacillus *A. lignieresii*

is effective in most cases. However, penicillin is less

is the causative organism and it produces small

satisfactory. Some workers advocate the streptomycin

abscesses commonly referred to as ‘sulphur granules’ in

should be injected into the lesion. Besides being

soft tissues. The bacteria do not survive outside the animal host for longer than five days but have frequently been isolated from the faeces of normal healthy animals. It is considered to be a normal inhabitant of the upper respiratory and alimentary tracts.

Animals that have been affected and untreated for two weeks or more and animals that do not recover within two weeks of treatment should be slaughtered.

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Prevention

Abscesses of the jaw

In most situations the disease of wooden tongue is sporadic. However, herd outbreaks have been known to occur, which may be the result of feeding hay or straw containing thistles, gorse or brambles. In any case the size varies but frequently they are about the size of a tennis ball (7.5 cm or 3 inch diameter).

affected cattle should be isolated to prevent spread of
Diagnosis is usually confirmed by paracentesis. Treatment
the infection and feeds such as those described above
ment by incising into the abscess, removing the pus
should not be offered. The reduction in UK incidence
and flushing with clean water should only be attempted
in recent years may well be related to a sharp decrease
if a soft area is apparent on the abscess surface. If palpation
in the use of hay and a corresponding increase in silage
pation of the abscess reveals the external surface to be
as a method of grass conservation.

hard all over, treatment should be delayed until a softening
and pitting of one area is noticeable. This is called
pointing of the abscess. Abscesses that are opened and

The jaw

drained prematurely have a tendency to recur.

A number of conditions affect the jaw of adult cattle,

Retropharyngeal abscess

usually the lower jaw or mandible. Fractures of the
Abscesses of the retropharyngeal lymph nodes occur
symphysis (annual incidence 1 per 10 000) are a result of

occasionally. These are presented as discrete round swellings the size of a tennis ball (7.5 cm or 3 inch diameter) on a hard surface. Fractures of the mandible (1-2 cm) behind the vertical ramus of the mandible. The usually result from accidental collision with a farm vehicle or occasionally a pathological fracture due to entering a pharyngeal wound. Tuberculosis needs to be considered in countries where it is present. The treatment is the same as for jaw abscesses. There are a few cases of lumpy jaw (actinomycosis) or neoplasia. Jaw abscesses are seen in calves and growing cattle. In calves these are the sequel to untreated calf diphtheria but in growing cattle they may be caused by infections being introduced once encountered such a round hard swelling the size of a tennis ball in a Friesian cow. As the swelling was hard defined infection of the mandible, but only occurs rarely. However, the author with no defined softening the farmer was advised to wait

three weeks, by which time lancing the abscess should be

Fractures

possible. Three weeks later, as no softening had occurred and the cow was experiencing some difficulty with swal-

lowing, the animal was anaesthetized and the pharynx explored, where a tennis ball was discovered!

stained, of the lower lip will be noticed and some excess salivation. The animal continues to eat, but with

Lumpy jaw (actinomycosis)

obvious difficulty.

Examination of the jaw will reveal bruising of the

Lumpy jaw is a chronic infectious disease characterized lower lip and excess mobility of either side of the

by suppurative granulation of the bones of the head, mandible. The front teeth may or may not be displaced, particularly the mandible and maxilla. There is gross loose or even lost.

swelling, abscesses are present, fistulous tracts and

These animals will recover in about three weeks,

extensive fibrosis all contribute to the granulomatous lesion. Its occurrence is quite rare in the UK (1–2 per 100 000 annual incidence) but is said to be more common in the western and mid-western states of the USA. However, it is found at varying incidence levels worldwide where grazing cattle exist.

provided they are isolated from the herd and food is cut and brought to the animal. Grazing or feeding from self-feed silage faces will prove difficult for affected animals. Abnormal positioning of the front teeth may be present after healing has completed.

Trauma-induced fractures of the mandible may be

Aetiology and pathogenesis

more difficult to manage. If there is no obvious displacement of bone at the fracture site and no dislocation, The causative organism is *Actinomyces bovis*, a Gram-positive filamentous anaerobe, which is a normal inhabitant of the mucous membranes of the oral cavity, upper placement at the fracture site or dislocation is present,

respiratory tract and digestive tract of most animals.

casualty slaughter is usually the wisest course of action.

The organism gains access to the soft tissues as a result

Pathological fractures due to infection will not heal

of mucosal damage caused by sharp objects or erupting

and immediate slaughter should be advised.

teeth. It generally affects cattle two to five years old.

Alimentary Conditions • 825

The organism causes a low-grade inflammatory reac-

Jaw abscesses may result from infection gaining

tion. There follows a proliferation of connective tissue,

access during tooth eruption.

invasion with leucocytes and the resulting formation of

Occasionally, heifers will experience problems with

a walled tumour-like mass. The granuloma then invades

feed when teeth are erupting. This will be particularly

the bones of the mandible or occasionally the maxilla.

noticeable if the forage is in the form of self-feed silage

The hard, immovable, circumscribed lesion that results

and the clamp well compacted.

may reach a considerable size (15–25 cm in diameter)

Foreign bodies do occasionally become lodged and will eventually interfere with mastication. The space between the teeth or between the teeth and the buccal mucosa. development of the lesion takes several weeks. Inter-

*connecting abscesses and fistulae breaking to the exterior and discharging small quantities of pus are a frequent sequel. The pus may contain yellow granules that, on compression and staining, will reveal the Gram-
Anatomy*

Cattle have both temporary and permanent dentition. positive filaments of A. bovis. The granulomatous lesion The upper front teeth are absent and the premaxilla is

will continue to invade the soft tissues of the head and attached by a layer of fibrous tissue covered by a thick

neck and the teeth are frequently displaced or become horny epithelium. There are 20 deciduous teeth with a dislodged and the lower jaw may be displaced laterally.

dental formula of:

Pathological fracture of the mandible may occur (see p.

0

3

0

824).

I-

C-

P-

M-

¥ 2 = 20

3

1

3

0

Pathology

In the permanent dentition the three pairs of pre-

*Rarefying osteitis, osteoporosis interspersed with gran-
molars in each jaw are supplemented by three pairs of
ulomatous tissue and pockets of thin pus-containing,
upper and lower molars:*

yellow, sand-like granules are the main pathological

0

0

3

3

changes found in this condition. The soft tissues of the

I–

C–

P–

M–

$\text{¥ } 2 = 32$

head, the oesophagus and oesophageal groove may also

3

1

3

3

be involved. The lymph nodes are not involved.

The front teeth, three incisors and one canine are all spatulate and arranged in a broad arch. The fixation

Diagnosis

of the teeth allows some dorsoventral movement

Diagnosis is straightforward and based on the clinical within the alveoli. There is a large gap or diastema

signs. Confirmation of diagnosis is made by staining the separating the front teeth from those of the cheek. The crushed yellow granules found in the pus and demon- cheek teeth increase in size caudally and the lower pre- strating the Gram-positive filamentous rods.

molars and molars are narrower than the upper ones.

The age of tooth eruption varies between *Bos taurus* and *Bos indicus* and also depends on breed, sex and state Treatment of nutrition. Examination of the permanent incisors is Treatment with iodides as recommended for actino- frequently used to give an indication of age.

bacillosis is sometimes advocated (see p. 823). Injecting

The deciduous teeth have little use in age determi- the swelling with penicillin is also occasionally recom- nation. Calves are frequently born with fully erupted mended; however, because of the nature of lesions such incisors and all will be in place by one month of age.

treatment is unsatisfactory. Intramuscular injection of The average age for tooth eruption in European-type penicillin may assist, but once better casualty slaughter cattle is shown in Table 48.1. However, the standard

should be advised as lesions are likely to recur.

deviation is approximately 10 per cent of the average age. Thus for a given age of development, e.g. 2.5 years, 95 per cent of the population will be 2.5 years \pm 0.5

Teeth

years, i.e. 2–3 years.

Dental problems are rare in cattle. Fractures of the mandibular teeth occur occasionally and traumatic in-

The oesophagus

juries can result in incisor teeth being displaced or lost.

Excessive wear of the incisors indicates advancing age.

Emesis or vomiting (see p. 139)

Discoloration of the teeth may result from prolonged treatments with oxytetracyclines during the teeth devel-

Emesis (vomiting) is not a common sign in ruminants.

opment phase (see p. 1043). Discoloration and pitted

Reverse peristalsis of the oesophagus is a normal

enamel will occur as a sign of fluorosis (see p. 949).

physiological process in rumination.

Average age for tooth eruption in European-type

Oesophageal trauma

cattle (Bos taurus).

The usual cause of lacerations to the oesophagus is the

Tooth

Deciduous

Permanent

*result of careless use of a probang, stomach tube, or
bolusing, which can sometimes cause rupture. As the*

1st incisor

Before birth

22 months

probang is rarely used in cattle practice nowadays

2nd incisor

Before birth

27 months

this problem is uncommon. If rupture of the oesopha-

3rd incisor

Before birth to 2 weeks

37 Months

gus is suspected, then immediate slaughter should be

Canine

Before birth to 2 weeks

44 months

recommended.

1st premolar

Birth to 3 weeks

27 months

2nd premolar

Birth to 3 weeks

24 months

3rd premolar

Birth to 3 weeks

33 months

Oesophageal stenosis

1st molar

—

6 months

Stenosis due to pressure outside the oesophagus can be

2nd molar

—

12 months

caused by gross swelling of the mediastinal lymph

3rd molar

—

24 months

nodes. This used to be a problem associated with tuberculosis, but more recently it has been reported to be

associated with lymphomatosis. Oesophageal stenosis caused by swollen mediastinal lymph nodes resulting in

However, vomiting (when rumen contents are

*chronic bloat in calves three to six months old is some-
ejected from the mouth) does occasionally occur in acti-
times reported in calves recovering from pneumonia.*

*nobacillosis of the oesophageal groove or with painful
conditions of the mouth caused by teeth erupting. The*

Choke

*ejection of rumen contents is also frequently seen in
the terminal stages of milk fever, the result of*

Oesophageal obstruction or choke is a relatively

rumen pressure caused by tympany accompanying

common problem in cattle. As cattle consume food

oesophageal relaxation that occurs with hypocalcaemia,

rapidly, incomplete mastication is normal. Regurgitation not by reverse peristalsis. Grain overload (rumen acidosis), traumatic reticulitis, diaphragmatic hernia, vagal ensure food is ground into small particles. Apples, potato indigestion and abomasal impaction have all been tubers or portions of root vegetables, such as turnips, reported to produce emesis, but in the author's experience vomiting is not normally associated with these relatively large pieces, often without any mastication. The conditions. Emesis as a herd problem has been reported occurrence of choke therefore mirrors the fashions in to occur following the consumption of spoiled maize feeding cattle. When potatoes are in excess and in silage. In the UK poisoning with azalea or rhododendron species (see p. 943) is probably the most common and turnips were widely used choke was common. The cause of vomiting in cattle, although poisoning by lily presentation of the root crops also affects the occurrence

of the valley (*Convallaria majalis*) and sneezeweed of choke. If large roots such as fodder beet or mangolds (*Helenium loopesii*) has been reported to cause are fed whole then choke is uncommon, but if chopped vomiting in cattle in the USA.

they will attempt to swallow without mastication pieces of root that are too big to pass down the oesophagus.

Strip-grazing root crops, such as stubble turnips, also

Diseases of the oesophagus

rarely results in choke. Presumably cattle bite relatively

Disorders of the oesophagus are relatively rare.

small pieces at a time. This is probably why the most

Mucosal disease/BVD does produce shallow ulcerative

common causes of choke are apples and potatoes, which

lesions of the oesophagus (see p. 853). Malignant

are the right size to obstruct the oesophagus, are difficult

catarrh (see p. 935) also produces characteristic lesions

to masticate being round and slippery and are rarely

of the oesophagus and occasionally oesophageal lesions

chopped before being fed. If potatoes are being fed,

may be found in calves suffering from calf diphtheria

ample bunker space is essential for all cows to eat simultaneously (see pp. 250, 822). Primary conditions of the oesophagus are:

the feed bunker may cause the cow eating inadvertently to swallow a whole potato it might otherwise chew.

- *Traumatic lacerations*
- *Stenosis caused by pressure from outside the*

Signs

oesophagus

- *Obstruction or choke*

Profuse salivation, followed by bloat, are the principal

- *Dilatation (see p. 139)*

signs of choke because the obstructed oesophagus pre-

Alimentary Conditions • 827

vents swallowing of saliva and eructation of rumen gas.

is in distress because of bloat, this is relieved by insert-

Affected cows will look distressed, the head is extended forwards and frequent attempts to swallow are often using a standard bovine trochar and cannula.

evident. Excess chewing movements are frequently
If the obstruction is in the cervical oesophagus a
observed and coughing, due to saliva accumulation in
spasmolytic such as hyoscine N-butylbromide and dipy-
the pharynx, may occur.

rone or butylscopolamine bromide and metamizole is
The extent of the rumen tympany is related to the
administered by intravenous injection and five minutes
length of time choke has been present and the nature
allowed to elapse before attempting to move the
of the object. A round object such as an apple or potato
obstruction. The spasmolytic will relax the smooth
will completely obstruct the oesophagus and death
muscle of the oesophagus, which is in spasm and con-
from bloat may be a sequel. If the offending object is
traced firmly around the obstruction. Five minutes
irregularly shaped, such as a root or a portion of a
after the spasmolytic has been administered the opera-
potato, some gas may escape past the obstruction and
tor, using his fingers in the jugular groove with an
death from bloat will be avoided.

assistant holding the head forward, gradually pushes the obstruction up the oesophagus until it reaches the

Diagnosis

laryngeal area. Frequently, when at this position and the spasmodic has taken effect, upward pressure with

Diagnosis is usually made on the basis of the signs of the fingers will cause the object to be ejected into the excess salivation, rumen tympany, forward extensions mouth. If this does not happen then a mouth gag has to of the head and the history of the availability of offend- be applied to keep the mouth open and a second assis- ing feeds. The obstruction is usually at one of three sites: tant places his forearm into the mouth and pharynx

- Alongside the larynx in the upper oesophagus

and, simultaneously with the operator pushing the

- At the entrance to the chest

object upwards, will grasp it and be able to remove it.

- In the thoracic portion of the oesophagus.

If the object is large and appears reluctant to move, this latter procedure can be carried out under anaesthesia.

The cervical sites are more commonly involved than the

It is the author's experience that anaesthesia has not thoracic oesophagus and occasionally the obstruction been required since spasmolytics have been available. will be between the laryngeal area and the entrance to If the obstruction is in the thoracic oesophagus this the chest.

will have been detected by the use of a stomach tube.

Usually the obstruction can be seen and palpated as a

The application of gentle pressure with the stomach tube

discrete swelling, although if it is present alongside the

will dislodge the obstruction and push it into the rumen.

larynx confusion with the thyroid cartilage may occur. If

Again spasmolytic administration will enable the

in doubt or an obstruction in the thoracic oesophagus is

obstruction to be more readily moved. On occasions the

suspected the passage of a stomach tube will aid the diag-

obstruction will move on into the rumen without pres-

nosis. If the stomach tube reaches the rumen and the

sure following the spasmolytic injection. If the efforts

tympany is relieved, an obstruction, if it existed, will

described do not remove a thoracic oesophageal

probably have been dislodged. The differential diagnosis, a probang may be used. However, care must be exercised when using the probang, as the pressure and tympany may be mistaken for bloat. Bronchitis, applied must not be too severe for fear of lacerating the oesophagus. If the probang fails to dislodge the obstruction should not be confused with choke. Once the obstruction has been located, either by palpation or by stomach removed from the oesophagus manually via the rumen. In preference to a rumenotomy and if it is certain that (p. 908). Excess salivation is the only sign both rabies and the obstruction is caused by vegetable matter, a rumen choke have in common so the possibility of rabies must be eliminated by assessing other signs before proceeding along the diagnostic pathway to choke.

macerate. This may take two to four days but is usually effective.

Treatment

Before treatment commences, the animal will need to

Prevention

be restrained in a cattle crush or at least tied in a byre stanchion. A bulldog-type nose handler is used by an
Choke can be prevented by the shredding or chopping
assistant further to restrain the animal and keep the
of root vegetables into small pieces that will not cause
head from moving. The first element of treatment is to
obstruction. If potatoes are fed, ample feeding space is
assess the severity of the rumen tympany. If the animal
required and if they are offered at pasture they should
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be placed in the field before the cows enter. Cows will
Ruminal tympany syndrome: Carcinoma of the reticu-
follow a vehicle that is unloading food and will attempt
loruminal cardia or thoracic oesophagus will produce
to eat while on the move. If they attempt to eat pota-
intermittent ruminal tympany following a period of one

toes or apples while on the move they are more likely to six months of gradual weight loss. The passage of a to choke. It is rare for apples to be intentionally fed to stomach tube will prove difficult. Papillomata of the cattle. However, when cattle graze orchards, windfall mouth and pharynx will usually be present and profuse apples should be collected before allowing cattle access. diarrhoea is a common feature.

This will not only prevent choke but also rumen acidosis (see p. 829) from overeating apples.

Wasting and diarrhoea syndrome: If the carcinoma is present in the dorsal pigmented area of the rumen symptoms of rumen indigestion will be present. The tumours

Upper alimentary squamous

in the rumen can reach 30–50 cm (12–20 inches) in

cell carcinoma (see also p. 946)

diameter. A slow loss of weight over up to nine months

Occurrence

is followed by diarrhoea, which contains much

fibrous undigested material. Ruminal tympany is

Squamous cell carcinoma of the upper alimentary tract

usually present and the abdomen is often pendulous.

occurs sporadically in cattle grazing bracken areas.

Papillomata are usually found in the mouth or pharynx.

The problem usually affects beef cows and has been reported in Scotland and North Wales. It occurs on

Diagnosis

farms with a high bracken cover but is less common than another bracken-induced disease, enzootic haema-

The diagnosis may at first be difficult, as the signs may turia (see p. 947). Both conditions have been reported

resemble choke (p. 826) or rumen indigestion (see p.

in the same animal. The disease generally occurs in

829). However, the history of the slow weight loss, the

animals over six years old. Almost invariably alimen-

presence of bracken and the occurrence in older beef

tary papillomata, in which papilloma virus can be

cows will considerably aid the diagnosis. As papillomata

demonstrated, occur in animals affected with bracken-

are almost invariably found in the mouth or pharynx of

induced squamous cell carcinoma. It is thought there-

affected animals a thorough examination of the mouth

fore that the disease is caused by a toxic factor present should be made if the other signs are present. Johne's in bracken that activates papillomata in the alimentary disease (p. 857) may also be confused with this condition tract to develop into squamous cell carcinomas.

but the distinguishing feature is the consistency of the faeces. In Johne's disease the faeces are homogeneous,

Signs

there being no signs of undigested fibrous material.

The clinical signs can be divided into four main syndromes that are related to the site of the carcinoma,

Treatment and prevention

which can occur in the pharynx, oesophagus or rumen.

There is no treatment and as the condition is invariably fatal affected animals should be slaughtered as soon as

Oropharyngeal syndrome: The presence of the carcinoma possible.

noma in the oropharyngeal region will produce a chronic

To prevent the condition, cattle should not have access

wasting condition of one to six months' duration. A

to bracken. Where bracken exists on a farm cows will

*cough may be present in the last two to four weeks of the
rarely eat the bracken if adequate food is available.*

*disease and excess salivation and drooling will often be
evident. Snoring may also be a feature and halitosis is
frequent. Examination will reveal a tumour in the mouth*

Diseases of the rumen

*or pharynx. Papillomas will also be present. The sub-
mandibular lymph nodes may be enlarged.*

*There are a number of diseases that affect the rumen,
all of which cause inappetence, reduced milk yield and
Oesophageal syndrome: If present in the oesophagus
failure to thrive. An essential element in a thorough
the carcinoma will produce signs similar to choke
clinical examination of the bovine is to observe whether
with partial or complete occlusion of the oesophagus.
the rumen is tympanic by observing distention of the
Halitosis, coughing and drooling may be present and
left sublumbar fossa, the animal is chewing its cud,
gurgling sounds may be heard coming from the
eructating normally and normal rumen contractions are
oesophagus. The passage of a stomach tube may prove*

taking place. Rumen contractions can be detected by difficult. The history will include a gradual weight loss palpating with slight pressure in the left sublumbar over a period of one to three months. Papillomata may fossa and detecting the rumen movements, which occur be present in the mouth or pharynx. A palpable mass approximately every 30 seconds. Eructation will accompany every second rumen contraction.

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Indigestion

Diagnosis

Simple indigestion is to be suspected if the rumen contractions are weaker than normal, are less frequent and the clinical examination has revealed no other evidence of disease. There is very little in the way of laboratory tests of disease.

that can be useful. An increase or decrease in rumen pH

Simple indigestion is frequently seen in dairy cows

may be a useful guide but tends not to be used in practice when new feeds are suddenly introduced to the diet. Intakes of large quantities of very wet grass, frosted the most useful clinical indicator and may suggest a 'feed problem'. If only one cow in the herd is affected the contaminated silage, or the sudden introduction of large quantities of concentrate feeds may all predispose to the problem'. If only one cow in the herd is affected the history may not be helpful and the diagnosis more difficult. However, if the whole herd or a substantial part of the herd were suddenly affected with inappetence it would be quite logical to suspect a 'feed problem'. will also precipitate indigestion, as is seen in feedlot beef cattle when rations containing monensin sodium are first introduced. Dairy cow concentrate rations containing as reticulitis, lesions of the oesophageal groove, early milk

little as 0.005 mg/kg dry matter (DM) of lincomycin have fever and acetonaemia. A thorough clinical examination will eliminate these other diseases.

dairy herds with a consequent 50 per cent reduction in the herd milk production. The lincomycin contamination

Treatment

tion occurred at the compound mill where the milling

Many animals suffering from indigestion will recover

equipment was used to produce dairy concentrate

spontaneously in two to three days if they are removed

immediately following the production of a pig feed con-

from the offending diet and allowed access to good

taining lincomycin.

quality hay.

Relatively minor changes in rumen pH are likely to

A large number of treatments for indigestion have

cause atony of the rumen. The intake of any indigestible,

been employed and were administered as drenches.

stale or sour feeds will interrupt the process of rumen

Historically mixtures containing nux vomica, gentian,

fermentation, which changes the pH resulting in rumen sodium bicarbonate, magnesium carbonate, etc. were atony. The sudden introduction of new feeds to which frequently used and acted by altering the rumen pH and the rumen is unaccustomed will have a similar effect. stimulating appetite and even providing vitamins and The rumen is a versatile fermentation chamber and trace elements essential for rumen microbial synthesis. will accommodate a wide, variety of feeds, e.g. 100 per However regulations on the use of licensed medicines cent cereal diets in feedlot beef production, extremely in food animals have resulted in all such preparations wet grass in some seasons and highly fibrous foods such being withdrawn by the manufacturers. as hay or straw. However, if changes to the diet are made If the rumen is atonic for more than 24 hours the the rumen flora have to adapt to the new feed and to administration of rumen inoculum obtained either from prevent indigestion occurring, the changes should be an abattoir or a healthy cow will aid recovery. Probi- gradual. Thus new food should be introduced in small

otics may also be helpful.

quantities at first, with the intake being gradually increased daily.

Prevention

The prevention of indigestion is not always straightfor-

Signs

ward. The avoidance of abrupt changes in feeds and of indigestible, sour or putrefied feeds is relatively easy.

The first clinical sign of simple indigestion will be

However, cows will break out into pastures where they

reduced appetite and in dairy cattle a simultaneous

should not be and will, if given the opportunity,

milk yield reduction will be apparent. The reduction in

consume foods of dubious quality in large enough

feed intake may be as much as 50 per cent with no other

quantities to be harmful. The frequency of occurrence

obvious signs. The cow may be slightly depressed.

of simple indigestion on a farm is likely to be inversely

Rectal temperature will be normal and heart rate raised

related to the quality of management that exists.

slightly to around 80/minute. There may be mild diar-

rhoea and a mild rumen tympany may be present. In

Rumen acidosis (grain overload,

chronic cases of indigestion undigested fibre may be

overeating syndrome, barley poisoning)

present in the faeces. Rumen contractions will be

reduced in strength and frequency, often to as few as

Wherever intensive livestock production is practised

one contraction every 3 or 4 minutes.

acute indigestion as a result of excessive intakes of

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grain, beans or compound feed is common, either as a

Chemical damage to the rumen mucosal epithelium

result of cattle gaining accidental access to grain stores

results in bacterial and fungal organisms penetrating

or by the sudden introduction of unlimited supplies of

the rumen wall and in chronic acidosis will lead to the

grain to the diet. There are two distinct syndromes asso-

occurrence of liver abscesses, which are frequently seen

ciated with overeating.

in beef cattle raised on 100 per cent cereal diets.

The most common is acute acidosis as the result of

Urine output will be decreased as a result of the consuming excess carbohydrate, which rapidly ferments dehydration and will also be acidic, containing high levels of lactate. The damage to the rumen epithelium and depression. Accidental access to compound feed leads to a chronic rumenitis, which in turn causes an increased incidence of bloat. This is often seen in feed-concentrate, is the most common scenario but the lots as a cause of sudden death. Although the main signs problem is also seen in feedlot beef cattle fed *ad libitum* of acidosis are the result of lactate absorption an addi-cereal-based diets, particularly when the diet is first tional component may be the absorption of endotoxins introduced. Excess intake of apples by cattle grazing released from the destruction of large numbers of orchards is also a common cause of rumen acidosis.

Gram-negative bacteria in the rumen.

Alkalosis: A less common syndrome occurs as a result

Signs

of excessive intake of highly fermentable proteinaceous

feeds, e.g. soya bean, which results in excess ammonia

The speed of onset and the severity of clinical signs will

production in the rumen leading to alkalosis with

depend on the quantity and nature of the food consumed

excitement and hyperaesthesia.

and whether the rumen is adapted to that particular

feed. Newly introduced feeds may well prove fatal to

animals that are not accustomed to the particular feed

Pathogenesis

in quantities that other animals consume regularly. Also,

The rumen can be visualized as a continuous culture fer-

mentation vat. The rumen microflora constitute the

diets will sometimes overeat for no apparent reason.

culture, which grows on the substrate or medium being

Feedlot cattle being fed cereal diets are probably in a

provided by the feed the animal consumes. The rumen

continuous state of mild acidosis and a relatively small

microflora form a balanced colony of bacteria and

increase in intake will produce enough lactic acid to

various protozoa. If the feed content changes the

destroy the remaining Gram-negative bacteria and balance of the microflora will need to change, e.g. produce clinical signs of acidosis. Problems also arise increase in intakes of highly fermentable carbohydrate should cattle on ad libitum feed run out of cereal and will lead to streptococci and lactobacilli organisms pre-then are immediately introduced to the old level. Water

dominating. With a normal balanced microflora the fermentation end products are the volatile fatty acids deprivation can also produce a problem.

mentation end products are the volatile fatty acids Clinical signs usually become apparent 12–36 hours (acetic, propionic and butyric acids), which are absorbed after the engorgement. Incoordination and ataxia are from the rumen. Bacterial cell proteins for digestion and the first signs to be noticed with the stockworker report-absorption in the lower intestine and water-soluble ing the cattle as 'drunk'. The animals will be anorexic, vitamins are also products of the fermentation process. they may appear to be blind and will rapidly become The streptococci and lactobacilli organisms that weak and depressed. The rumen will be distended predominate when excess carbohydrate is ingested

producing abdominal pain, which causes the animal to produce lactic acid as a fermentation end product. The animal may stop to grunt or grind its teeth and ruminal movements cease. Lactic acid production increases the rumen osmotic pressure. Dehydration becomes apparent within 24–48 hours and pressure and fluid is drawn into the rumen from body tissues. Severe diarrhoea will be evident in animals that do not die immediately. The rumen pH also drops and the majority of Gram-negative bacteria and protozoa are destroyed. The faeces will be a pale 'pasty' colour. Respiratory rate will be raised because of the acidosis. The pH may drop to 4.5. The lactic acid is converted to sodium lactate, which is absorbed directly from the rumen into the blood or is passed down the intestinal tract and absorbed from the abomasum or small intestine. The pulse weak. Severe cases will become recumbent with the head resting on the shoulder, as in milk fever. Death will occur within 24–48 hours in acute cases.

intestine produces an osmotic gradient and draws water

*In feedlot cattle that recover some animals fail to
into the small intestine, thus contributing to the diar-
thrive even after apparent recovery and this can be
rhoea. The sodium lactate in the blood reduces blood
due to chronic rumenitis, liver abscesses, or chronic
pH.*

laminitis (p. 420).

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Alkalosis: The signs are not completely typical but

Table 48.2

McSherry's solution.

*they do involve muscle tremors, convulsions and slow
shallow respiration. In the later stages there may be*

Sodium chloride

4.95 g

dyspnoea and hyperpnoea.

Sodium acetate

7.50 g

Potassium chloride

0.75 g

Calcium chloride

0.30 g

Clinical pathology

Magnesium chloride

0.30 g

Affected animals are dehydrated and show haemocon-

Water

1 litre

centration. Blood pH may also be depressed. The pH of rumen contents will be 4 or lower and urine pH around 5.

However, in practice, laboratory tests are rarely performed because of the necessity to institute treatment quickly and the diagnosis is rarely a problem. In per cent sodium bicarbonate solution intravenously. Up to 60 l of electrolyte solution will be required for an adult cow over a 24-hour period. Up to 300 ml of 5 per cent

dioxide levels need also to be measured.

At post mortem of acute cases the rumen will be dis-

sodium bicarbonate solution may also be administered, tended, containing excess fluid with evidence of grain the quantity required depending on the degree of acidosis particles present. Rumen pH evaluation at post mortem is of no value unless done immediately after death down of the respiratory rate during its administration, if because post-mortem changes will increase the pH. given slowly. Supportive therapy should include intravenous calcium borogluconate (400 ml of 30 per cent may be characterized by rumenitis, although to observe solution) and 400 ml of 40 per cent dextrose solution. this the examination must be performed soon after For less acute cases that are still standing, the oral death. Multiple liver abscesses may also be present in administration of magnesium hydroxide, magnesium carbonate or aluminium hydroxide at a dose rate of 1 g/kg body weight mixed in 10 l of warm water and Alkalosis: If the less common syndrome of alkalosis is administered by stomach

tube will help restore rumen

present due to the ingestion of excess soya beans or pH. The dehydration and acidosis is corrected using similar high-protein feeds, the rumen pH may be alkaline and the urine and blood pH will also be raised. On occasions there is a paradoxical acidic urine.

tion of a vitamin B/vitamin C mixture is widely used in the UK with apparent success. Doses of 30–50 ml intra-

Diagnosis

venously are frequently used and are thought to aid detoxification of lactate in the liver.

Diagnosis is not usually difficult and is frequently made Antihistamines are also considered to be of value by on the history together with signs of incoordination and some clinicians. Calcium borogluconate and 40 per cent ataxia. In feedlot beef animals it is usual for several of dextrose are also useful as supportive therapy when the group to be affected, but with dairy cattle any administered intravenously.

number may be affected, depending on how many cows had accidental access to the grain store, which is the most common scenario. Cows grazing orchards will bean or high-protein engorgement should consist of an consume large quantities of apples, particularly after a electrolyte mixture containing excess chloride such as storm, and again the history should be of considerable Ringer's solution. Volumes of 30–50 l will be required help in reaching a diagnosis.

over a 24-hour period, but no bicarbonate should be Laboratory tests are not normally necessary and used. All concentrate feeds should be withdrawn from unless a result can be available within an hour or so are the diet and only hay or silage offered.

of no value in the practice situation because to be effective, treatment must be administered as quickly as possible.

Prevention

sible. The prognosis for peracute recumbent cases is very poor, but for the less acute standing cases the prognosis

Bulk storage of concentrates in large bins has reduced

*is relatively good provided treatment is not delayed.
the incidence of acidosis in many dairy herds where
accidental access of feed stores with concentrate stored
in bags was once quite common.*

Treatment

In feedlots where concentrated food is fed ad libitum, In peracute recumbent cases the rumen should be

*often to the exclusion of long fibre, introduction to the
emptied by rumenotomy (pp. 834, 1106) and the dehy-
concentrate must be gradual and the feed bunkers*

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*should be of adequate size to allow all animals to feed
ticularly towards the end of the feeding period. Com-
together. The feeders should not be allowed to empty
plications such as peritonitis, septicaemia or even endo-
to avoid excessive intake by hungry animals once the
carditis may occasionally be evident.*

*feeders are refilled. Continuous access to the food will
allow 'little and often' intakes of food. Ideally, some*

Diagnosis

roughage should always be available even if it only rep-

The diagnosis in the live animal is extremely difficult. It represents 10 per cent of the DM intake. Roughage availability because the reduced growth rate and inappetance are. It has been shown to reduce the incidence of liver abscesses in cereal-produced beef in the UK. It is also advisable that the grains should not be finely ground, at post mortem will be the best diagnostic indicator. which in itself encourages rapid fermentation. The grain should be cracked or rolled and more recently whole

Treatment and prevention

grain has been fed without seriously affecting the digestion of the cereal.

The treatment of individual cases by the time they show inappetance and poor growth rates is unrewarding

Rumen parakeratosis

because of the severity of the damage to the rumen wall and liver. However, if the problem is suspected (by the Parakeratosis of the rumen epithelium is a common post-mortem examination of some animals from the

sequel to the feeding of 100 per cent concentrate diets same farm at the abattoir) feeding 10–20 per cent of the to cattle. The disease is characterized by enlargement diet in the form of long roughage will prevent deterioration and hardening of the rumen mucosa papillae and may ration and allow the animals to reach slaughter weight. affect 100 per cent of animals reared on 100 per cent Prevention depends on the inclusion of 10 per cent long cereal diets. The disease syndrome also includes the fibre in the diet, although recent experience has shown associated lesions of liver abscesses and possibly laminitis that feeding whole grains will reduce if not eliminate tis. Parakeratosis may occur as a secondary stage of the problem of rumen parakeratosis and liver abscesses. acute rumen acidosis.

Bloat (rumen tympany) (see also p. 234)

Pathogenesis

Bloat or rumen tympany is a disease easily recognized The association between liver abscesses, lesions of the and feared by cattle farmers. Bloat refers to an excess-rumen mucosa and the feeding of 100 per cent concen-

sive accumulation of gas in the rumen and, because concentrate diets to ruminants has long been recognized. The of a failure to eructate, rumen distension occurs, frequently resulting in death. It is a major cause of death the consequent increase in lactic acid production produces an inflammatory reaction of the rumen mucosa. There are two types of bloat, gaseous bloat or secondary rumen tympany and frothy bloat or primary rumen tympany. This damage to the mucosa allows debris to adhere to the mucosa causing ulceration and infection and resulting in abscess formation in the rumen wall. The rumen papillae become enlarged and thickened and may clump together to form bundles in response to the inflammation that causes an oesophageal obstruction or that interferes with eructation will produce gaseous bloat. of keratinized epithelial cells, particles of food and bac-

The condition is generally sporadic in occurrence and teria. A sequel to the damage to the rumen mucosa is the is less common than frothy bloat. The following conditions will lead to gaseous bloat:

that laminitis is a later sequel but, as laminitis has been found in the absence of rumen and liver lesions, the role

- *Lesions of the oesophageal groove, e.g. vagus indigestion, abscessation or infection with A. lignieresii of acidosis in the aetiology of laminitis is far from clear. obstruct the groove and prevent eructation (pp. 826, 837).*

Signs

- *Physical obstruction of the groove with afterbirth Rumen parakeratosis does not necessarily produce has been reported (p. 836).*

signs of disease, although it has been reported that the

- *Physical obstruction of the oesophagus with potato addition of 10 per cent hay or silage to the diet of 100 toes or other root vegetables causing choke will per cent cereal-fed cattle will improve appetite and*

prevent eructation (p. 826).

weight gain. Some individual animals that are seriously

- *Pressure on the oesophagus by enlarged mediastinal affected with abscesses in the rumen wall and liver may or bronchial lymph nodes prevents gas escaping show reduced appetite and reduced growth rate, par-through the oesophagus. This is a common problem in Alimentary Conditions • 833*

growing cattle three to six months old, particularly bloom stage, undoubtedly predispose to the occurrence those that have been affected with pneumonia (p. 239).

of frothy bloat. Frothy bloat does also occur in feedlot

- *Inability to eructate is also a feature of tetanus (p. cattle, particularly if fed on finely ground grain. This 733) and milk fever (p. 781) and gaseous bloat is a may be due to the gases being trapped by the fine par-frequent feature of these diseases.*

tics of feed, but the rapid fermentation that follows

- *Prolonged lateral recumbency as a result of disease the feeding of finely ground grain is undoubtedly a con-or animals that are cast for prolonged surgery will*

tributary factor. Adaptation to a particular feed is also frequently lead to gaseous bloat because of the important. As the rumen microflora adapts itself to the inability of the rumen gas to escape. Cattle that particular pasture or ration there is a tendency towards inadvertently fall in dorsal recumbency into ditches reduced susceptibility.

will generally die of bloat if not retrieved in time.

- Severe damage to the rumen epithelium following

Signs

acute acidosis may lead to rumen atony and accu-

Rumen tympany, as evidenced by distension of the left mulation of gas (p. 829).

sublumbar fossa, is well recognized by most stockwork-

- Excess cereal ingestion usually results in gaseous ers and should present no problems. With severe bloat (p. 829).

tympany the animal will be exhibiting signs of pain, e.g.

Frothy bloat (primary rumen tympany): Frothy bloat is kicking its ventral abdomen and bellowing. The bellow- much more common than gaseous bloat and usually

ing can frequently be heard up to 500 m (1/3 mile) away.

affects several animals in the group at the same time.

In acute frothy bloat the disease progresses rapidly and

Although frothy bloat does occur in feedlot cattle it is

the animal soon becomes recumbent and can die in

more generally associated with pasture feeding. Pas-

30–60 minutes from the onset of tympany.

tures that are most commonly incriminated in the cause

Pathology

of frothy bloat usually contain high levels of legumi-

nous plants, particularly clover or alfalfa.

Post-mortem bloat in ruminants that die from other

Frothy bloat is extremely common in some countries,

causes can be confusing to the diagnosis of the condition.

e.g. New Zealand (p. 127), due to the high content of

If the animal is seen soon after death, death from bloat

clover in the sward. In these situations the problem is

will be obvious because of the gross abdominal disten-

well recognized and anticipated so that prevention reg-

ion and at post-mortem examination oedema in the

imens are universally applied. In the UK, frothy bloat

inguinal and ventral perineal region together with con-
can appear suddenly without warning on lush spring or
gestion and haemorrhage in the anterior parts of the
autumn pastures and up to 25 per cent of the herd may
carcass will be evident. The liver will be extremely pale
be suddenly affected. Some of these pastures may not
due to compression and the rumen will be grossly dis-
contain a high clover content. The onset may be ex-
tended and contain froth. The quantity of froth present
tremely sudden, often 4–6 hours after milking and return
declines after death. Rupture of the diaphragm and the
to the grazing areas. The only warning that the farmer
abdominal musculature, particularly in the inguinal
experiences is the bellowing of several cows in extreme
region, may also be apparent. Death from gaseous bloat
pain, and on reaching the field there may be several cows
is less common, but the absence of froth and the finding
recumbent or even dead, with many others exhibiting
of a primary lesion should differentiate it from frothy
rumen tympany to varying degrees of severity.
bloat.

Pathogenesis

Diagnosis

Bloat is the result of the inability to eliminate gas from

The preliminary diagnosis presents no problems and is based on distension of the left sublumbar fossa. If only based on distension of the left sublumbar fossa. If only onondary to some other condition or disease.

one animal is affected in the herd the bloat is probably

With frothy bloat eructation is prevented by the a gaseous bloat but if several animals are affected to accumulation of froth, which prevents gas escaping into varying degrees and they are at pasture the diagnosis the oesophagus. The production of froth or foam is a will certainly be frothy bloat. However, if there is any result of the raised viscosity of the rumen fluid and the doubt the passage of a stomach tube will provide the small bubbles of gas, the natural product of rumen fermentation. If the problem is one of gaseous bloat and the mentation, cannot coalesce. Under certain conditions stomach tube reaches the rumen and possibly removes some naturally occurring plant substances, e.g. saponins,

an obstruction on the way, the gas will escape through pectins, hemicellulose and certain proteins, will raise the the stomach tube and the rumen will rapidly revert to viscosity of rumen fluid. There also appears to be an its normal size. If gaseous bloat is confirmed and the individual animal susceptibility. Succulent, high-protein bloat relieved, a full clinical examination should be performed to ascertain the cause of the failure to eructate.

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If the bloat is due to froth little or no gas will escape standard surgical closure. Antibiotics are administered via the stomach tube, which will itself become blocked postoperatively and recovery is usually uneventful. with froth.

Chronic gaseous bloat in calves and feedlot animals is often treated by establishing a 10–15-cm length rumen fistula. Under local anaesthesia the skin and Treatment

musculature of the sublumbar fossa are incised and the The traditional treatment for bloat was the passing of a

rumen exposed. The rumen is then incised and the 5 mm diameter trochar and cannula into the rumen via rumen wall sutured to the skin. Alternatively, a prosthetic device may be sutured into the abdominal wall the left sublumbar fossa. Many farmers possess such a trochar and cannula. However, this instrument is of little use because gaseous bloat can nearly always be relieved by stomach tube and only when this is not possible should a rumen trochar and cannula be used. For primary cause of the bloat will have corrected itself. In frothy bloat the cannula itself becomes blocked with froth and does little to relieve the tympany.

Prevention

Treatment for all but the peracute cases necessitates the passing of a stomach tube and, if this reveals the bloat to be frothy, antifoaming agents that reduce the a sudden acute outbreak is encountered at pasture the

viscosity of the rumen contents and disperse the froth cattle should be removed immediately, provided with dry food such as hay or straw and all cows showing any degree of rumen tympany. Vegetable oils such as linseed, peanut, corn or soya bean oil are all useful antifoaming agents. Traditionally, 500 ml of linseed oil to which is added 50 ml of turpentine was for at least 10 days.

effectively used as a bloat drench, but this does tend to Where risk pastures exist, e.g. those containing high taint the milk. Oil mixed with detergent will disperse proportions of legumes, gradual access to the pasture faster in the rumen ingesta.

should be practised, starting with 10 minutes a day and Proprietary bloat drenches containing silicone or increasing by 10 minutes each day. Long fibre should be poloxalene are available (Table 48.3) and are equally fed before access is allowed to the pasture. Strip-grazing effective. Within five minutes of administration of the

to restrict intake can be practised, but is not favoured
antifoaming agent the animal will start to eructate and
by stock farmers who currently prefer paddock grazing
in most cases the tympany will be relieved within one
or set stocking. However, during high-risk periods in
hour. If tympany still exists after one hour, a further
problem areas these methods alone will not be satis-
administration of an antifoaming agent by drench may
factory. In New Zealand and Australia, where the risk
be given. If tympany still exists one hour after the second
from bloat can be exceedingly high, during the spring,
drench the diagnosis should be reassessed and a stomach
when the pasture is fast growing, the only satisfactory
tube passed to ensure there is no oesophageal obstruc-
method of control has been the daily administration
tion. Once the diagnosis of frothy bloat has been estab-
of antifoaming agents by drench after milking. Oils
lished in a group of affected animals the less acute cases
may be given at doses of up to 240 ml/day in high-
can be treated by drenching alone.
risk periods, although 60–120 ml/day would be more

In peracute cases where death is imminent and the common. Poloxalene, a non-ionic surfactant, is frequently used at 10–20 g/head per day and up to 40 g/day emergency rumenotomy (p. 1106). A 10–20 cm vertical incision is made in the mid point of the sublumbar fossa, If strip grazing is practised, the antifoaming oils can be using a sharp knife. On incising the rumen there will be emulsified with water and sprayed on the grass daily. an explosive release of rumen contents and marked relief Addition to the water supply is sometimes used, but for the cow. Following the release of the frothy rumen effectiveness does depend on adequate individual intake. contents the wound is cleansed and sutured using the Poloxalene can be added to grain mixtures or compound feed or even mineral blocks. However, in grass-rich areas, grain is rarely fed to cattle, particularly in the

incision is made in the mid point of the sublumbar fossa, If strip grazing is practised, the antifoaming oils can be using a sharp knife. On incising the rumen there will be emulsified with water and sprayed on the grass daily.

an explosive release of rumen contents and marked relief

Addition to the water supply is sometimes used, but

for the cow. Following the release of the frothy rumen

effectiveness does depend on adequate individual intake.

contents the wound is cleansed and sutured using the

Poloxalene can be added to grain mixtures or com-

pound feed or even mineral blocks. However, in grass-

*rich areas, grain is rarely fed to cattle, particularly in the **Table 48.3***

Treatments available for frothy bloat in the UK.

bloat-risk season of fast grass growth, and mineral

blocks suffer from variable individual intake. Daily

Name

Active ingredient

Manufacturer

drenching has therefore become the preferred method of bloat prevention. The rumen implantation of a slow-

Birp

Dimethicone

Arnolds

release device containing an antifoaming agent has

Bloat Guard

Poloxalene

Agrimin Ltd

been developed in New Zealand and Australia.

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The ultimate objective in bloat control is to develop

(1)

Pyloric obstruction and abomasal impaction;

pastures that have a low bloat-producing potential yet

(2)

Ruminal distension with atony;

still possess the characteristics for high levels of

(3)

Ruminal distension with hypermotility. In some production. The direction of development will be to cases combinations will occur.

develop strains of leguminous plants that have a low bloat-producing potential. To date some progress has

Pyloric obstruction and abomasal impaction: If the abo-been made in identifying the strains of red clover with

masum is involved there is a pyloric stenosis, which pre-

the ability to produce less bloat, although it is recog-

vents ingesta leaving the abomasum. The ingesta

nized that sainfoin produces fewer tympany problems

accumulates, the abomasum becomes distended and can

than clover. At the moment, pastures should not

be palpated in the abdominal flank. Later the rumen

contain more than 50 per cent clover until such strains

becomes atonic and distends with fluid and ingesta.

of clover are developed (p. 128).

Ruminal distension with atony: This is due either to a primary effect of the paralysed vagus nerve on rumen

Vagal indigestion

function or it can occur following (1) from a backflow

Vagal indigestion is a chronic condition of adult cattle from the distended abomasum. The effect of this is to with a slow insidious onset, but is still a differential produce a characteristic shape to the abdomen. The diagnosis of rumen atony, simple indigestion or mild left flank is distended and well rounded as in mild bloat bloat. The incidence of vagal indigestion is now quite and the right flank is distended in the lower regions of rare, the annual incidence being less than 1 in 10 000, the abdomen, giving the right flank a pear-shape appearance having decreased in recent years, probably mirroring the decline in the incidence of traumatic reticulitis.

some cases of vagal indigestion where only the rumen is affected the organ will be grossly distended, containing

Aetiology

large quantities of fluid. Rumen contractions are infrequent and no rumen sound will be audible. Faecal output

Vagal indigestion is caused by interference with the is decreased and the faeces are frequently of a pale pasty function of the vagus nerve. The condition has been

consistency. Rectal temperature is normal, the heart rate produced experimentally by severing the vagus (Xth cranial) nerve. The clinical syndromes vary slightly to around 40/minute. There is also dehydration, depending on the site of the nerve severance. In practice, the most common cause of nerve damage is the of body condition. Rectal examination will reveal adhesions formed around the reticulum in advanced grossly distended rumen, reaching into the pelvic canal, cases of traumatic reticulitis. The vagus nerve passes and also the ventral rumen distended well over to the through the diaphragm in the region of the reticulum right side of the abdomen. The abomasum is not usually palpated per rectum. The fluid content of the rumen and inflammation and infection that follow traumatic reticulitis. Other lesions that may damage the nerve would abdominal wall on both flanks.

include actinobacillosis of the reticulum, infections of the mediastinal lymph nodes, e.g. tuberculosis, ruptured

Ruminal distension with hypermotility: There is slight diaphragm or pleurisy. Sometimes cattle that survive

ruminal tympany, and frequent and forceful rumen contractions. Recent body weight loss will be evident.

of vagal indigestion. This could be the result of damage to the nerve caused by the torsion.

a gross distension of the dorsal sac of the rumen and the ventral sac will possess a U-shaped distension.

Because of the loss of function of the vagus nerve, ingesta are not transported from the rumen to the abomasum or from the abomasum through the pylorus,

Clinical pathology

with the consequent result that the rumen and often the abomasum distend with fluid and ingesta producing

The experienced clinician will make his diagnosis on the abdominal distension.

clinical signs described and there are no laboratory tests

that can be considered specific for vagal indigestion.

The dehydration will increase the packed cell volume.

Signs

If pyloric stenosis is present a metabolic alkalosis will

The clinical signs are variable in that the abomasum is

be present with the effect of reducing serum chloride to

not always directly involved. Depending on the level at

40–50 mmol/l. An elevation of rumen chloride concen-

tration (above 30 mmol/l) indicates abomasal reflux

which the vagus nerve is damaged various syndromes

can develop that are clinically classed as follows:

into the rumen.

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Diagnosis

Cold cow syndrome

Veterinary advice is usually sought by the stockworker

Another rumen indigestion syndrome that has been

because it is noticed that the cow has slowly developed

reported from different areas of the UK has been

an abdominal distension and it is considered that the

named the cold cow syndrome. This syndrome usually

animal may be affected with bloat. A thorough clinical examination must be performed to establish the differential diagnosis.

to change in pasture use. Both poor and lush pastures have been involved and the occurrence is not related to faeces, the left flank is rounded and distended, the right levels of fertilizer use. The morbidity is high and varies from 8 to 100 per cent of the herd, but mortality is nil. is due to fluid accumulation in the rumen and abomasum, and if the rectal temperature is normal and heart recovers within two days. The condition was first recognized in Northern Ireland in the late 1970s and in the southwest of England in 1982 and has been reported in brought to the veterinarian's attention before all the

several years since.

signs have fully developed and perhaps the right flank distension is not obvious. The insidious onset will distin-

Aetiology

guish the condition from bloat as will the fluid-filled rumen. Abomasal impaction must be distinguished by The aetiology is unknown but several suggestions have ballottement, which will indicate firm abomasal contents been made. These include unusually high levels of in impaction and fluid contents in pyloric stenosis.

soluble carbohydrate found in grass being grazed by Accumulation of fluid within the peritoneal cavity affected herds (Jack, 1985). Other suggestions have from ascites, peritonitis (p. 849) or ruptured urinary been the oestrogenic zearalenone or other metabolites bladder can be differentiated by paracentesis. Hydrops of field microfungi or the presence of high levels of allantois or amnii (p. 1119) will also produce severe soluble proteins or a protein metabolite. Climatic con- abdominal distension, but will be differentiated on ditions have been investigated and cases have occurred

rectal examination.

during periods of frost, cold wet weather and during warm dry springs. Large night/day fluctuations in atmospheric temperature of the order of 17–18°C

Treatment

(30–32°F) accompanied one series of outbreaks.

The prognosis in vagal indigestion is generally poor to

Clinical pathology

hopeless and in most cases immediate slaughter is advised. If treatment is embarked upon because of the Blood samples from affected cows have been examined value of the animal the dehydration and any chloride for a wide range of biochemical parameters but no deficit should be corrected. As much as 40–50 l of abnormalities have been detected.

Ringer's solution may be required over 24 hours and given intravenously. It must be remembered that oral

Signs

fluid therapy must not be contemplated because of the A sudden onset of ataxia and incoordination, with a few fluid retention in the rumen.

animals being weak and becoming recumbent, followed by a copious non-smelling acute diarrhoea are the principal clinical signs. The cows behave as though they are drunk. The cows are characteristically cold to the touch and rumenotomy may be performed. Adhesions around the reticulum can be palpated. A rumenotomy partially empty the rumen is then performed and the reticulum and oesophageal groove examined for tumours or are normal. Appetite is much reduced if not absent and other lesions. The author, on one occasion, found a complete afterbirth wedged in the oesophageal groove in a cow that had not eaten since calving 10 days previously of the disease is short as appetite and milk yield return and was showing signs of rumen atony and distention to normal in two to three days. If cows regaze the same pastures later in the season no disease is seen.

recovery.

Even if tumours, adhesions or oesophageal lesions

Treatment and prevention

are discovered the prognosis is still likely to be hopeless so surgery should only be contemplated in valuable

Because of the rapid recovery treatment appears

animals and a poor prognosis given before surgery

unnecessary, except that the herd should be housed on commences.

dry food for 24 hours and then moved to new pasture.

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Symptomatic treatment of recumbent cows may be

cerning eating habits of the cow, it is quite common for required.

cattle to ingest metallic objects with their food. Some

Until the aetiology of this condition is understood,

abattoir surveys have demonstrated over 50 per cent of preventative measures will not be possible.

cattle reticula to contain foreign objects of metal, wood or stone. If pieces of metal wire or nails 5–10 cm long

The oesophageal groove

are ingested these will accumulate in the reticulum and when rumen contractions occur may penetrate the Lesions of the oesophageal groove will interfere with the reticulum wall. The incidence of this disease varies con-normal rumen digestion process. Tumours or granuloma-siderably around the world. The incidence in the UK tous lesions will occlude the oesophagus and prevent has declined in the last 30 years and the present annual eructation and lead to a gaseous bloat or secondary incidence is around 5 cases per 10 000 cows. However, rumen tympany. The same lesions may interfere with in some areas the disease is still extremely common and normal rumen contractions and cause signs of simple indi-may reach an annual incidence of 100–200 per 10 000 gestion. Although not common (annual incidence 1 per cows.

10 000 cattle) actinobacillosis of the oesophageal groove This disease is probably related to standards of man-does occasionally occur. Some lesions of the oesophageal agement that exist on the farm, e.g. rusty, poorly main-groove may be due to upper alimentary squamous cell

tained, barbed wire fences are a frequent source of the carcinoma in bracken areas (see pp. 828, 946).

offending wire. It is thought that the incidence has

Signs

declined since string has replaced wire to secure bales of hay and straw and much barbed wire fencing has

The presenting signs of actinobacillosis of the oesophageal groove will be inappetence, reduced milk yield and possibly mild tympany evidenced by distension of the left sublumbar fossa. The rumen contraction will be weak, occurring once every 1 or 2 minutes. Rectal picked up by hay or silage making machinery.

temperature and heart rate will be normal. Examination of the faeces will reveal strands of undigested fibre.

Although this latter finding is in itself only an indication

Pathogenesis

of indigestion, and several lesions of the rumen or rumen

Metal objects that are ingested invariably lodge in the wall or dental problems may lead to undigested fibre floor of the reticulum due to their relative mass and the appearing in the faeces, the most common cause is likely position of the reticulum. It is only short sharp objects to be actinobacillosis of the oesophageal groove.

that penetrate the reticulum wall and these are usually 5–10 cm long. The penetration occurs as a result of the

Diagnosis

ruminal and reticular contractions. On entering the wall

Only a tentative diagnosis can be made based on the of reticulum the wire will continue to penetrate until it clinical observations described above. Although diagnosis reaches the peritoneum. Infection from the rumen then nosis would be confirmed by performing an exploratory follows the wire and a localized peritonitis is produced, laparotomy and a biopsy of any oesophageal groove causing local abscess formation and adhesions. If the lesion discovered, this is unlikely to be performed in direction of the wire is forward the diaphragm and peripractice because of economic considerations.

cardium may be punctured, which produces a localized pleurisy and pericarditis (p. 731). If the direction of the

Treatment

wire is left or right of the forward direction the diaphragm may not be involved but extensive peritonitis (p. 849) could well develop, with adhesions containing a variety of abscesses being produced. If the prognosis in these cases must be guarded because of the uncertainty of the diagnosis, cows presenting the above described lesions are worthy of treatment. If the peritonitis is extensive, adhesions between the reticulum and liver or spleen may be evident. Sequelae to traumatic reticulitis include localized or diffuse peritonitis, otic of choice is streptomycin and a dose of 5–6 g daily for 10 days is effective in a proportion of cows exhibiting the above described signs.

Traumatic reticulitis (traumatic

Signs

reticuloperitonitis, hardware disease, wire)

The condition is generally progressive and the clinical signs change as the disease progresses from the initial acute phase through a subacute to a chronic phase.

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In the initial acute phase the cow is anorexic and milk production is reduced. The cow may exhibit an arched back and the abdomen is tucked up. A grunt may be heard when the animal walks, although on occasions there will only be an indication of peritonitis and not specific for traumatic reticulitis. Grunting can also be induced by applying sharp pressure just to the left of the xiphoid (102.5–104°F) and although the frequency of rumen

process using a clenched fist. This will specifically indicate pain in the region of the reticulum.

Contractions may be reduced they are often increased to three or four per minute. Respiratory movements

The most successful diagnostic test is known as the 'reticular grunt' or Williams test (Williams, 1975). This will be shallow, increased in rate and mostly thoracic.

Frequently, farmers mistakenly diagnose the condition as pneumonia. The heart rate will be raised to around 75–90/minute. The test uses knowledge of the cycle of reticulo-rumen contractions:

(1)

Contractions of the reticulum, followed by contraction of the rumen. There is no eructation at this

The acute phase may last three to five days and then the rectal temperature will fall to around 39°C (102°F) stage.

or sometimes to normal. The subacute phase will last

(2)

Following relaxation of the reticulum and rumen,

several weeks, showing signs of mild indigestion.

an independent contraction of the rumen occurs.

Rumen contractions will be weak and infrequent, there

This contraction is accompanied by eructation.

may be a mild tympany and undigested fibrous mate-

(3)

Relaxation of the reticulum and rumen completes

rial may be evident in the faeces. Sometimes apparent

the cycle. The 'reticular grunt' is based on the cor-

recovery from the acute phase will occur and the animal

relation of pain with contractions of the reticulum.

will return to near normal production, but a relapse will

Since the reticular contractions occur in conjunc-

occur several weeks later. This relapse is usually asso-

tion with rumen contractions, the clinician should

ciated with the onset of pericarditis. The animal will be

observe for signs of pain during or just before the

reluctant to move, may occasionally grunt but signs of

non-eructating rumen contractions. These signs of

cardiac insufficiency will predominate. A firm pro-

pain will be a mild grunt, and shuffling of the

nounced jugular pulse will be noticeable, together with forelegs. To help detect the grunt, observation of oedema of the brisket and auscultation of the lungs may the left costal arch may reveal the animal holding reveal signs of congestion or even pleurisy. Auscultation its breath just before it grunts.

of the heart will sometimes be difficult, as the heart sounds will be muffled and difficult to hear. However, This test is specific for traumatic reticulitis, but is only splashing and tinkling sounds over the heart region will effective in the acute phase of the disease.

confirm the presence of pericarditis (see p. 731).

Diagnosis of the subacute phase may prove extremely difficult, as the signs are often vague and only indicative

Pathology:

of a non-specific indigestion. If such cases are encountered and a diagnosis of indigestion made, but a return to

During the acute phase there will be a measurable normal is not rapidly achieved, subacute traumatic reticulitis/peritonitis must be considered as a differential

rising to 30 ¥ 109/l (30 000/ml). Ketone bodies may be diagnosis.

present in urine or milk indicating a secondary acetone-

The use of metal detectors has been advocated by

aemia. During the subacute phase, when peritonitis is

some workers. However, because metal objects are frequently found in the reticulum a positive metal detector test will often be misleading.

At post mortem, the degree of peritonitis can be dramatic with adhesions between the reticulum, rumen,

Diagnosis of pericarditis is more straightforward. The diaphragm, liver and spleen. Incising through the adhesions may reveal multiple abscesses. Abscesses may also to auscultate the heart in detail. Muffled heart sounds

be present in the liver. The pericarditis, when present, will

and splashing, fluid or tinkling sounds around the heart also be dramatic with gross thickening of the pericardium

will confirm pericarditis.

and large quantities of pus present in the pericardial sac,

so-called bread and butter heart. Pleurisy, and localized

Treatment

pneumonia and abscessation may also be present.

Various treatment regimens have been advocated for traumatic reticulitis and they can be classified into sur-

Diagnosis

gical and conservative.

The diagnosis of the acute phase is based on the signs

If a diagnosis is made during the acute or subacute

described and discovered during the clinical examina-

phase and there are no signs of pericarditis, the surgical

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approach has much to commend it. In many cases the

There are three important conditions that probably

diagnosis is only tentative and the rumenotomy is

have a similar aetiology and epidemiology, yet produce

exploratory to confirm diagnosis or establish another

widely differing clinical syndromes. These are left dis-

agnosis. Under paravertebral anaesthesia, an 18-cm

placement of the abomasum (LDA), dilatation and

vertical incision is made in the sublumbar fossa, the

torsion in the right flank (RDA) and ulceration.

rumen wall exteriorized and a 10–15-cm incision made

Impaction of the abomasum has also been reported.

into which a McLintock ring is fitted, thus temporarily

The three important conditions (LDA, RDA and ulcer-

fixing the rumen wall to the exterior and preventing peri-

toneal contamination with rumen contents. A scrubbed

of cattle and have not been recorded in wild ruminants.

arm is inserted into the rumen and the reticulum located.

In fact their occurrence, particularly LDA, is almost

Each crypt of the reticulum should be explored as the

entirely restricted to dairy cattle and rarely found in

offending wire may have penetrated to the extent that

suckler beef cows. Diet undoubtedly has an important

very little is left protruding into the reticulum. Adhe-

role in the aetiology of these diseases, with the use of

sions between the reticulum and diaphragm or abdomi-

concentrated cereal-based feeds and low-fibre diets

nal floor can be detected by attempting to lift the

generally being incriminated. In some countries the

reticular wall. Once located, the offending wire should feeding of root crops that are heavily contaminated be slowly withdrawn back through the reticular wall and with soil, sand and gravel has also been incriminated. removed from the rumen. The rumen incision is closed In the UK and USA, LDA is much more common than using Lembert sutures and the abdominal wall incision RDA or ulceration. However, reports from Scandinavia closed in the usual way (see p. 1106).

would indicate that RDA is much more frequently seen Five days of antibiotic treatment should follow the there than in the UK or USA and may be the result of surgery to prevent the spread of the peritonitis initiated much greater use of root feeds such as fodder beet.

by the foreign body. This operation can be very satisfactory to conduct in practice and the majority of cases

Displacement of the abomasum to the left

make an uneventful recovery.

The conservative treatment involves restricting the Left displacement of the abomasum is by far the most animal's movement by tying it in a byre stall with the

common of the abomasal diseases encountered in cattle front feet raised 35–40 cm higher than the hind feet for in the UK or USA. Its occurrence is almost entirely three weeks. Parenteral antibiotics will also be given for confined to dairy cattle, although it has very occasional five to seven days, and in some countries a magnet will ally been seen in bulls, where it is probably secondary be inserted into the reticulum using a balling gun. to some other condition. The author has, on one occasion only, diagnosed the condition in a Friesian bull and recommended, as there is little likelihood of recovery. If pericarditis is evident immediate slaughter must be in that case the animal was suffering from severe endocarditis. On no account should a rumenotomy be considered. It was likely that this particular LDA was a secondary because even if the wire is located, removing it may well onduary condition brought about by the inappetence cause the heart to stop and death during surgery will be caused by the endocarditis. The overall incidence varies the result. considerably between years and between seasons. In

some years the annual incidence can be as high as 25–30

Prevention

per 10 000 and in others as low as 4–6 per 10 000.

There appears, in the UK, to be a definite seasonal

The main thrust of prevention must be to avoid leaving

pattern to the incidence with the majority of cases

wire or nails lying around to be picked up by cattle

occurring in late winter–early spring, i.e. January to

during feeding. The use of metal detectors on forage

April, the period of winter housing in the UK.

harvesting equipment to prevent damage to the equip-

However, it is now being seen throughout the year,

ment has undoubtedly reduced the incidence of metal

although at lower numbers during grazing. In the USA,

objects being found in cattle feeds. Some workers have

LDA is reported to be more common in the winter

advocated the routine use of magnets. These are

housing period. However, the problem is encountered

inserted into all cattle on the farm. There is no real evi-

in countries where spring calving predominates and the

dence that these have been successful.

cows are at pasture during the susceptible period, e.g. Ireland, Australia and New Zealand, although the inci-

Abomasum

dence varies considerably between farms. The author has experienced the condition in eight cows in a 110-Diseases of the bovine abomasum comprise an inter-cow herd in one year, all occurring from January to esting group of conditions only really appreciated in April; yet there are many farms that have never, know-comparatively recent years (Pinsent, 1978).

ingly, experienced the condition. Breed susceptibility

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has been investigated and there has been no authorita-80–100/minute. Rumen movements will usually be tive confirmation that there is a genetic predisposition. absent or at least infrequent and in some cases palpation However, it is thought that the condition generally of the rumen in the left sublumbar fossa will be impossi-affects the higher yielding cows, mainly in early lacta-ble because of the presence of the gas-filled abomasum tion, although occasionally during late pregnancy.

between the left abdominal wall and the rumen. A

Rothera's test on urine or milk will usually be positive

Aetiology and pathogenesis

and thus in many instances the cow will be presented as

a suspected case of acetonaemia. In the more acute cases,

The precise aetiology of LDA is not readily understood

which are more common in late pregnancy, distension of

but the occurrence of the problem soon after, or occa-

the left flank will be evident.

sionally just before, parturition would suggest that the

presence of the gravid uterus or the process of parturi-

tion predisposes to the condition. Certainly it has been

Diagnosis

observed that in normal cows in late pregnancy the

The diagnosis of LDA is relatively easy, providing the cli-

presence of the gravid uterus displaces the abomasum

nician always keeps the condition in mind when making

forwards and to the left, and after calving the organ

a clinical examination of dairy cows. Confirmation is

returns to its normal position. To remain displaced after

based on auscultation, percussion and auscultation, or

calving, the abomasum must have developed atony and ballottement and auscultation of the left flank. The the subsequent accumulation of gas. Atony of the abo- stethoscope is placed on the last intercostal space in line masum is likely to be caused by one of four factors: with the lower limit of the left sublumbar fossa and the penultimate rib is percussed by 'flicking' it with the finger.

(1)

Feeding of rapidly fermentable concentrate feeds, If a 'ping' or high-pitched resonant sound is heard, this is which have a tendency towards the production of indicative of a gas-filled organ inside the abdominal wall. acidosis.

Should the first attempt fail to elicit the characteristic

(2)

The accumulation of sand or gravel in the aboma- 'ping' the stethoscope is moved so that an area repre- sum, which damages the abomasal mucosa. senting a 20 cm square forward and below the first stetho-

(3)

Stress conditions or metabolic diseases that

ical effect of displacement by the gravid uterus may well described above may frequently elicit the same tinkling have some involvement in originally displacing the abomasum. The diagnosis, by auscultation of the tinkling sounds without recourse to ballottement, is to be located between the left abdominal wall and the rumen favoured because ballottement may elicit splashing the atonic nature of the abomasum and the presence of sounds from the rumen that can be difficult to distinguish gas will prevent the organ returning to its normal position. from the high-pitched tinkling sounds diagnostic of LDA. However, a negative diagnosis can fairly quickly be achieved by using the percussion and auscultation

Signs

technique over a 20 cm square area forward of and The clinical signs of LDA can vary considerably, although ventral to the lower limit of the left sublumbar fossa. in general the signs are similar to chronic acetonaemia. Only when this technique produces a pinging noise Mild cases are encountered that show little more than a

should it be necessary to spend time in auscultation alone.

slightly depressed appetite, rumination and milk yield.

All cows that are presented with inappetence and have

At the other extreme, acute cases can be encountered

a normal rectal temperature, particularly in early lacta-

with complete inappetence, absence of rumination, loss

tion or late pregnancy, should be subjected to the per-

of condition, scanty diarrhoea and grunting with some

cussion and auscultation technique to eliminate LDA as

signs of mild colic. The most common clinical picture is

part of a normal clinical examination. If this is performed

one of refusal to eat concentrates, some reduction in milk

the clinician is unlikely to fail to diagnose LDA.

yield and scanty soft or pasty faeces. Rectal temperature

However, it must be said that a small number of cases

will be normal but the heart rate may be raised to

are not diagnosed at the first examination and a diag-

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nosis of acetonaemia or, occasionally, indigestion is

It is essential if the LDA has been corrected by

made and the corresponding treatment administered.

rolling that the animal is re-examined 48 hours later to
Such cases will show a temporary recovery but the signs
ensure that a relapse has not occurred. In the experi-
will relapse two to five days later. It is essential when
ence of the author, and many others, relapse is to be
treating acetonaemia or indigestion that the farmer is
expected in over 75 per cent of cases of LDA that have
instructed to seek further veterinary advice if the con-
been corrected by the rolling technique. Quite fre-
quently if relapse does occur, the signs may not be so
severe as those present before the correction and the
days previously, either by the farmer or a veterinarian.
animal may well complete its lactation, albeit with a
Following a complete clinical examination the differ-
reduced total milk yield. In such cases, chronic LDA
ential diagnosis of LDA is related to whether the high-
exists and ulceration of the abomasum with adhesions
pitched resonant sounds can be confused with anything
to the abdominal wall may well develop, thus shorten-

else. Once heard, these sounds are never forgotten.

ing the productive lifetime of the animal.

However, similar sounds do occur in conditions of the

The surgical approach to treatment consists of

rumen that produce rumen atony, a rumen mildly dis-

laparotomy, returning the abomasum to the right side

tended with gas accumulation, as is seen in vagal indi-

of the abdomen and suture fixation of the organ to the

gestion, actinobacillosis of the oesophageal groove,

abdominal wall. Many different surgical techniques

localized peritonitis or mild rumen tympany. However,

have been described for this procedure but the one

if the tinkling sounds are heard spontaneously and not

favoured by the author is the right flank approach with

induced by ballottement, the likelihood of misdiagnos-

the animal standing. The animal is starved of water and

ing these other conditions as LDA is slim. The author

food for 24 hours prior to surgery to reduce the size of

has on one occasion, having made a diagnosis of abo-

the rumen. Using paravertebral anaesthesia, a 20-cm

masal dilatation in the right side of the abdomen by rec-

*incision is made in the abdominal wall, starting at the
ognizing the tinkling sounds, performed a laparotomy
lower limit of the right sublumbar fossa and extending
to discover the sounds were produced by a large sub-
vertically downwards. The left arm then enters the
peritoneal abscess in the upper right abdomen.*

*abdominal cavity and moves carefully down the right
side of the abdomen, along the floor and up the left side
with the hand always in contact with the peritoneum.*

Treatment (see also p. 1109)

*The hand can then locate the distended abomasum sit-
The treatment for LDA falls into two categories, con-
uated high in the left flank between the rumen and the
servative or surgical.*

abdominal wall. The hand is then placed over the top

*Conservative measures include drug therapy and
of the abomasum and downward pressure exerted.*

rolling. Drug therapy using calcium borogluconate

Several attempts at downward pressure may be

solution, neostigmine and saline cathartics has been

required to disperse the gas present in the organ. The

attempted with very little success. The usual conservative treatment is to roll the cow. The cow is cast, using the Reuff's method, on to its right side. The cow is then rolled into dorsal recumbency and kept in this position for 5 minutes. During this time the animal may be rocked to the left and right and the abdomen massaged. When the organ has been brought across to the right side, the pylorus is located and brought to the abdominal wall incision. Using non-absorbable suture material, left side lateral recumbency and maintained in this position for a further 5 minutes, allowing time for the abomasum to return to its normal position. During this

sum close to the pylorus and this is then sutured to process, splashing and gurgling sounds can be heard the peritoneum and abdomen wall at the base of the coming from the abdomen as the abomasum moves. incision. The abdominal wall incision is closed in the The animal is then allowed to rise. The left flank is then normal way and postoperative antibiotics administered. auscultated to ensure the abomasum is still not present Appetite is stimulated by the administration of rumen- in the LDA position.

stimulant drenches and the animal allowed immediate One variation of this procedure reported to be successful by one UK practitioner (B. Jeffrey, pers. comm.) used this technique for over 20 years and on one occasion only has correction not been possible. In this case restrain the animal in this position for 30 minutes. The adhesions were present between the abomasum and the gas present in the abomasum allows the organ to 'float' left abdominal wall. A second incision was then made

back to its normal position.

in the left sublumbar fossa, and the adhesions were

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broken down revealing a perforated ulcer. The ulcer

Prevention

was excised, sutured using a purse string suture and the

Advice on prevention is difficult because the precise

abomasum returned to the right flank where an assis-

nature of the aetiology is unknown. Furthermore, most

tant located and sutured it as described above. This

cases occur only sporadically and it is rare for farms to

right-sided approach has been used widely in the UK

experience more than one or two cases in a season. If the

and is suitable for operating on the farm.

incidence is higher on a particular farm, attention to the

The right paramedian approach is also widely used.

feeding regimens may prove worthwhile. Dry cows

The animal is cast into dorsal recumbency and local

should be fed a diet of long fibre and very little concen-

anaesthesia administered for an incision posterior to

trates. Dry-cow diets should always contain less than 30

the sternum and midway between the midline and the per cent concentrate on a DM basis. Maize silage, which right subcutaneous abdominal vein. The abomasum is itself contains up to 50 per cent grain on a DM basis, located, returned to its normal position and fixed using should be restricted to no more than 15 kg/day during the catgut or monofilament nylon sutures 2–3 cm from the dry period. The change in diets that occur at calving margin of the incision. This technique does return the should be made as gradually as possible. Ideally, 2 kg abomasum to its normal position, whereas in the former daily of concentrates for the last two weeks of the dry the fixation of the pylorus to the abdominal flank period will help the rumen microflora adjust to the wound results in the abomasum being slightly out of increased concentrate intake that occurs in early lacta- position. In the author's opinion the abomasopexy tion. The approach to prevention of LDA is much the should be performed with non-absorbable suture mate- same as the approach to the prevention of acetonaemia. rial as the few cases that have relapsed following

abomasopexy were mostly sutured with catgut.

A modification of the right paramedian approach, the

Right-sided abomasal dilatation

Sterner–Grymer closed suture technique, has been

and torsion

gaining popularity in recent years and with consider-

Occurrence

able success (Gordon, 2001). The cow is cast onto her

right side and rolled into dorsal recumbency, as above,

Right-sided abomasal dilatation and torsion occurs

and the hind legs restrained. The abomasum is auscul-

much less frequently than LDA in the UK and USA,

tated by percussion using a stethoscope and should be

although it appears to be more common in Denmark.

on the right of the midline. A DA Trochar and Cannula

The annual incidence in the UK is of the order of 2–3

(Kruuse UK Ltd) is inserted firmly through the skin,

per 10 000. It normally occurs in early lactation and

muscle and peritoneum 10 cm caudal to the xiphoid

rarely in the dry cow, but although predominantly

process, 10 cm to the right of midline and into the

affecting dairy cows it has been reported in bulls, young abomasum, avoiding the right subcutaneous abdominal animals, beef cows and feedlot cattle.

vein. The trochar is removed and a toggle attached to a suture (Kruuse UK Ltd) quickly inserted through the

Pathogenesis

cannula. Minimal gas should be released when placing

As with LDA, the aetiology of RDA is not fully under-

the first suture. The procedure is repeated with a second

stood but the pathogenesis is probably similar to that

toggle inserted 5 cm cranial to the first, allowing as

of LDA. Atony of the abomasum followed by the

much gas as possible to escape from the abomasum to

accumulation of feed, fluid and gas produces a grossly

reduce tension on the sutures. The two toggle sutures

distended organ. The presence of gravel and sand in the

are tied together and the cow rolled onto her left side

abomasum has commonly been observed in affected

and allowed to stand.

animals, which may account for the higher incidence in

Another technique involves a left flank approach,

Denmark, where large quantities of fodder beet are fed pushing the abomasum back to the right side and then that often is contaminated with soil. Torsion is frequently a sequel to the dilatation and this is purely a rumen to the floor of the abdomen by using a large mechanical effect of the increased weight and size of needle and a long suture. The suture needle is passed the dilated organ. The torsion can be in several directions, e.g. the organ may be rotated dorsally 90–180°, or wall to the exterior. The needle is removed from the counterclockwise up to 180° as viewed from the rear or suture and the procedure repeated with the other end the torsion may incorporate the omasum.

of the suture material and the suture tied as a mattress suture on the exterior of the ventral abdomen. This produces an adhesion between the ventral rumen and the ventral abdomen that will prevent a recurrence of the

Signs

The onset of dilatation is insidious with inappetence,

LDA.

milk yield reduction and varying degrees of ketosis.

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Rumination ceases and rumen contractions are weak

However, in valuable animals and less severely

*and infrequent. Faecal quantity is reduced but its con-
affected cows, surgery can be successful (p. 1112). Intra-*

sistency is usually diarrhoeic, foul smelling and often

venous drip therapy should be set up immediately using

contains occult blood. Rectal temperature is normal

Ringer's solution or isotonic sodium chloride. Cows

and heart rate raised to 80–100 beats/minute. Mild colic

with RDA plus torsion will be suffering from metabolic

signs may also be evident and there is often a notice-

alkalosis and will be short of chloride ions. Sodium

able distension of the right flank. Once torsion occurs,

bicarbonate or sodium lactate should not be used. To

the signs become peracute. Then there is a subnormal

restore normal hydration, 40–50 l of electrolyte are

temperature, with a heart rate up to 160/minute, cold

likely to be required over a 24-hour period.

extremities and extreme dullness. These signs indicate
Having set up the intravenous drip, surgery should
severe shock and frequently colic may be observed. At
commence immediately. Under paravertebral anaesthe-
this stage the animal is anorexic and the rectum will be
sia an incision is made in the right abdominal wall with
empty except for some tar-like mucus.

the animal standing. If the animal cannot stand it is likely
her condition is so severe that surgery would not succeed

Diagnosis

and casualty slaughter should be advised. On entering
the abdomen and the abomasum located the first proce-
Diagnosis should not present any problems if the tech-
dure is to deflate the organ using a wide bore (12G)
niques of percussion and auscultation, ballottement and
needle and rubber tube. Having deflated the organ the
auscultation and auscultation alone are applied to the
direction of the torsion should be identified and an
right flank in the same way as described for the diag-
attempt made to correct the torsion without removing
nosis of LDA on the left flank. The same 'ping' and tin-

the fluid. If correction of the torsion is not possible then
kling sounds if heard on the right flank will indicate an
half of the fluid should be siphoned out of the aboma-
RDA. Determination of whether torsion exists will rely
sum. This will ease the recognition of the torsion direc-
on the severity of the signs exhibited, e.g. the presence
tion and more readily allow repositioning of the
of a very fast heart rate, subnormal temperature, the
abomasum. Abomasopexy is carried out as for LDA cor-
signs of shock and the consistency of the rectal contents.
rection. The abdomen is closed in the normal way. If
Rectal palpation may also reveal the presence of a
RDA exists without torsion the abomasum should be
grossly dilated viscus in the right sublumbar region. Dif-
emptied of fluid by siphoning and then the organ opened
ferential diagnosis of RDA plus torsion will include all
and all debris, which may include straw or hairballs,
causes of acute abdominal obstruction, particularly
stones or gravel, should be removed. The abomasum inci-
caecal dilatation and torsion, torsion of small inte-
sion is then closed, the abomasum is returned to its

stines, intussusception and perforated abomasal ulcer. normal position and the abdomen closed in the normal. However, being able to palpate the organ on rectal way. Abomasopexy is not normally required in this situation. Fluid therapy is continued until the animal has sounds should not cause problems in diagnosis.

rehydrated and postoperative antibiotics are administered for five days. Following recovery diarrhoea will be

Treatment

present for two or three days.

If torsion is not present there is often a temptation to try. Recovery rates of 75–80 per cent are reported by some conservative treatments using antacids by mouth and workers in hospital situations and around 50 per cent spasmolytics, vitamins or antibiotics by injection. This is when the omasum is involved in the torsion. Recovery not to be recommended. Although some cases of dilatation rates for on-farm surgery are likely to be lower and this tion do recover spontaneously, many do not. Many cases must be appreciated before embarking on surgery in

will remain dilated and a chronic state of abomasal
preference to casualty slaughter. The likelihood of recovery
dilatation develops where the animal loses weight and
ery will depend on the length of time that elapses from
milk production, and eventually is culled as a 'poor doer'.
torsion occurring to operation and the level of shock that
Often the dilatation progresses to torsion and surgery to
exists at the time operation commences.

correct abomasal torsion is much less successful than correcting
dilatation without torsion. This is because of the

Prevention

severe shock that is induced in the animal by the torsion.

The decision on whether to attempt surgery in cases of

Measures to prevent RDA are the same as for LDA

RDA plus torsion will depend on the degree of shock that

(p. 842) but as the condition is relatively rare, specific

exists. In severe cases casualty slaughter should be

measures to prevent RDA are academic. If root

advised. Recently, successful treatment of dilatation

vegetables are used in large quantities for fodder they

without torsion has been reported using metoclopramide

should be washed before feeding to remove the soil hydrochloride, although not licensed for this purpose. contamination.

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Abomasal ulceration

The diagnosis of abomasal ulcers with perforation is difficult. The signs are of mild colic and pain in the right Ulceration in the form of small multiple ulcers occurs in ventral abdomen. Confirmation will only come from an the abomasal mucosal surface in a number of systemic dis-exploratory laparotomy. Abomasal ulcers may well be eases but these are rarely diagnosed, their presence being present in many apparently normal cows and undoubt-masked by the other signs present of the systemic disease. edly many do heal, but the sudden onset of signs asso- However, a syndrome of peptic ulceration in adult ciated with acute haemorrhage or perforation would cattle does occur sporadically and may result in perfo-indicate that until such dramatic consequences occur ration and peritonitis or haemorrhage, which can be the ulcers may not be harmful.

mild and recurrent or acute and be a cause of death. The actual incidence of abomasal ulceration is probably

Treatment

much more common than generally realized as many cases are difficult to diagnose and may produce little

Treatment is purely academic. Ulcers without perfora- harmful effect until perforation or haemorrhage occurs.

tion or haemorrhage are unlikely to be diagnosed. The

The aetiology is uncertain, but feeding regimens involv-

prognosis in haemorrhaging ulcers is so uncertain that

ing a sudden introduction of concentrate feeds are likely

casualty slaughter should be advised. Perforated ulcers,

to be implicated much the same as in the presumed aeti-

if not sealed with omentum, are usually only discovered

ology of LDA and RDA. The author has experienced

at post-mortem examination. Ulcers that have perfo-

three sudden deaths in a herd of 120 dairy cattle that

rated and become sealed with omentum are unlikely to

were caused by perforated abomasal ulcers. The abo-

be diagnosed,except on exploratory laparotomy in a cow

masum contained large quantities of sand and gravel,

showing signs of right-sided anterior abdominal pain and which were the result of a depraved appetite. The cows when discovered they are probably best left undisturbed. were constantly eating soil, which was later confirmed to be due to hypocuprosis. The aetiology of peptic abo-

Prevention:

masal ulceration is therefore similar to LDA and RDA and is considered by some workers to be another manifestation of the same syndrome. Certainly, ulcers are best advice on prevention is to follow that for LDA and found in the abomasum of both LDA and RDA.

RDA (p. 842).

The ulcers occur singly or occasionally in twos and threes and vary in size from 2 to 6 cm in diameter. Fungal

Abomasal impaction

hyphae are frequently found in the depths of the ulcers, Impaction of the abomasum may occur occasionally but which tend to extend into the submucosal layers until, in is certainly of no great significance in adult cattle. Its some cases, perforation occurs. If the perforation occurs

annual incidence would be less than 1 per 100 000 and at a point covered by omentum the ulcer may be sealed appears to be more common in beef cows than dairy by omental adhesion. However, if the ulcer perforates at cows as a result of feeding poor quality fibrous material a point lateral to the omental covering, the abomasal contents spill into the peritoneal cavity and death soon follows. The impaction occurs with the accumulation of large quantities of fibrous food, sand or gravel close to the pyloric outlet. The onset is insidious, with a gradual loss may be only temporary and the blood vessel heals, but in milk production and inappetence. Progressively, more often the haemorrhage does not stop and the rumen impaction occurs, rumination ceases and the animal dies from blood loss. This can be a cause of sudden death. The annual incidence for abomasal ulcers with rate will exceed 100/minute. At first there is little or no

haemorrhage would be in the order of 5–10 per 10 000.

abdominal pain but progressively pain becomes evident

in the anterior right ventral abdomen (as distinct from

Diagnosis

the left anterior abdomen in traumatic reticulitis).

The only occasion where the syndrome can be diag-

Pinching of the withers at this stage may elicit a painful

nosed with certainty is when haemorrhage occurs. A

grunt. It is unlikely that a positive diagnosis will be

cow presented with inappetence, reduced milk yield

made without recourse to an exploratory laparotomy

and passing black tarry faeces, which contain large

when an enlarged doughy abomasum will be palpated.

quantities of occult blood, will almost certainly be suf-

Pinsent (1977) was of the opinion that many cases of

fering from a haemorrhaging abomasal ulcer. Severe

abomasal impaction reported in the past may well have

cases will be anaemic, the heart rate fast and loud and

been vague indigestion as workers reported enlarge-

death can occur within 24 hours. The prognosis in such

ment of the fundus of the abomasum, which contained

cases is extremely guarded for, although animals do dry rumen contents, and an accumulation of fluid within appear to make a recovery, relapses are common. the rumen.

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However, if such a case is encountered at exploratory of management, whether extensive or intensive. A thor-laparotomy, abomasotomy and removal of the offend-ough clinical examination will be required to differenti-ing contents can be attempted.

ate the many problems that produce colic. By far the most common cause of colic is tympanic intestinal colic, which

Abomasal impaction in calves

is very similar to the syndrome so frequently seen in the horse. Intussusception, caecal dilatation and prolapse of Abomasal impaction does appear to be more common the small intestine through the mesentery are much less in calves than adult cattle. Calves from three weeks to common and in the UK each would have an annual inci-three months can be affected, although six to ten weeks dence of less than 1 per 10 000.

is the most common period. Depraved appetite, causing the calves to eat bedding and lick hair, is thought to be

Signs

the cause. Finely ground grains made into pellets have also been incriminated in the aetiology, presumably the

The clinical signs of colic in cattle are firstly reduced result of rapid fermentation. Coarse ration where the appetite or even anorexia, reduced milk yield and a grain ingredients are rolled or cracked is preferred to noticeable change in behaviour – kicking at the ventral pellets by many calf rearers because fewer ‘digestive abdomen, shifting weight from one hind foot to the other, upsets’ appear to occur.

licking at the flank or chest wall, frequently lying down and then standing and generally restless. The intensity of

Diagnosis

the signs exhibited will vary with the degree of pain. On some occasions the signs are quite mild, with only occa-

Affected calves usually have a brown, mild diarrhoea sional kicking at the ventral abdomen, and may be missed and normal rectal temperature. The most striking

by all but the most astute stockworker. The above signs feature is the result of ballottement of the lower right are all indicative of pain, but not necessarily abdominal abdomen, which will reveal loud splashing noises over pain. Conditions that will produce similar signs include a large area, usually indicating an enlarged abomasum photosensitization (p. 884), particularly if the teats are containing excess fluid.

affected, strangulated scrotal hernia, uterine torsion, urolithiasis (p. 263) and ureter obstruction.

Treatment

Treatment using antacids such as magnesium hydroxide

Diagnosis

or magnesium carbonate or mild laxatives such as liquid

The diagnosis of the cause of the signs of colic will include paraffin or linseed oil may be helpful. In early cases surgical interference to empty the abomasum can be effective. It is interesting to note that the abomasum of veal the more acute the problem, the faster and weaker will

calves slaughtered at 14–16 weeks old and reared solely
be the pulse. Abdominal sounds may be present or
on liquid milk substitute diets frequently contains one
absent. In intestinal obstruction the sounds will be
or several hairballs many up to 20 cm in diameter
absent. The mucous membranes will be injected in acute
without abomasal ulceration or any abnormal effects
problems and the eyes sunken. Ballottement of the
being noticed before slaughter.

ventral abdomen should always be performed and may
reveal splashing fluid sounds. A rectal examination

Colic and acute intestinal obstruction

should also be performed. In intestinal obstruction the
rectum will be empty and sticky. Enlarged viscera or
abnormal positioning of the viscera may be palpated on
Colic signs are frequently reported in adult cattle. They
rectal palpation. A detailed description of the use of
may indicate a tympanic intestinal colic or they may
rectal palpation in the diagnosis of abdominal disorders
signal some more serious problem. This section will
has been recorded by Stober and Dirkson (1977).

discuss the differential diagnosis of colic signs and will

In many cases of intestinal obstruction diagnosis

describe in detail: tympanic intestinal colic, intussus-

may only be confirmed on exploratory laparotomy.

ception, caecal dilatation and torsion, prolapse of small

Exploratory laparotomy as a diagnostic procedure as

intestine through a ruptured mesentery and torsion of

opposed to its use solely as a surgical treatment is to be

small intestine around the root of the mesentery.

recommended (Pinsent, 1978) where the attitude of the

farmer and conditions conducive to surgery exist. Many

Occurrence

farmers, with their increased education and training, will

Colic is a sporadic condition affecting only individual

understand the value of an exploratory laparotomy if the

animals, it being extremely rare for more than one animal

problem is fully explained. Furthermore, exploratory

to be affected at any one time. Signs of colic are reported

laparotomy may well reveal the cause of acute intestinal

quite frequently in all ages of cattle and under all types

obstruction and allow surgical correction and recovery

to take place, whereas if surgical intervention is unnecessary rolling of cows to correct uterine torsion or left displacement of the abomasum. The entire small intestine reduced. The author has, on two occasions, performed an exploratory laparotomy where an unknown intestinal obstruction was thought to exist to find that on both as red gut and is associated with the feeding of milk substitutes. It appears to have increased in incidence in the abdomen and a loop of small intestine had become recent years and is more commonly seen in loose-housed, entwined around the strand, thus causing the obstruction. Severing the strand released the bowel and in both milk is uncontrolled and can be quite excessive. All cases the animal made an uneventful recovery. though the aetiology is uncertain, the pathogenesis is

thought to be the rapid fermentation of lactose in the

Tympanic intestinal colic

ileum, which leads to gas production and gross dilatation of the intestine, which then twists at the mesentery root.

Tympanic intestinal colic is by far the most common

Affected calves are normally three to six weeks old and

condition to produce colic signs. It can occur at any age

are presented showing severe colic signs with death fol-

and calves are presented with colic signs as frequently

lowing within 12 hours. It can also be a cause of sudden

as adult animals. The signs are of sudden onset, but in

death. Diagnosis is rare in the live animal, mainly because

lactating cows they are frequently observed at milking

of the rapid progression of the disease and it is not pos-

sible to differentiate it from tympanic intestinal colic.

At post mortem the findings are quite dramatic. The

Diagnosis

small intestines are grossly dilated with gas, are a bright-

As all the usual colic signs are present the clinical exam-

red colour and the whole of the intestinal mucosa is

ination will reveal normal rectal temperature, raised bright red. To detect the torsion, the post mortem must heart rate (80–90/minute) and normal mucous membrane performed with care because if the abdomen is fully branes. Abdominal sounds will be present, sometimes opened and the intestine allowed to spill out, the at an increased intensity. Rectal examination will reveal torsion will untwist and therefore not be detectable. faeces in the rectum and it is unlikely that any abnormality to the viscera will be palpated. It is important to torsion are those of severe colic. Rectal temperature ensure that the differential diagnoses mentioned above may be raised, the pulse weak and fast, mucous membranes are not present. Uterine torsion will only be present in branes are injected and the eyes sunken. Rectal examine pregnancy but photosensitization is fairly common ination will usually reveal multiple gas-distended loops (5–10 per 10 000) and must be eliminated from the diagnosis of small intestine in the right side of the abdomen and nosis by careful examination and palpation of any white

these may distend into the pelvic cavity. The site of the areas of skin.

mesentery twist will normally be beyond reach, but

In the calf, abdominal palpation should be performed. tense strands of mesentery may be palpable.

formed, as intussusception (p. 847) is relatively common and can sometimes be palpated. The diagnosis will be

Treatment

confirmed by response to treatment or spontaneous

Immediate surgery is indicated. Laparotomy is performed recovery within 24 hours.

formed under paravertebral anaesthesia in the right sub-

Treatment

lumbar fossa. Loops of distended bowel may protrude through the abdominal incision. The root of the mesen-

The most effective treatment is the intravenous administration will be located in the region of the left kidney and

istration of a spasmolytic such as hyoscine- N-butylbrothe direction of the twist should be determined. The

mide and dipyrrone or butylscopolamine bromide and torsion is corrected by manipulation of the intestines,

metamizole. Oral treatment with mild purgatives, such as some of which will need to be exteriorized to allow room linseed oil or liquid paraffin, has been used in the past but in the abdomen to correct the twist. This surgery is certainly unnecessary and not as effective as spasmolytics and may well be contraindicated.

the animal is not too severely shocked when the operation commences. Follow-up treatment with intravenous

Torsion of intestines (red gut in calves)

fluids and antibiotics should always be administered.

Torsion of the intestines around the root of the mesen-

Prolapse of intestines through mesentery

tery is extremely rare in adult cattle (1 per 10 000), but has been reported to occur spontaneously and the cause

This condition is usually only seen in adult cattle and is is unknown. It has also been reported to occur follow-more common than torsion at the mesentery root. Its

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annual incidence is still only approximately 1 per 10 000.

Usually, the dilated caecum will be palpable at the

The acute nature of the signs is identical to torsion at entrance to the pelvic cavity and many even protrude the mesentery root and rectal palpation will reveal gas-into the pelvic cavity. If the caecum has 'kinked' rectal distended loops of small intestine, which make it clinically indistinguishable. If untreated, death will occur detect the dilated organ.

within 12 hours.

Treatment

Treatment (see p. 1113)

Successful treatment using butylscopolamine bromide

Immediate exploratory laparotomy will reveal the and metamizole (Buscopan) has been reported in cases grossly gas-distended intestine, but it may be difficult to of dilatation where torsion is absent. It would be wise to distinguish from torsion of the intestines. Palpation of monitor the effect of treatment by rectal examination at the root of the mesentery must be performed first to distinguish the condition from torsion. One clue to the

instigated if there is no response or torsion occurs. If problem being one of prolapse of intestine through medical treatment fails or torsion occurs, surgical intervention will be the presence of normal intestine. Palpation of the gas-distended intestine will reveal that it is not too shocked and where the condition has not been protruding through a hole in the mesentery. The hole present for too long.

must first be enlarged and the intestines slowly fed back

A right-flank laparotomy is performed and, on explo-

through the aperture, after which the mesenteric

rupture is sutured. It must be emphasized that surgery

Palpation of the root of the mesentery should be carried

should only be contemplated if the animal is not too

out to try and determine whether torsion exists.

severely shocked. As death can occur quickly with this

The first stage of the operation is to siphon off the

condition, casualty slaughter is commonly advised.

fluid present in the caecum, thus reducing its volume to

a manageable size. It is possible in some cases to siphon off 30–40 l of dark, foul-smelling fluid. When as much

Caecal dilatation and torsion

fluid and gas as possible have been removed a purse-

Caecal dilatation is a distinct clinical entity in adult

string suture is used to repair the incision through which

cattle. Its occurrence is sporadic (approximately 1–2 per

the siphon tube was inserted and an attempt is made to

10 000) and usually occurs in early lactation, although

relocate the caecum back to its normal position. If the

the condition has been reported in bulls. The aetiology

caecum is kinked or twisted, removal of the fluid may

is thought to be related to high levels of volatile fatty

well allow the torsion to correct itself. The laparotomy

acids in the caecum, which originate from the rumen or

is closed in the normal way. Postoperative treatment is

from fermentation of undigested starch in the caecum.

usual with fluid therapy if dehydration is evident and

The fatty acids cause atony of the caecum and the gas

antibiotics to prevent peritonitis developing. This can

accumulates. Mild dilatation probably causes no signs,

be a rewarding operation and most animals make an
but severe dilatation will produce typical colic signs.
uneventful recovery.

However, many cases of caecal dilatation progress to
caecal torsion. Strictly speaking this is not a torsion but

Intussusception (see p. 1114)

due to the gross size of the dilated caecum and the large

Intussusception or telescoping of the bowel occurs in

quantity of fluid it contains, the distal end falls forward

adult cattle and calves. The annual incidence in adult

producing a kink in the organ. Occasionally, the weight

cattle is around 1 or 2 per 10 000, but may be more

of the distended caecum produces a torsion at the

common in calves. The condition is caused by strong

mesentery root and the colon, caecum and small intes-

peristaltic movements of the intestine and either the

tine are involved in a torsion rather similar to the

small intestine telescopes into small intestine or occa-

tion of the small intestines at the mesentery root.

sionally through the ileo-caecal valve into the caecum.

In calves the condition is usually a sequel to profuse

Diagnosis

diarrhoea, but this appears not necessarily to be the
Diagnosis is based on the presence of the signs of colic,
case in adult cattle. Some workers have suggested that
although sometimes the colic signs are quite mild. Bal-
a tumour or inflammatory growth in the lumen of the
lottement of the right flank will elicit copious splashing
affected part may be a causative factor in adult cattle.
and fluid sounds and the right flank may be noticeably

Signs

distended. Rectal examination will reveal an empty
rectum and if a reliable history is available the animal
When the intussusception first occurs there will be mild
will not have passed any faeces for 24 hours or more.
signs of colic. These signs frequently go unnoticed by

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the stockworker or if they are seen they are discounted
prompt, on no account should an attempt be made to
because they do not last for long and the animal makes
unravel the intussusception. Although this may well be
an apparent recovery. Two to three days later the

possible, the offending length of gut must be considered animal's milk production declines, inappetence sets in to be diseased because it will certainly re-form as an and the astute stockworker may notice the animal to be intussusception within 24 hours of correction. Some constipated. This is the most frequent time veterinary workers have suggested that if intussusceptions are not attention is sought.

removed surgically, but conservative treatment principles applied, the necrotic tissues of the intussusception Examination will reveal a normal rectal temperature, the heart rate raised to 80–120/minute and auscultation will slough out in 10–14 days and the animal will will reveal bowel stasis. A rectal examination will reveal recover. This approach to treatment is not to be re-an empty rectum or the presence of scanty bloodstained commended because, in the author's experience, faeces or thickened mucus. On questioning, the stock-animals that are not treated surgically will die.

worker may admit that colic signs were evident two to The intestinal anastomosis does require some surgi-

three days earlier.

cal skill but should not be beyond the majority of large animal surgeons. The technique has been performed quite satisfactorily on the farm.

Diagnosis

In many cases rectal palpation will reveal a hard,

Intussusception in calves

sausage-shaped mass in the right abdomen. The absence of any dilated organ and complete bowel stasis

In calves intussusception is a sequel to acute diarrhoea.

will indicate a strong likelihood that an intussusception

Signs of colic will be present in the initial stages of the is present, even if it cannot be palpated, although simple disease, but frequently the animal is presented because rumen indigestion may be difficult to differentiate. Pal- it is not defecating or is collapsed. Occasionally, the pation of the offending intussusception may be made intussusception can be palpated through the abdominal easier if the floor of the abdomen is raised using a pole wall and if the calf is not in an advanced state of shock under the ventral abdomen and lifted by two persons.

an exploratory laparotomy may be considered and the
The course of this disease is not as acute as other causes
offending lesion removed surgically and intestinal anas-
of intestinal obstructions; thus if the clinician is not
tomosis performed. Unfortunately, in calves the condi-
certain of the diagnosis, mild purgatives such as liquid
tion is most commonly encountered at post mortem.
paraffin or linseed oil may be administered orally and
the case re-assessed 24 hours later. These will do no

Diaphragmatic hernia

harm if an intussusception exists and, if the problem is
one of indigestion, faeces will be passed within 24 hours.
Diaphragmatic hernia has occasionally been reported in
It is important to instruct the stockworker to isolate the
cattle but the condition is rarely diagnosed and the inci-
animal in a clean pen for this period so that any faeces
dence is probably less than 1 per 100 000 per year.
voided will be observed. One of many problems with
Diaphragmatic hernia can be a congenital or acquired
loose-housing systems is that accurate history of
defect of the diaphragm with partial or complete entry

whether an animal is eating forage, defecating or urinating is frequently unavailable.

of abdominal organs (usually the reticulum) into the thoracic cavity. Congenital defects usually manifest before

If no faeces have been voided in the 24 hours of

the animal is 12 months old and acquired defects are

isolation an exploratory laparotomy must be

usually a sequel to traumatic reticulitis or trauma in contemplated.

adult cattle.

The most commonly prolapsed organ seems to be the reticulum and the result is interference with the motility

Treatment

ity and function of the rumen and reticulum. Colic signs

An exploratory laparotomy in the right sublumbar

will be noticed at the time of the rupture but they do

fossa should be carried out if the farmer is willing.

not persist. Signs similar to vagus indigestion may be

Failing this, casualty slaughter should be performed.

apparent, although low-grade pain in the posterior

Exploration of the abdominal cavity will reveal the

thorax/anterior abdomen region, rather like traumatic hard mass of the intussusception, although when it is reticulitis, has been noticed. Respiratory signs may be exteriorized it may not be recognizable as such. It may appear due to reduced thoracic space. A definitive appear more like a bloodstained tumour. Normal gut diagnosis is unlikely to be made without recourse to an should be identified entering and leaving the mass, exploratory laparotomy, although radiography may be which should then be surgically removed and intestinal useful.

anastomosis performed. If the mass is recognizable as Successful response to surgical correction has been an intussusception, as may be the case if diagnosis was reported, particularly in Indian buffalo, although this

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is only likely to be successful with relatively small there is a reluctance to move and walking sometimes ruptures and in cases where the vagus nerve is not instigates grunting. Appetite and milk production are damaged. The cow is positioned in dorsal recumbency

invariably depressed and rumination ceases.

and the incision made in the xiphoid area. Having retrieved the reticulum from the thoracic cavity the

Diagnosis

hernia is repaired with monofilament nylon or by the use of a fine nylon mesh.

The diagnosis of peritonitis can be difficult and is based on the history and clinical findings. However, the condition should be suspected in all cows that are pre-

Fat necrosis (lipomatosis, peritoneal

sented with the above clinical signs and a thorough fat necrosis)

examination may reveal which organ is responsible.

Lipomatosis or peritoneal fat necrosis occurs sporadically (annual incidence 1–5 per 100 000) in old cows. Ballottement of the lower right flank may reveal splash-sounds, indicating the presence of fluid in the peritoneum, and also may cause the animal to grunt. For a more detailed description of the diagnosis of traumatic

present any signs and the condition is detected at reticulo-peritonitis see p. 837. The history may also be routine rectal examination. Advanced cases of the condition will show signs of weight loss, underperformance abdominal surgery. If metritis is suspected, a rectal or vaginal examination will aid the diagnosis. Abdominal palpation findings when large hard masses can be palpated in the abdominal cavity. Occasionally, fat in the fluid, which will be foul smelling and contain a large number of white blood cells.

completely occluded and it is nearly impossible to carry out rectal palpation because of lack of space.

The severity and extent of the peritonitis are usually reflected in the severity of the clinical signs. If the peri-

There is no treatment and slaughter should be advised. On post mortem as much as 20–25 kg of

expiration. When only a limited area of the peritoneum hard necrotic fat may be present in the omentum and is involved, as in traumatic reticulo-peritonitis, pain will mesentery.

only be evident when the exact location is percussed.

It is important to attempt to determine the cause of the peritonitis in order to give an accurate prognosis

Peritonitis (see also p. 141)

and to determine the line of treatment that should be followed.

Peritonitis is a local or general, acute or chronic, inflammation of the peritoneal cavity. Peritonitis usually

Prognosis

occurs as an accompanying condition of other specific diseases, e.g. traumatic reticulo-peritonitis (p. 837) or

Localized peritonitis has a favourable prognosis, pro-metritis (pp. 519, 521). The most common cause of peri-

viding the offending organ can be identified and cor-tonitis is traumatic reticulitis followed by peritonitis

rective action taken. However, in acute generalized

as a sequel to metritis, dystokia or retained afterbirth.

peritonitis the prognosis can be poor, particularly if the

However, peritonitis may be a sequel to abdominal

peritonitis is a sequel to abdominal surgery, dystokia or

surgery, a ruptured abomasal or intestinal ulcer, penetration of a ruptured abdominal viscus. The prognosis will be

related to the severity of the clinical signs, the degree of

depression, the weakness of the pulse and the extent of

infected umbilicus in calves, rupture of the rectum,

of the signs of toxæmia.

uterus or large intestine, tuberculosis, liver abscesses

and chronic right- or left-sided displacement of the

Treatment

abomasum. Peritonitis can also be associated with

septicaemic conditions such as anthrax and calf

Treatment may first be directed to correction of the

septicaemia.

initial problem. However, the peritonitis itself will be

treated with large doses of antibiotics administered

intraperitoneally, intravenously and intramuscularly.

Signs

An initial dose of 5 g of benzylpenicillin and 5 g of dihydrostreptomycin is administered into the peritoneum. The clinical signs include a raised rectal temperature, which is frequently in the range 39–40°C (102.5–104°F), via the right sublumbar fossa, using a 2-inch 16-gauge needle followed immediately by 5 g of procaine penicillin G and 5 g of dihydrostreptomycin by intramuscu-

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lar injection and every 12 hours for three to five days. If systemic disease or other signs present, the cow must be removed from its present food source, isolated and given only dry feed, such as hay, to eat. Treatment with intramuscular injection at a dose of 50 000 iu twice daily for three days in addition to antibiotic therapy. Some workers have reported success using heparin by antibiotics such as streptomycin may be instituted, (Breukink, 1980). The author can report successful although the efficacy in these situations is not proven.

treatment using intraperitoneal injections of metronidazole. Spasmolytics may help and oral therapy with gut sedatives such as chlorodyne or adsorbents such as kaolin cannot now be used on animals intended for human consumption. are frequently used but again their efficacy is not

proven. The addition of glycine/electrolytes to the drinking water has more recently been suggested, but in cases where severe dehydration is present intra-

Diarrhoea

venous administration of 20–40 l of balanced electrolytes is indicated.

Diarrhoea in adult cattle is a frequent sign and is If the whole herd or group is affected, one must present in many diseases. It may occur sporadically, remember that lush spring grass will produce diarrhoea affecting only individual animals, or it may be present as will wet autumn grass in the UK. Wet grass silage will in a large number if not the whole of the group. If the also produce fluid faeces. In some areas of the UK and

whole group is affected, one must consider the feed or in other countries molybdenum toxicity is common, e.g. the possible presence of a virus infection as in winter on the so-called teart pastures of Somerset, and this will dysentery. Blood may be present, in which case dysentery produce severe diarrhoea in the whole grazing herd but is used to describe the sign. The diarrhoea may be can be corrected by the administration of copper in the very watery or even projectile as in redwater (pipe stem form of copper sulphate to the diets.

diarrhoea). The colour and odour may be distinctive.

An infectious cause of a whole group of cattle to be

Dark, foul-smelling, liquid faeces would indicate the affected with diarrhoea is winter dysentery (see p. 852); presence of occult blood and haemorrhage in the upper

infection with *Salmonella typhimurium*, *S. montevideo* small intestine. Pale, pasty-coloured faeces may indicate

or *S. goldcoast* may spread rapidly through a herd but rumen acidosis. Diarrhoea with air bubbles is free-the animals will also be pyrexia.

quently attributed to Johne's disease. Endotoxaemia

Thus diarrhoea is a sign of many disease conditions

from a coliform mastitis or metritis will produce dark,

of cattle but frequently enteritis will occur in individual watery faeces. Infections with agents such as *Campylobacter* spp. or BVD/mucosal disease virus will also remain a challenge for the bovine practitioner.

Salmonella spp. often produce dysentery. Intestinal parasitism will produce diarrhoea of varying intensity depending on the severity of the

Salmonellosis

problem, although this more commonly affects growing cattle than adults.

Infection of adult cattle with a variety of *Salmonella*

Non-inflammatory diseases, e.g. cardiac failure, lymphoma

spp. is frequently encountered in cattle practice (see

phosarcoma and systemic amyloidosis, also produce

Chapter 15). In the UK as many as 100–200 herds per

diarrhoea by increasing intestinal secretion into the

10 000 may suffer the disease each year where it affects

bowel lumen.

a considerable proportion of the herd. The incidence of

The clinician is frequently presented with an adult

sporadic salmonellosis, where only one or two animals
bovine where the only sign is an afebrile diarrhoea.

in the herd are affected, may be as high as 500–1000 per

Clinical examination may reveal sluggish rumen move-

10 000 herds. These sporadic occurrences are usually

ments but no other signs of indigestion or of abomasal

abortions due to *S. dublin*. A variety of *Salmonella* spp.

disease. Many of these cases will be the result of inges-

have been known to affect cattle but the two most

tion of toxic plants if the cattle are grazing or being fed

prominent are *S. dublin* and *S. typhimurium*. Salmo-conserved fodder and
frequently the cause may be a

nella newport, *S. montevideo* and *S. goldcoast* are less small batch of soiled or
spoiled silage.

frequently reported and sporadic outbreaks with other

Some cows with such acute diarrhoea die rapidly and

species occasionally occur. The most serious problems

post-mortem examination reveals a severe enteritis, fre-

are associated with enteritis and septicaemia, but spo-

quently haemorrhagic, but the examination for infec-

radic outbreaks of abortion with no concurrent septic-

tions or parasite counts proves unrewarding. One

caemia or enteritis are frequently encountered due to usually assumes that the cause of death is poisoning but

S. dublin. Epidemiologically, abortion appears different frequently the toxic agent is not discovered. When pre-formed from the enteritis and septicaemia syndrome and will

sented with individual cows showing diarrhoea and no be dealt with separately (p. 582).

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Epidemiology and aetiology

Signs

The principal signs produced by Salmonella infections The aetiological agents S. typhimurium and S. dublin are acute enteritis and septicaemia with a sudden onset

are those most frequently encountered although other of severe diarrhoea or dysentery. Frequently, the mucous species are reported sporadically. The epidemiology of membrane lining of the small intestine is passed with the S. dublin appears to differ from that of S. typhimurium. faeces and, occasionally, blood clots are present.

It has been known for some time that to establish S.

The rectal temperature is 40.5–41.5°C (105–107°F)

dublin carrier status in the adult cow there needs to and the cow is severely depressed. The severity of the

be damage to the liver and/or bile ducts. The most signs varies considerably. If the disease occurs at or frequent cause of liver damage is liver fluke and in near parturition, septicaemia sets in and mortality may areas where liver fluke is endemic, the incidence of *S. dublin* enteritis is more common. The source of *S. dublin* infections. *Salmonella typhimurium* pro-infection for *S. dublin* is therefore carrier cows, most duces morbidity varying from 10 to 70 per cent of the of which are suffering liver damage from liver fluke herd however mortality is generally not high, although it and if liver fluke is endemic in a herd, *S. dublin* will be higher if infection occurs at or around calving. may spread rapidly. In areas where *S. dublin* infection If infection with *S. typhimurium* or *S. dublin* occurs is unusually high, the organism can frequently be during late pregnancy, seriously affected animals will isolated from rivers, streams and ditches. It is difficult frequently abort. This is probably a result of the fever to postulate the role this contamination has in and septicaemia rather than the infection directly the spread of the infection as one would expect the

affecting the fetus or placenta. *Salmonella dublin* will watercourses to be infected if carrier cows exist in the

cause abortion without enteritis and septicaemia being area.

present but the epidemiology and pathogenesis is dif-

The carrier status probably also exists with *S.*

ferent. This will be discussed under *Salmonella dublin*

typhimurium, but this is much less common than with

abortion (see p. 582). Animals that abort following sep-

S. dublin. The majority of *S. typhimurium* outbreaks in ticaemia and enteritis frequently develop acute septic

adult cattle are of sudden onset and indicate a recent

metritis and peritonitis and frequently die.

introduction of the infection into the herd. The most

common source of infection is probably purchased con-

taminated compound feeds. These feeds often used to

Diagnosis

contain processed animal protein, which was frequently

The diagnosis is confirmed by the isolation of *Salmo-*

infected with *Salmonella* spp. Cross-infection to cereals nella spp. from faeces of affected animals. The faeces of also occurs when common storage bins and mixing

all cattle presented with acute enteritis accompanied

equipment are used. Cross-contamination of animal with fever should be sampled and tested for *Salmonella* feeds may also be caused by wild birds or rodents that spp. by bacteriological isolation. Affected animals may live in animal feed production premises. A further should be kept in isolation until the result is known.

source of infection for *S. typhimurium* is rivers or streams. In many rural areas public sewerage facilities

Treatment

do not exist and septic tanks or cesspits are used. If these do not function satisfactorily, raw or part-treated Antibiotics administered parenterally are used for the sewage finds its way into rivers. Human infection has treatment of salmonellosis. Due to resistance to some also been the cause of infection when cattle have grazed

antibiotics an in vitro antibiotic sensitivity test should be fields in which human defecation is known to have

performed as soon as possible. Resistance to penicillin, occurred or fields that are situated next to lay-bys on streptomycin, the tetracyclines and ampicillin is wide-busy main roads.

spread with *S. typhimurium*. Neomycin and framycetin
On occasions cows have been shown to be excreting
are frequently effective and resistance to chlorampheni-
S. typhimurium but yet not show signs of disease, i.e.
col and gentamicin is rare. The combination of trimetho-
they are carrier cows. The presence of a carrier cow
prim and sulphadiazine is also frequently used. There is
within a herd may go undetected and no problem exists
always some controversy regarding the use of antibiotics
with the rest of the herd. The reason why the infection
in treating salmonellosis in adult cattle. Septicaemic
does not spread in these situations is not clear. It may
cases and complications following abortion should
be that a minimum infective dose is required to estab-
always be treated with antibiotics. However, in a herd
lish disease or the immune system of the animals may
outbreak, some animals will be less severely affected
be depressed with a virus infection spreading within the
than others and will recover spontaneously without the
herd and this allows *Salmonella* infection to develop
use of antibiotics. Antibiotic therapy will need to be

into disease.

administered for three to five days.

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Oral rehydration therapy is indicated where dehydration is evident and if the dehydration is severe, intravenous administration of 30 l of balanced electrolyte solution will prove to be a valuable support to anti-infective therapy. Recovery time can be quite variable.

limited to the calves in endemically infected herds. In the

Cattle with mild enteritis will recover within two days,

case of S. dublin enteritis the main thrust in control follows—whereas severe septicaemic cases may take one to two

weeks to recover their previous appetite and will not

If this is successfully achieved, enteritis caused by S.

normally return to previous levels of milk production.

dublin will be infrequent.

Attempts to identify carrier cows within endemically infected herds are sometimes recommended. This is

Control

achieved by faeces sampling the entire herd and cul-

Once the diagnosis has been confirmed, it is important

turing the samples for Salmonella spp. Unfortunately, to limit the spread within the herd. Isolation of affected

Salmonella spp. are excreted intermittently in carrier animals will help but by the time cows are identified

cows and animals need to be sampled on several occa-

with enteritis, Salmonella organisms will be isolated sions, probably as many as six or seven, to be reason-from any site on the farm frequented by the cattle. Vac-

ably certain all carrier cows are identified. This

cination of non-affected animals should be considered.

procedure has proved to be neither practical nor nec-

In the UK the live vaccine prepared from an avirulent

essary in most outbreaks.

strain of S. dublin is no longer available. A dead vaccine prepared from formalin-killed cells of S. dublin and S.

Prevention

typhimurium (Bovivac S, Intervet Ltd) is available and To prevent infection with S. typhimurium may be diffi-has been used to control outbreaks of S. dublin or S.

cult because of the frequency in which it is found in

typhimurium. The whole herd should be vaccinated
animal feeds. Contamination of feed can also be caused
with two doses 21 days apart and booster injections
by rodents and birds. However, legislation has been
administered two to three weeks before calving to
implemented in the UK to supervise all protein
boost the passive immunity available to the calves. If
processing plants and to ensure the protein material is
exotic Salmonella species are identified consideration sterilized. In recent years
infections entering the
should be given to the use of autogenous vaccines using
herd via purchased feeds have become less frequent.
the same protocol as stated above. Control measures
Preventing infection spreading from the human
should also include monitoring the environment,
population to cattle can be achieved to some extent by
including the wildlife, and husbandry measures put in
the supply of clean wholesome water for cattle to drink.
place to reduce the risk of spreading infection. Cattle
For the effective control of a variety of infections
slurry should only be applied on land to be ploughed or

*cattle should not be allowed to drink from natural
on grass for conservation at least three to four months
watercourses. Effective fencing of fields may also be
before the grass is cut.*

required, particularly alongside main roads and lay-bys

*If S. dublin is identified as the causal organism, a repto prevent humans using
grazing fields as a lavatory.*

*resentative number of faeces samples (usually six to
twelve) should be examined for the presence of liver*

Winter dysentery

*fluke ova and if positive the herd should receive appro-
priate anthelmintic therapy.*

As the name implies, winter dysentery occurs during the

*It is essential when salmonellosis occurs on a farm
period of winter housing and affects cattle of all ages.*

that the utmost care should be taken regarding personal

It is obviously an extremely infectious disease because

hygiene. All the staff should be made aware of the

when it enters a herd it rapidly spreads through all

zoonotic implications and should not consume farm

animals in the herd. Also, once infected and the animals

milk during the course of an outbreak. In the UK, Sal-
have recovered, the herd will not usually experience the
monella isolation must be reported to the relevant
problem for four or five years, thus indicating develop-
departments of agriculture, who monitor the infection
ment of a herd immunity.

nationally. Very little action is taken against the farmer
Moreover, once one herd in an area becomes
unless there is a perceived human health risk, e.g. fresh
infected, it appears to spread rapidly to other herds
milk is being used for yoghurt, cream or cottage cheese
nearby. Perhaps this is a disease that can be spread by
production, in which case the public health authorities
veterinary surgeons! However, it is interesting that
may insist on pasteurization of the milk prior to pro-
there has been very little mention of its occurrence in
cessing. The question is frequently asked: should recov-
the UK during the last 15–20 years, whereas during the
ered cases be kept and will they become permanent
late 1950s and early 1960s it was frequently encoun-
carriers? Practice experience would indicate that in the

tered. It has been reported to be present in several

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countries in Europe and the northern States of the USA

However, a proportion of the cases are more severely

and Canada have experienced large outbreaks in past

affected and some may become weak enough to

years. Although the morbidity is almost 100 per cent,

become recumbent. Severely dehydrated animals will

mortality is normally absent. Milk production, however,

require intravenous administration of 20–30 l of bal-

may be reduced by up to 50 per cent and take up to two

anced electrolytes and electrolytes added to the drink-

weeks to recover and so it is of economic importance.

ing water should prove advantageous. Oral astringents,

such as 50 ml of 5 per cent copper sulphate administered

Aetiology

every 12 hours, may prove effective. Morphine and

chloroform (chlorodyne) reduce intestinal motility and

Campylobacter fetus var. *jejuni* was once thought to be absorbents such as kaolin or bismuth salts have also

the causative organism. But the speed of spread, the

been recommended. Oral sulphonamides have been short incubation period of around three days and the reported to be useful by some practitioners.

inability to reproduce the disease using cultures of C.

jejuni lead one to suspect the aetiological agent is a virus, which to date has not been identified. However,

Prevention

coronaviruses have been encountered in several out-

This is an extremely infectious disease and when it breaks and are now often considered to be involved in occurs in an area cattle or unnecessary personnel the causation of some outbreaks.

should not enter the farm. Veterinary surgeons should take the utmost care in thoroughly disinfecting their

Signs

boots and protective clothing before entering and leaving farms when this infection is present locally.

The main sign is of a severe, watery, dark brown diarrhoea with a foul-smelling odour. The faeces may be tinged with blood. The diarrhoea is sometimes

Bovine virus diarrhoea/mucosal disease

described as explosive or projectile. At first only one or two cows in the herd are affected, but within three or four days several cows will show signs and within two weeks all the older cattle on the farm will have become affected. The rectal temperature is usually normal and appetite is reduced in only a small number of more severely affected animals. However, milk yield may be reduced by up to 50 per cent and such animals can cross the placenta and damage the fetus.

In 1946, veterinary workers at Cornell demonstrated that a virus was the cause of a transmissible bovine diarrhoea and thereby named BVDV. The virus has since been reduced by up to 50 per cent and such animals may show signs of abdominal pain, e.g. colic, arched back or a 'hunched up' appearance may be apparent. The course of the disease in most cows is two to three

been identified worldwide as a most serious cause of days.

cattle disease, particularly reproductive disease.

Diagnosis

Aetiology (Figs 48.1 and 48.2)

The severity and speed of spread of the disease present no real problems in diagnosis except with the initial Infection with BVD virus is generally a mild or even cases, which may be misdiagnosed as a poisoning subclinical event, except on two occasions, i.e. infection (Chapter 54) or non-specific enteritis. Once several of the pregnant animal and in mixed infections, e.g. with cows are affected and the disease is occurring during other respiratory viruses such as respiratory syncytial the housed period, diagnosis is based on the clinical virus (RSV), infectious bovine rhinotracheitis virus signs present.

(IBR) or parainfluenza virus (PI3). Recently a more Bovine virus diarrhoea would involve fewer animals, virulent BVDV type 2 has been reported in North which would have pyrexia and characteristic oral

America.

lesions. Coccidiosis (see p. 282) normally affects

*Many isolates of BVDV occur but all appear to cross-
younger animals, but this can be differentiated by the
react with convalescent BVDV sera. There are two dis-
presence of tenesmus and the identification of oocysts
tinct biological forms of the virus called biotypes,
in the faeces.*

*distinguishable in tissue culture, cytopathic and
non-cytopathic. This distinction is important in the
pathogenesis and understanding of mucosal disease.*

Treatment

Cytopathic virus in tissue culture causes severe damage

*Most affected cattle recover spontaneously in two to
to the cells and complete destruction within 48–72
three days and treatments do not speed recovery.*

hours. The non-cytopathic virus causes no cell damage

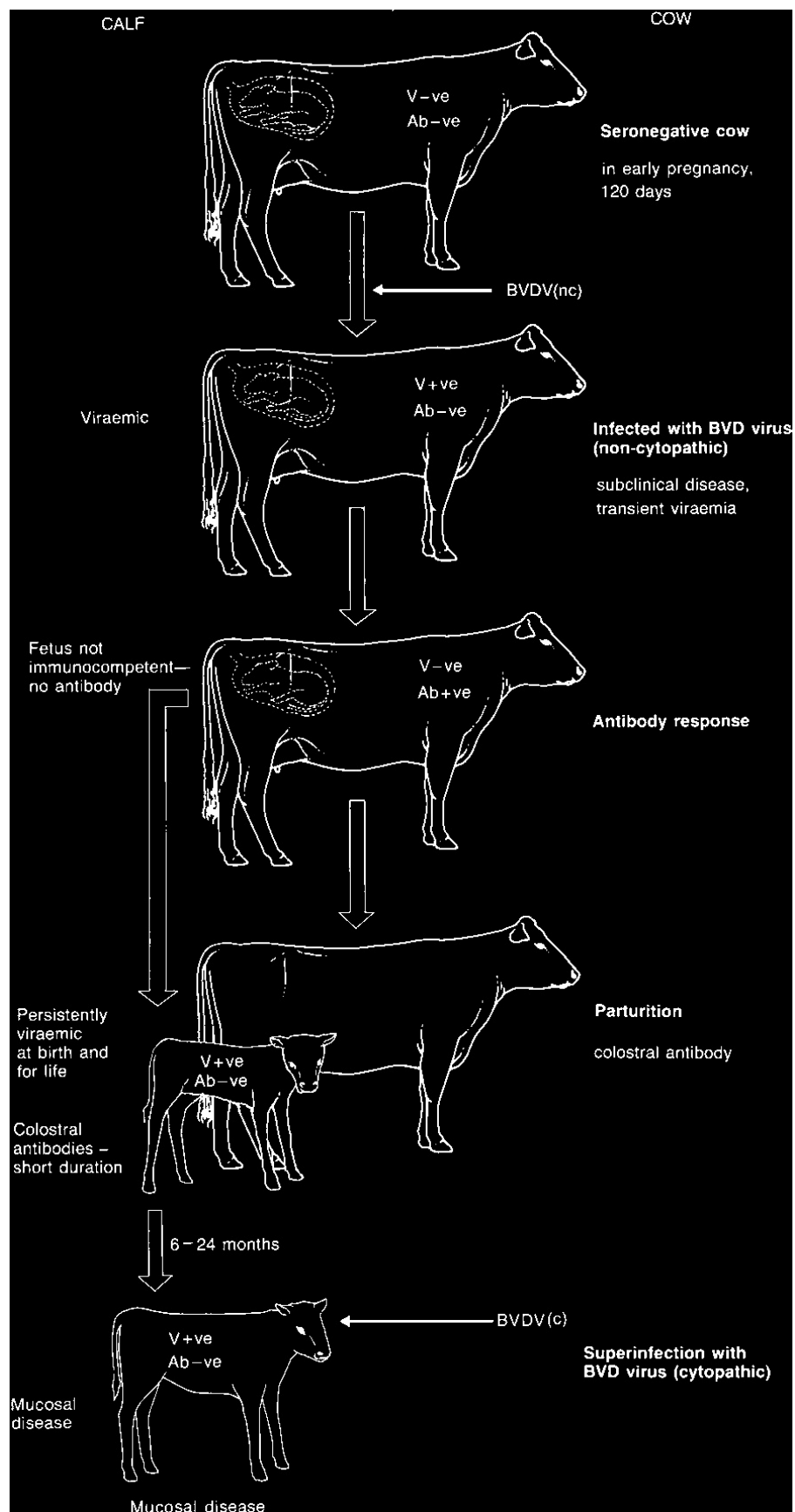
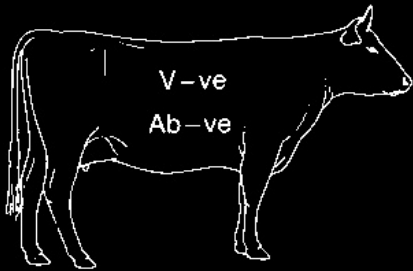
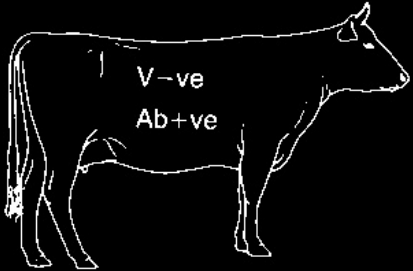
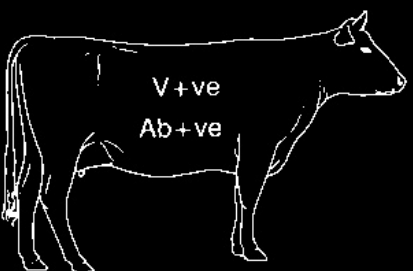
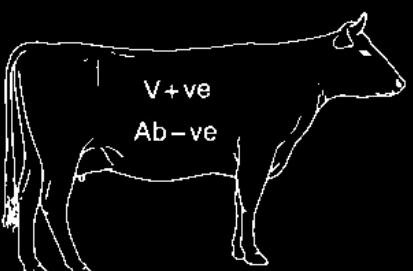


Fig. 48.1

Hypothesis for the aetiology of mucosal disease. V, BVD virus; Ab, antibody to BVD virus; nc, non-cytopathic; c, cytopathic (after Brownlie, 1985).

Category	Animal	Status	Result of exposure to BVD virus	Final antibody status
1	 <p>V -ve Ab -ve</p>	No previous exposure to BVD virus	Transient mild infection	+ ve
2	 <p>V -ve Ab +ve</p>	Previous exposure to BVD virus from 120 days gestation onwards	Immune	+ ve
3	 <p>V +ve Ab +ve</p>	(a) Acute viraemia presently sero-converting (b) Persistently viraemic; occasionally these animals may have low levels of antibody	Will become immune May later succumb to mucosal disease	+ ve ± ve
4	 <p>V +ve Ab -ve</p>	(a) Acute viraemia (b) Persistently infected with BVD virus	Will become immune May later succumb to mucosal disease	+ ve - ve

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Fig. 48.2

Combinations of BVD virus

and antibody in cattle and their significance (after Brownlie, 1985).

and is identified by staining with fluorescein-labelled and may result in abortion, mummification or early fetal BVDV antisera.

death. If the fetus survives until neonatal life, it may Non-cytopathic virus is the major biotype that causes have developed a state of immune tolerance and then disease, whereas cytopathic virus, isolated from field the virus will persist for life. The fetus at birth will be cases of mucosal disease, is usually only found as a virus positive, but seronegative to the persisting virus.

superinfection of a pre-existing non-cytopathic

Infection after 120 days will still cause damage to the virus infection. Bovine virus diarrhoea virus is shed fetus, but it does not become immunotolerant and in nasopharyngeal secretions and urine and perhaps viraemic. Such damage would include cerebellar by aerosol droplets. Faeces are a poor source of hypoplasia resulting in ataxic calves (see p. 900).

virus.

Infertility: Infection of the cow in early pregnancy will cause infertility due to embryo or early fetal death. Evi-Pathogenesis

dence of this has been seen in North America, where

Postnatal infection of the young or growing animal with

an infected bull was used on two groups of heifers. One

non-cytopathic virus is usually a subclinical event. In

group was seropositive and the other seronegative. The

most herds where the virus is present there is no

seropositive heifers conceived normally (about 70 per

disease-related problem. However, infection of the

cent pregnancy rate) but the seronegative heifers suf-

feronegative pregnant cow prior to 120 days of gesta-

tion, before the immune system of the fetus has become

rate) that lasted several weeks. These heifers eventually

fully developed, can result in disease. When the virus

conceived when they had seroconverted some six to

has crossed the placenta, the fetus becomes infected

eight weeks after the initial infection.

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Mixed infections: Bovine virus diarrhoea virus is

Diagnosis

immunosuppressive. Although infection of calves with BVD virus alone is generally subclinical if RSV, IBR or clinical signs and laboratory tests. Pyrexia, diarrhoea, P13 viruses are also present the pneumonia will be erosions in the mouth and on the muzzle and recent more severe than infection with respiratory viruses weight loss in an animal six to 18 months old should be alone (see Chapter 17).

highly suggestive of mucosal disease. In the live animal, clotted and EDTA blood samples and a nasopharyn-

Mucosal disease: Mucosal disease occurs as a result of geal swab should be taken and must be submitted to the

a calf that was born viraemic and seronegative become laboratory within 24 hours for both virus isolation and ing superinfected with cytopathic virus. Mucosal for BVDV antibodies. It is also helpful to submit fresh disease is generally sporadic in nature affecting young spleen, mediastinal lymph node, thymus and small in-cattle six to 18 months old. It is usual to find only one,

testinal tissue (particularly with Peyer's patch tissue) two or three cases occurring on any one farm at the for virus isolation. Bulk milk antibody levels can be same time; however, larger outbreaks can occur. The ascertained.

disease is characterized by weight loss, severe diarrhoea and inevitably death. Death from mucosal disease usually occurs two to three weeks after infection with Differential diagnosis cytopathic virus.

The main features of mucosal disease are mucosal erosions, diarrhoea and death. Foot-and-mouth disease (p.

Signs

693), malignant catarrh (p. 953) and rinderpest (p. 707) are the principal differential diagnoses. With foot-and- The first signs to appear are anorexia accompanied by mouth disease, morbidity is 100 per cent and vesicles reddening and erosions around the dental pad, along precede the mucosal ulcerations. With malignant the gingival border and under the tongue. These are fol-catarrh, corneal opacity is a feature; there is also gas-

lowed by reddening and erosion around the muzzle.
troenteritis and enlarged lymph nodes. Lymph node
The erosions are shallow, varying sizes and shapes and
enlargement is not a feature of mucosal disease. Rinder-
frequently coalesce. These are to be distinguished from
pest has vesicles preceding the erosions, morbidity is
ulcers following vesicular damage, e.g. in foot-and-
high and intestinal oedema and lymph node enlarge-
mouth disease (p. 693) or vesicular stomatitis (p. 710),
ment are common. Salmonella (pp. 225, 850) enteritis
which are deeper than mucosal disease erosions. Diar-
should not present a problem with differential diagnosis
rhoea then follows and sometimes lameness may be
as it generally affects either young calves or adult
apparent due to heat and reddening on the coronary
animals and there are no mouth lesions. Acorn poison-
band and erosions present in the interdigital space.
ing can look similar on post mortem, but biochemistry
Because of the sudden onset, diarrhoea is often the first
and histology of the kidney should distinguish it from
sign that is noticed although salivation is frequently

mucosal disease (see p. 951). Other poisoning events present. The animals are occasionally pyrexia. Weight may also be considered as a differential diagnosis, but loss is rapid and death inevitably occurs five to ten days after the characteristic oval erosions are generally absent. later. Acute infections in adult cows can lead to diarrhoea and agalactia.

Treatment

Necropsy

There is no effective treatment for mucosal disease. If the disease is suspected all efforts should be towards a post-mortem diagnosis so that effective control measures may be instituted. The post-mortem findings are usually strongly suggestive of mucosal disease. Oval erosions or shallow ulcerations may be seen in the buccal cavity, oesophagus, abomasum, the small intestine beneath the Peyer's patches and in the colon. Oedema and erythema in the intestinal epithelium may also be a feature. The prime

When a positive diagnosis has been made, the dam of

Control

When a positive diagnosis has been made, the dam of

sites are the oesophagus, small intestine and abomasum.

the affected animal and all the animals in the same

Care must be exercised in opening the small intestine,

group as the affected animal should be blood sampled

which must be opened at the mesenteric attachment in

and the blood cultured for the presence of virus. Per-

order not to incise through the lesions. Tissue culture

sistently viraemic cows invariably give rise to viraemic

can be used to grow virus from all the lesions, as well

calves so all dams of viraemic offspring need to be iden-

as from mesenteric lymph nodes, spleen, thymus and

tified. If infection is a recent introduction then several

tonsil.

calves in the same age group as the affected one may

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also be persistently viraemic. Most of these are likely to

‘sweeper’ bulls on breeding animals must include a

succumb to mucosal disease at some time in the future.

caution about the possibility of introducing BVDV into

When testing for BVDV, one should be aware of the

the herd.

possibility of sampling a calf that is only acutely infected and not persistently viraemic. To differentiate

Johne's disease (paratuberculosis)

this possibility all virus-positive animals should be retested at an interval of six weeks. Persistently

Johne's disease is a chronic, infectious enteritis that viraemic calves should be slaughtered. In the USA and results in progressive wasting and eventual death. The

elsewhere vaccines prepared from attenuated cyto-disease has been reported worldwide wherever ruminant pathic virus are available, but it is believed that these nants exist and the causal organism is the acid-fast bacterium. Inactivated BVD vac-

terium *Mycobacterium johnei* (paratuberculosis). This is now available in the UK for injection in cows

now often called *Mycobacterium avium* var. *paratuberculosis* prior to pregnancy (p. 1011) and in calves for respiratory

disease control (p. 1007).

between countries and within countries.

It is important to recall that surveys have suggested

There appear to be endemically infected farms on

that 1 per cent of adult animals in the national herd are which the incidence can be as high as two or three consistently viraemic. The retention of viraemic animals firmed cases every year, yet in the same district there in the herd is one strategy used to maintain herd immunity. However, there is always a risk that susceptible except occasionally in a purchased animal. In some cows in early pregnancy can become infected. There- areas of the UK the incidence has declined consider- fore, once persistently viraemic animals have been iden- ably over the last 30 years, although the incidence in a tified they should be slaughtered unless they can, with number of northern European countries appears to be certainty, be kept away from cows in early pregnancy. increasing. Recently concern has been expressed at the All newly introduced animals should be isolated and similarity of Johne's disease and Crohn's disease in screened for the presence of antibody and virus before humans. The possibility exists that some cases of mixing with the herd. Persistently viraemic animals

Crohn's disease may be caused by M. johnei.

should not be kept.

Pathogenesis

Control of BVD at herd level

Infection with M. johnei occurs in young calves usually Surveys have indicated that upwards of 60 per cent of

from their dams or contact with faeces of carrier cows.

UK cattle are seropositive to BVD infection and that

There follows a long incubation period of two to six

economic loss can be caused from infertility when

years during which lesions develop in the small intes-

seronegative animals are introduced into a seropositive

tine and the animals intermittently excrete the organ-

herd. This can be a common scenario with the introduc-

ism in the faeces. Not all infected animals progress to

tion of replacement heifers, whether home-bred or pur-

the disease state. Where the disease does develop, the

chased. Diagnosis of the seropositive status of the herd

organisms multiply and cause extensive lesions in the

can be achieved by testing bulk milk for antibody levels.

small intestine that produce overt clinical disease.

Recently, inactivated BVDV vaccines have become available in the UK. The vaccines have proven excellent

Signs

efficacy in preventing the fetus becoming infected if cows are vaccinated before they enter the breeding pro-

The usual presenting signs are profuse diarrhoea gramme. Recent reports would suggest that significant accompanied by gradual weight loss. Frequently, the improvements in fertility can follow the use of vaccination. stress of calving initiates the onset of signs and affected tion. Therefore vaccination of replacement animals cows are presented two to four weeks after calving.

before entering an infected herd is to be recommended.

Rectal temperature, appetite and ruminal contractions Booster vaccination at yearly intervals will probably be remain normal. Submandibular oedema is sometimes required so in time the whole herd will require vaccination annually. In advanced cases the weight loss leads to emaciation. The best control policy should be a combination of vaccinating breeding animals and

bubbles in the faeces.

eradicating all persistently infected animals.

Special regard should be given to bulls as they can

Necropsy

transmit BVDV in semen. Any veterinary inspection of

bulls must include a blood test to ensure freedom from

The main pathological features are thickening of the

BVDV. Furthermore, any advice about the use of

lower part of the small intestine, the ileo-caecal valve

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and sometimes the colon. The mucosal surface has a

stages of the disease. Antibiotics effective against

corrugated appearance. The organism may be present

Gram-negative bacteria, e.g. streptomycin, have been

on the mucosal surface or tissue sections of the intes-

used but without any long-term success. A short remis-

tinal wall may reveal both intracellular and extracellu-

sion of the diarrhoea is sometimes possible following a

lar organisms.

seven-day course of streptomycin and may be consid-

ered if for some reason it is not practical to cull the

Diagnosis

affected animal immediately. If dehydration is evident, rehydration with intravenous fluid therapy may allow

Any debilitating disease that results in emaciation may the animal to be sent for human consumption.

be confused with Johne's disease. However, the profuse diarrhoea, frequently containing bubbles, will distinguish the condition from weight loss caused by liver

Control

fluke (p. 276), liver disease, chronic traumatic reticulo-

As infection with the Johne's disease organism occurs peritonitis (p. 837) or malnutrition. Johne's disease

in calfhood the main plank in any control programme

should always be considered as a possible diagnosis

is to separate the calves from their dams immediately

when a cow, four years old or more, and recently calved

after birth and rear them completely separate from the

is presented with weight loss and chronic diarrhoea. The

adult herd. They should not be allowed access to faeces

history of the prevalence of the disease on the farm will

from adult cows at any time during the growing period

also be helpful. Diagnosis is best confirmed by the and all drinking water should be from uncontaminated demonstration of clumps of acid-fast bacteria in smears sources, namely mains water.

of faeces stained with Ziehl–Nielsen stain. As excretion In the UK and elsewhere a live vaccine (p. 1012) is can be intermittent, repeat testing is sometimes necessary. The faeces sample is best taken from the rectum vaccine used is licensed by the Department of the Environment, Food and Rural Affairs (DEFRA) and can using a gloved hand, scraping faeces from the rectal mucosal surface. Failure to detect the organism in faecal only be used on farms where a positive diagnosis has smears does not rule out the possibility of Johne's been made from faeces or post-mortem material. disease. PCR tests are now also used for the antigen, Calves are separated from their dams at birth and the but with the same provisos mentioned above. However, vaccine administered subcutaneously in the brisket the organism can usually be detected at post mortem in

area in the first seven days of life. A fibro-caseous stained impression smears of the mucosal surface of the nodule 2–5 cm in diameter is produced at the injection terminal ileum or in histological sections of the same site and this remains for life.

area of ileum.

Vaccinated animals will produce positive reactions to An ELISA test is now available which has a high sensitivity and specificity in animals with clinical disease, during tuberculosis testing. Usually, the avian reaction although the sensitivity is reduced in animals infected is greater than the bovine so differentiation from tuberculosis is possible if the comparative intradermal tuberculin and are a valuable herd screening test.

culosis test is used.

In some countries, Johnin is used as a diagnostic Vaccination in endemic herds has met with considerable success in reducing the incidence of disease in

venously. Injected intradermally, Johnin produces an
them, but not necessarily infection. However, to be suc-
oedematous swelling at the site of injection in some
cessful separation of the calves from the adults and
cattle that are infected with Johne's disease. When
good hygiene is necessary in addition to vaccination.
injected intravenously, Johnin will initiate a rectal tem-
perature rise of at least 0.8°C (1.5°F) 4–8 hours after
injection. However, as with the complement fixation

Tenesmus

test, the use of Johnin as an accurate diagnostic indica-
tor is not to be regarded as reliable.

Tenesmus or ineffectual straining to defecate is com-
If a definite diagnosis is required, the organism can
monly a sign of disorders of the pelvic cavity, the rectum
be cultured from faeces, portions of terminal ileum or
and some diseases of the alimentary canal.

mesenteric lymph nodes, but positive results will not be

Tenesmus can be produced by profuse watery diar-
available for six to eight weeks.

rhoea or dysentery, constipation, parturition, prolapsed

vagina or rectum, vaginitis, urethral calculi, cystitis, lipo-

Treatment

matosis of the pelvic cavity and coccidiosis. Tenesmus is

Treatment of Johne's disease is not to be recommended,

also an important sign of ragwort poisoning. Manual

as the clinical signs are only evident in the terminal

examination of the rectum or vagina will produce tenes-

Alimentary Conditions • 859

mus, which will be all the more severe if diarrhoea is

faecal material will have entered the pelvic and abdom-

present. Rectal or vaginal lacerations as the result of

inal cavities. The judicious clinician will advise immedi-

sticks or broom handles being inserted into the vagina

ate casualty slaughter in such cases.

or rectum by sadistic individuals will also cause tenes-

mus. In practice, the most commonly encountered

reason for tenesmus is a cow that continues to strain

Recto-vaginal fistula

after calving. This can be due to a second calf in the

Recto-vaginal fistulae are invariably the result of severe

pelvic canal, a retained afterbirth or lacerations to the

dystokia, usually in first-calving heifers. Tearing of the vaginal wall. The most effective treatment in these situations is to remove the calf if one is present or administer a local epidural anaesthetic.

the floor of the rectum and a fistula results. These A thorough examination is essential in cattle exhibiting tenesmus. Vaginal examination should be carried out using a vaginoscope to prevent further damage, recommended because pneumovagina or vaginal contamination with faeces produces infertility. Although apparent by separating the vulval lips. A rectal examination will not occur in cows so affected if natural service is used, it may be successful using artificial insemination because the semen is deposited in the

should be used because this procedure may exacerbate anterior cervix or the body of the uterus, thus bypassing the condition. Every effort must be made to identify the vaginal damage.

cause of the tenesmus so that corrective therapy can then be applied.

References

Diseases of the rectum and anus

Breukink, H.J. (1980) The effect of heparin in the treatment of general peritonitis in cows. In *Proceedings of the XIth Inter-*

Rectal prolapse

national Congress on Diseases of Cattle, Tel Aviv, pp. 1442–5.

Rectal prolapse is less common in cattle than in other

Brownlie, J. (1985) Clinical aspects of bovine virus diar-

rhoea/mucosal disease complex in cattle. In Practice, 7, species. However, it may occur as a result of prolonged

195–202.

tenesmus associated with vaginal lesions, or coccidiosis,

Gordon, P. (2001) A simple technique for correction of left dis-and it has been associated with laurel poisoning. *The*

placed abomasum. UK Vet, 5, 33–5.

condition appears to be more common in the Hereford

Jack, E.J. (1985) The cold cow syndrome – the Cornish experience. In Proceedings of British Cattle Veterinary Association Meeting, London, January 1985, p. 203.

Pinsent, P.J.N. (1977) The diagnosis of the surgical disorders Treatment of the bovine abomasum. Bovine Practitioner, 12, 40–

The rectum, if not excessively swollen, can be replaced
57.

under epidural anaesthesia and a pursestring suture

Pinsent, P.J.N. (1978) The diagnosis of the surgical disorders using umbilical tape inserted around the anal ring.

of the bovine abomasum. Bovine Practitioner, 13, 45–
50.

In recurrent cases a submucosal resection may be

Stober, M. & Dirkson, G. (1977) The differential diagnosis of required. abdominal findings (adspection, rectal examination and exploratory laparotomy) in cattle. Bovine Practitioner, 12, 35–9.

Rectal tears

Williams, E.I. (1975) The ‘reticular grunt’ test for traumatic Rectal lacerations are occasionally produced during

reticulo-peritonitis. Bovine Practitioner, 10, 98.

rectal examinations. They may also be produced by

sticks or poles being inserted into the rectum by sadistic individuals. If the tear is completely through the

Further reading

rectal wall repair via a laparotomy may be possible, although it is not easy to reach the pelvic cavity from a

Brownlie, J. (1985) Clinical aspects of bovine virus diarr-

laparotomy incision. Also, to succeed the repair must be

rhoea/mucosal disease complex in cattle. In Practice, 7, effected immediately the laceration occurs, otherwise

195–202.

Chapter 49

Respiratory Conditions

A.H. Andrews and R.S. Windsor

Acute exudative pneumonia

860

ing often at inspiration, particularly the latter. Cranio-

Aspiration pneumonia

860

ventrally, there may be moist sounds and there may be

Bovine farmer's lung

861

pleuritic rub (sandpaper-like) sounds in a few cases.

Bovine tuberculosis

862

Chronic suppurative pneumonia

864

Peracute pleuropneumonia

865

Necropsy

Diffuse fibrosing alveolitis

865

At post mortem there are dark areas of consolidation

Dusty feed rhinotracheitis

866

in the ventral parts of the apical and cardiac, and in

Fog fever

866

Thrombosis of the caudal vena cava

867

some animals, the thoracic lobes. The areas of pneumo-

Contagious bovine pleuropneumonia (CBPP)

868

nia may be small and scattered, but in more severe cases there are large areas of consolidation and, in some animals, abscess formation. Microscopically, there is

Acute exudative pneumonia

exudation and vascular congestion with the bronchioles and alveoli showing infiltration with neutrophils and

Aetiology

macrophages (Pirie, 1979).

This is thought in many cases to be a primary bacterial

condition and usually *Actinobacterium* (*Actinomyces*, *Diagnosis*

Corynebacterium) *pyogenes* (Gram-positive rods) can *Diagnosis* depends on the history of usually only a

be isolated, or in some cases *Mannheimia* (*Pasteurella*)

single animal being involved with pyrexia and respira-

haemolytica and *P. multocida* (Gram-negative short tory signs normally being evident.

rods) (Pirie, 1979).

Differential diagnoses involve chronic pneumonia but

normally the animals are less ill and several are affected.

Epidemiology

Inhalation pneumonia usually results in a very dull animal and also there is often a history of drenching.

This condition is not uncommon and is usually seen as respiratory disease in individual animals. It can be present in cattle of any age, particularly when there has

Treatment and control

been chronic pneumonia in the housing period. In can When treating, the affected animal should be isolated. be seen in dairy-bred cattle as well as in suckler animals, Antibiotic therapy with oxytetracycline, penicillin and both indoors and at grass. Individual cases usually occur streptomycin, ampicillin, amoxycillin, cephalosporins, but outbreaks can follow some form of stress. The con-sulphadimidine, and trimethoprim and sulphadiazine dition is one of sudden onset and is mainly differenti-for three to five days is usually successful. Most cases ated from acute viral pneumonia by the fact that it respond well to therapy, but a few cases relapse and some usually affects individual animals.

ultimately develop chronic suppurative pneumonia.

Prevention is by trying to ensure adequate ventila-

Signs

tion when housed and to avoid chilling.

The animal shows signs of suddenly going off its feed and is dull. There is an oculo-nasal discharge which may

Aspiration pneumonia

be mucoid or mucopurulent. The temperature is usually 40–41°C (104–107°F), respiratory rate is between 20

Aetiology

and 60/minute, usually with hyperpnoea. There is often some coughing but this is not pronounced. On auscul-

This is also known as inhalation pneumonia and

tation there are usually squeaks, humming and wheez-

although not a common condition, it still occurs too

860

Respiratory Conditions • 861

frequently. Obtaining an adequate history is important

Differential diagnoses include septicaemia, which has

and often the stockworker may realize what has hap-

fewer respiratory signs, enteritis but then diarrhoea is

pened, but will be reluctant to admit it or even that the

present, and acute exudative pneumonia, but in this

animal has been drenched. Obstruction or paralysis of

case there is no history of drenching and usually the

the larynx, pharynx or oesophagus may produce the animal is less dull.

problem, as with parturient paresis, or the rupture of a pharyngeal abscess or the products of laryngeal diphtheria. The signs will depend on the nature of the fluid

Treatment and control

introduced, the quantity and the bacteria introduced. If

If there is to be a hope of effective therapy, it must be

a large quantity is administered into the lungs, then

administered as soon as possible after the drenching

instantaneous death may occur. If the substance given

incident. The use of antibiotics or a sulphonamide is

is soluble, then absorption into the body is rapid

indicated and it is best to give the first dose intra-

because of the highly vascular nature of the lungs, and

venously. Thus oxytetracycline, amoxycillin, ampicillin,

few, if any signs will occur. Less soluble products will

sulphadimidine, sulphamethoxypyridazine or sulpha-

result in a varying degree of toxæmia and respiratory

pyrazole can be used. In exceptional circumstances

signs, which are often fatal, after between one and three

where allowed chloramphenicol might be indicated.

days.

Therapy should be continued in most cases for about five days. In addition, fluid therapy may be required.

Signs

The animal should be encouraged to eat and drink. It should be kept on its own in a well-bedded, airy pen.

In the peracute form death occurs rapidly after drench-

Control is by ensuring that all drenching and dosing

ing. However, in the acute form only one animal is

is undertaken slowly, allowing the animal time to

usually affected and there is a history of drenching.

swallow.

Signs develop rapidly and include a varying degree of dullness and inappetence, a cough and tachypnoea. The

temperature is usually elevated to about 40°C (104°F)

Bovine farmer's lung

and on auscultation there are areas of dullness present,

normally in the cranio-ventral parts of the lungs, and

Aetiology

moist bubbling and crackles may be heard in the area.

There is often also a pleuritic rub sound and some
This is a form of chronic atypical interstitial pneumo-
degree of thoracic pain. If the condition progresses, the
nia. The condition appears to be a chronic reaction to
signs of dullness and anorexia become more pro-
certain fungi found in badly made hay, such as Micro-
nounced, and there may be a fetid odour to the breath.
polyspora faeni.

In the subacute form there are few signs present
except for episodes of coughing and tachypnoea fol-

Epidemiology

lowing the introduction of the fluid. Some animals will
survive the immediate episode and become chronic

The problem is quite common during the winter in
cases. These will show ill-thrift and intermittent bouts
housed cattle fed poor quality mouldy hay or straw.
of respiratory problems.

More cases occur in the wetter western parts of Britain
and in other countries with high rainfall. Where much
rain falls in the summer months, hay may need to be

Necropsy

baled at very high moisture contents. This allows over-

At post mortem there is often an acute exudative or

heating to occur and thermophilic microflora then pre-

gangrenous pneumonia of the ventral parts of the

dominate. Disease is often only seen in adult cattle and

apical, cardiac and usually also the diaphragmatic

in some cases the farmer will also have farmer's lung.

lobes. In some animals there is extensive suppurative

In Britain this is defined as an industrial injury and is

necrosis.

considered to be due to the inhalation of dust from

mouldy hay or other mouldy vegetable produce. It

results in a defect in gas exchange due to a reaction in

Diagnosis

the peripheral parts of the bronchopulmonary system.

Diagnosis is helped if a true history is obtained and is

In cattle the condition is usually a herd problem but

indicative that the condition is present. Usually only a

occasionally a farmer will consider that there is sudden

single animal is affected and the signs are of sudden

onset in one animal. In such cases the examination of

onset. The respiratory signs are severe and there is other animals will show varying lesser degrees of the usually a leucopenia and neutrophilia present. problem.

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Signs

Control involves improving hay-making, which may mean the use of hay additives to upgrade hay quality.

The acute signs follow housing and there is often a Otherwise, the provision of silage might be useful but sudden onset of dullness, a fall in milk yield and a this does normally mean investment in new machinery decreased appetite. The animal shows respiratory signs, for producing the conserved roughage.

normally including some respiratory distress, and coughing. On auscultation there are crackles over the cranio-ventral parts of the lung. Although some cases

Bovine tuberculosis

are pyrexia, most animals will have a normal rectal temperature.

Aetiology

In the chronic form there is progressive weight loss and coughing, often with the production of green sputum. This is infection with Mycobacterium bovis (Gram-positive, acid-fast rods).

resolves during the summer months with outside grazing. Occasionally, such animals will develop a

Epidemiology

sudden crisis following a stress such as calving, a sudden

At one time the condition was very common in many countries. However, following tuberculin testing, pasteurization of milk and adequate meat inspection the disease is uncommon in most countries today, but is still seen periodically mainly in dairy herds. In most cases thoracic pain or alteration in the resonance of the thorax. Auscultation may produce harsh crackles over infection breaks out in the growing heifers or younger animals. The cranio-ventral aspects of the lung and in some cases

cows. The condition is still prevalent in south-west
there are widespread whistles, squeaks and wheezing. It
England, but is now being increasingly seen in other
is uncommon for animals to die of the condition unless
areas including the south Midlands, north-west England
there are complications.

and Wales. In many regions infection has reappeared
and is associated with the finding of tuberculosis in the
Necropsy

European badger (*Meles meles*). However, in many
cases infection can follow the purchase of a carrier

At necropsy all lung lobes may be affected and there is

without overt signs. Infection of deer with *M. bovis* can often overinflation of the
peripheral acini. Small grey-also spread disease and in New Zealand a problem

green foci tend to be present in the lobules. Histologi-

occurs with the brush-tailed possum (*Trichosurus*

cally, the alveolar walls show interstitial infiltration with

vulpecula). The organism is killed by sunlight, but is plasma cells, lymphocytes
and macrophages. Another

resistant to desiccation and can survive in a wide range

change is bronchitis obliterans and also epithelial granu-

of acids and alkalis. It is also able to remain viable for

lomata can occur.

long periods in soil that is moist and warm. In cattle faeces, M. bovis can survive for as little as a week or as long as eight weeks. Man can occasionally be infected and the disease can occur in goats and pigs, and very Diagnosis depends on a history of occurrence in wet occasionally in horses and sheep. Very occasionally areas and feeding poor quality mouldy hay in winter.

cattle can be infected with Mycobacterium tuberculosis, The problem improves in the summer. Signs include

usually because people tending or in close contact with loss of weight with the respiratory disease. An intra-the cattle are infected.

dermal skin test with Micropolyspora faeni produces a When infection is by inhalation, a lesion often occurs

reaction 4–6 hours after injection. On serological exam-

at the point of entry and the local lymph node. When

ination precipitating antibodies to Micropolyspora

ingestion is the route of entry, alimentary lesions are

faeni are found but they may also be seen in unaffected rare but lesions may be present in the tonsils, pharynx-cattle within the same herd.

geal or mesenteric lymph nodes. Lesions may then

disseminate from the primary areas to others.

When the badger is involved, most infection is

Treatment and control

thought to be by ingestion, but a higher infection level

The use of long-acting corticosteroids may help reduce

is necessary to establish alimentary than respiratory

signs. Where mouldy hay or straw has to be fed or used

infection. In most cases the lesions are respiratory and

for bedding then it must be shaken out outside before

are thought to be due to the inhalation of ruminal gases.

being offered to the animals. As human problems can

The organism can be present in sputum, milk, faeces,

arise, a face mask should be worn.

urine, vaginal and uterine discharges and any discharg-

Respiratory Conditions • 863

ing lesions. Entry is usually by inhalation (especially if

infection is active and if cases are 'open' and therefore

housed) or ingestion (when outside or badgers are the

likely to infect other animals. Active infection is desig-

source of infection). Drinking infected milk can infect

nated by lung infection with limited encapsulation and

the calf. Signs can occur in very young calves (under a hyperaemia. This categorization is now thought to be month old). Intercurrent disease in the herd such as erroneous and it is considered that most lesions are BVD may exacerbate the problem (Monies, 2000). potentially infective. Other organs often show small, Once in a herd, infection probably spreads from cow to transparent, shot-like lesions and these may also be cow by inhalation. However, spread from cows to calves present in the lymph nodes. Tuberculous cystitis and may be via the milk. Occasionally, intrauterine infection metritis tend to be open cases. Closed infection is seen has resulted from a coital transfer.

as discrete lesions enclosed within well-developed capsules. The enclosed pus tends to be caseous and yellow or orange in colour. In tuberculin reactors the apparent
Signs

absence of gross lesions of tuberculosis – so-called non- Various body systems can be infected. Often signs are visible lesion (NVL) reactors – does not necessarily few and usually are confined to the respiratory tract.

indicate that the animal is uninfected.

There is a soft, productive, chronic cough occurring once or twice at a time. It can be elicited by pressure

Diagnosis

on the pharynx. If the condition continues there is a marked increase in the depth and rate of respirations

Diagnosis depends on the history of an area where as well as dyspnoea. In advanced cases, areas of dull-tuberculosis occurs in cattle, badgers or other wildlife.

ness in the chest are heard on auscultation or percus-

The signs often result in chronic respiratory lesions with

sion. In other cases there are squeaks and whistles. A

loss of condition and a soft, productive, single cough.

snoring respiration can occur.

The comparative tuberculin test is useful. It uses avian

The alimentary form is unusual. There are few signs

(0.5 mg/ml) and bovine tuberculin purified protein

but occasional diarrhoea occurs. Bloat can arise

derivative (1.0 mg/ml) injected into the neck skin. There

through enlargement of the mediastinal and bronchial

is a greater skin thickness increase in bovine than avian

lymph nodes. Bone can be infected and meningitis tuberculin. Interpretation depends on whether there is occurs in calves.

no history of reactions, one or more reactions without Mammary involvement these days tends to be rare confirmation at post-mortem examination or a herd with but results in udder induration and the supramammary a recent history of reactions confirmed post mortem.

lymph nodes are enlarged. The udder form can be a Johne's disease (p. 857), skin tuberculosis (p. 886) or serious potential source of spread to humans. The avian tuberculosis can result in false positive bovine uterine form is also uncommon. Swelling of various tuberculin reactions but usually the avian reaction lymph nodes can occasionally be seen, and abortion increases more than the bovine. False negatives occur may sometimes occur.

following protracted infection, desensitization follow- A generalized form can occur with signs following ing tuberculin testing, early cases of infection, old cows calving. There is a progressive loss of condition with a

and those animals recently calved.

variable appetite. There may be a variable rectal tem-

The single intradermal test is used in many countries.

perature but usually it is only about 39.7°C (103.7°F).

Its main disadvantage is that it will give reactions to

The animals are more docile than normal but still bright

avian tuberculosis, skin tuberculosis or Johne's disease.

and alert.

A short thermal test can be used by injecting tuberculin

subcutaneously and measuring the animal's tempera-

ture every 2 hours. A rise in temperature of 1°C (1.8°F)

Necropsy

is considered significant. Intravenous tuberculin also

A focus of infection occurs within a week of bacteria

results in a temperature rise. The Stormont test has

entering the cow and, after the third week, calcification

been used to detect disease in infected cattle. Various

can occur. Depending on the route of entry, and where

serological tests have been used and recently the

the condition becomes generalized, one or several

enzyme-linked immunosorbent assay (ELISA) test has

lymph nodes may contain tuberculous granulomas. In shown promise. A gamma interferon test can be used the respiratory system it is the mediastinal or bronchial to determine infected cattle.

lymph nodes that are involved, possibly with abscesses in the lungs. The pus is thick, cheese-like and yellow or Differential diagnosis

orange in colour. Sometimes the pleura and peritoneum contain nodules.

Differential diagnosis includes enzootic bovine leuko- In practice, an attempt is made to determine whether sis (Chapter 43b) but this can be detected by serology.

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Chronic lung abscesses can cause problems in diagno-

More usually the animal becomes progressively

sis. Traumatic reticulitis (p. 837) may produce similar duller and thinner, with a fall in milk yield and inter-signs but there is usually a history of an acute attack.

mittent pyrexia, up to 40°C (104°F). A cough is usually

Chronic pericarditis (p. 731) can present problems but present with the production of mucus and there is a

will result in a jugular pulse and muffled heart sounds variable degree of tachypnoea. Thoracic pain may be and endocarditis (p. 726) cases usually produce a obvious by an abduction of the elbows and reluctance murmur. Contagious bovine pleuropneumonia (p. 868) to move, but in other cases it is only discernible on bal- can cause problems but can be differentiated by a com- lottement. On auscultation there are usually whistles, plement fixation test. Lymph node enlargement due to squeaks and wheezing sounds in the cranio-ventral part actinobacillosis (p. 823) may be difficult to detect but of the chest and there are often areas of dullness. can be done with a tuberculin test.

Necropsy

Treatment and control

If the main lesion at post mortem is a bronchopneu- monia, there is usually marked consolidation of the Treatment is not usually undertaken because of the cranio-ventral parts of the lung, with exudate filling the chronic nature of the disease and its potential zoonotic bronchi and bronchioles. On histological examination,

effects. Control in many countries, including North America and Europe, is by tuberculin testing and the main problem is a bronchiectasis, often bronchi in slaughter of reactors. Hygiene standards need to be the cranial and middle lobes, with dilated air passage-upgraded and efficient meat inspection and tracing ways, contain mucus and fibrous tissue. In severe cases back to the farm of origin is useful. Research work on the histological sections show complete destruction of vaccine production is being undertaken and may be the alveolar tissue. When lung abscesses are the main directed towards vaccination of wildlife such as badgers feature, these are usually found in the ventral lung and possums.

border. Necrotic tissues and pus-containing structures are found within a fibrous capsular wall.

Chronic suppurative pneumonia

Diagnosis

Diagnosis is based on a history of a chronic loss of con-

Aetiology

dition with respiratory disease in a single animal with

Various initial causes may result in one or more

signs such as pyrexia, thoracic pain and cough.

pathological conditions such as bronchopneumonia,

Differential diagnosis needs to include acute pneu-

bronchiectasis and pulmonary abscesses. These are

monia (p. 860), which may be in a single animal or

often encompassed by the term chronic suppurative

several animals; salmonellosis (p. 850; Chapter 15), but

pneumonia.

at this age there is usually diarrhoea; infectious bovine

rhinotracheitis (IBR) (p. 289) infection, but this usually

results in a marked conjunctivitis. Inhalation pneumo-

Epidemiology

nia (p. 860) on the other hand has a specific history and

tuberculosis (p. 862) will probably have a history of

Most cases occur in adult cattle rather than those still

herd infection, whereas malignant catarrhal fever will

growing. It is, however, a very common cause of res-

involve ocular lesions and enlarged lymph nodes, etc.

piratory signs in the individual animal. Often there has

been an outbreak of acute pneumonia in the history.

Treatment and control

Although most cases seem to progress slowly over a period of weeks or months, the odd case will appear to

Often therapy is of limited use. Any treatment may be of sudden onset, due to a rapid exacerbation of a need to be prolonged for 10 days to two weeks or more. suppurative area in the chest.

Antibiotic therapy with amoxycillin, ampicillin, oxytetracycline, penicillin and streptomycin, sulphadimidine, or trimethoprim and sulphadiazine may be helpful.

Signs

Most cases that respond are likely to break down again Severe signs of disease include a sudden marked loss of and so infected animals should be slaughtered when condition with dullness, obvious thoracic pain, pyrexia convenient.

(40.5°C; 105°F). In some animals there is halitosis due

Control involves culling animals that have had pre-to a necrotizing bronchopneumonia and pleurisy. Death vious bouts of respiratory disease and ensuring all cases

in these animals often occurs within a few days.

are treated early and thoroughly.

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Peracute pleuropneumonia

Diagnosis

Small numbers are affected with severe disease, which

A respiratory problem was described in the 1990s in

is often fatal. There is no apparent precipitating factor

Great Britain which appeared to be a relatively well-

except for calving. Post-mortem examination allows

defined syndrome and which at post-mortem examina-

diagnosis.

tion showed a pleuropneumonia (Harwood et al. , 1995).

While it looked like contagious bovine pleuropneumo-

nia, the history and epidemiology were not right and

Differential diagnosis

Mycoplasma mycoides subspecies mycoides was not Contagious bovine pleuropneumonia (CBPP) is an

isolated.

obvious differential and in many countries, as in Britain,

CBPP is notifiable and so it will be necessary to report

cases to the authorities (see p. 873).

Aetiology

Routine bacteriology of lungs when positive routinely

Treatment

*isolated Mannheimia (Pasteurella) haemolytica, usually of serotype A1.
Arcanobacterium (Actinomyces) pyo-Large doses of antibiotics, often with non-steroidal*

genes has been isolated from the necrotic areas within anti-inflammatory agents, can help in a few cases.

the lung. No further consistent bacterial or viral synergism has been recognized.

Control

At present there is no real advice to offer in herds

Occurrence

which for any reason have to buy in animals.

*All problems appear to have involved adult animals
and calf respiratory problems on the farms did not*

Diffuse fibrosing alveolitis

*appear to be unusual or of high incidence. Usually one
or a small number of animals are affected and most of*

Aetiology

these die. The remainder of the herd do not appear to

show any signs of disease. Animals in the immediate

The cause is unknown but many cases occur in animals

post calving period were most commonly infected.

with chronic bovine farmer's lung and have precipitat-

There was a history of purchase of cows and/or heifers

ing antibodies to Micropolyspora faeni. However, it is within the last 12 months on all farms and in several

probable that there are other precipitating causes as

there were imports from Europe as well as from the

some cattle do not possess antibodies to this organism.

United Kingdom. There was no history of recent transport or movement of the affected animals.

Epidemiology

The condition affects individual animals and is uncom-

Signs

mon although more frequently seen in herds with a

history of bovine farmer's lung. Both dairy and suckler

Severe respiratory problems are seen with dyspnoea,

cows are affected and cases can occur indoors or

hyperpnoea and tachypnoea. Most animals will die or

outside, particularly in animals older than six years. The

are humanely destroyed regardless of any antibiotic condition is usually a progressive problem and may regime. There are variable lung signs, including pleural actually start following a stress such as calving. The contractions, etc.

condition has normally been present for weeks or months before advice is sought and the animal will have lost condition with coughing or respiratory signs when sub-

Pathology

jected to mild exercise. Congestive heart failure occurs. The thoracic cavity contains severe changes with very in about 12 per cent of cases.

obvious fibrinous or fibrous pleurisy. There are strong attachments between the thoracic wall and the lungs;

Signs

pleural deposits can often be 1 cm thick. There are large amounts of pleural effusion and pulmonary. Affected cattle are bright and do not have a raised temperature or pulse rate. The appetite is good but there is lobular oedema, with parenchymal congestion and

a progressive loss of condition. The respiratory signs consolidation.

tend to be quite severe, with a persistent cough always

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present as well as tachypnoea and hyperpnoea present

Table 49.1

The fate of various-sized particles entering the even in the resting animal. On auscultation of the chest, respiratory system.

rhonchi (whistles, squeaks, wheezing) are heard over both lungs and crackling sounds in the cranio-ventral

Particle Fate

size (mm)

chest. There is no thoracic pain.

Depression and inappetence only occur in the late

>10

Removed in nasal passages

stages of the condition with congestive heart failure

2–10

Deposited at varying levels in respiratory tract,

resulting in subcutaneous oedema and an increased

*but above alveoli. The smaller the particle,
heart and respiratory rate. The liver may be palpably
the further down the respiratory airways it is
enlarged and there may be diarrhoea.
deposited. Removed by mucociliary action*

1–2

Deposited in alveoli

0.5–1

Exhaled with air

Necropsy

<0.5

Deposited in alveoli due to diffusion forces

*At necropsy alveolar changes predominate but there
may be bronchitis with excessive thick mucus in the
bronchi. There is thickening and fibrosis of the alveolar
walls. Histologically, large numbers of mononuclear*

Signs

*cells are seen in the alveolar air spaces. There may be
Following feeding or bedding, there is the sudden onset
hyperplasia of type 2 pneumocytes or metaplasia of the
of coughing. The cough tends to be dry and can be*

alveolar epithelium so that it contains ciliated and single or paroxysmal. Several cattle are normally mucus-secreting cells. Pulmonary hypertension can be affected. The animals are otherwise bright and alert; they eat well and there are no abnormal lower respiratory sounds. Respirations are normal in rate and extent,

Diagnosis

and temperature is normal. There is conjunctivitis and usually a copious ocular and nasal discharge, which is mucoid but sometimes slightly purulent. Diagnosis depends on the history, i.e. a single animal, with gradual loss of condition with respiratory signs present at rest, coughing, no thoracic pain or fever and a bright animal.

Treatment and control

Treatment involves replacing the feed or bedding used.

Treatment

Otherwise dampen down the feed before giving it, or molasses can be added to it. In the case of bedding, new

As the cause is unknown, little can be done to alleviate

bales should be opened up outside before the cattle are the problem. However, corticosteroids can reduce the bedded.

cellular changes in the lung. Casualty slaughter of the Control is by not feeding dusty hay. If the feed is animal should be undertaken before the loss of condition is too severe.

added to it. Dusty bedding should not be used. As the particles affecting the animals can affect humans, it is advisable for workers to wear face masks.

Dusty feed rhinotracheitis

Aetiology

Fog fever

Particles of different sizes meet varying fates in the res-

Aetiology

piratory system following inspiration (see Table 49.1).

Most of the particles will be in the nasal passages or the

This is a form of atypical interstitial pneumonia.

trachea, bronchi and bronchioles. The condition results

Although not fully authenticated, the condition is con-

from the introduction of dry, fine-particled feed, or very sidered to be a toxicosis following the ingestion of large dusty bedding.

quantities of l-tryptophan.

The introduction of a dusty dry feed to animals indoors causes the problem. The signs occur most fre-

Epidemiology

quently in the hour or two following feeding. Removal of the feed causes recovery in a few days. The condition

The condition is seen in cattle over two years old, par- occurs most commonly when the relative humidity is ticularly those in suckler herds, and affects several cattle low.

to a varying degree at the same time. Often the cattle

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have been receiving little nutrition and are put onto

Cattle slaughtered in the later stages do not usually

a more lush pasture in the autumn (September to show haemorrhages of the respiratory mucosa. There is November). The field may have been top-dressed with an overall pale pink colour with variable amounts of

*a nitrogenous fertilizer. The condition is normally seen
interstitial emphysema.*

*within two weeks of entry to the new pasture. The
Hereford and Hereford-cross breeds seem to be par-*

Diagnosis

ticularly susceptible.

It is thought that l-tryptophan in the grass is ingested

Diagnosis involves the history of a group condition,

and metabolized in the rumen to indole acetic acid

mainly in suckler animals moved to a lush pasture in

*(IAA), which is decarboxylated by Lactobacillus spp. to autumn. The signs help,
particularly the acute respira-produce 3-methyl indole (3MI). This metabolite
can*

tory signs with little to hear on auscultation, no cough

enter the blood and is usually acted upon by the mixed

and the animals being more tranquil than usual. Post-

function oxidase system to produce indoles and other

mortem findings indicate the condition, with pulmonary

metabolites in the urine. 3-Methyl indole can cause the

oedema and emphysema.

destruction of pulmonary cells such as type 1 pneumo-

Differential diagnoses include husk (p. 272), but a

cytes and monociliated bronchiolar secretory cells, cough is present and there would be a history of no resulting in various pathological changes. Mortality in vaccination. Pneumonic pasteurellosis (p. 286) would severely affected animals can be high (up to 75 per produce pyrexia and a mucopurulent discharge. Nitrate cent) but usually only a small number (5 per cent) are poisoning (p. 950) would produce some signs but the so involved.

blood would tend to be brownish and the urine contain methaemoglobin. Infectious bovine rhinotracheitis (p. 289) would usually involve pyrexia and a loud explosive

Signs

cough. Thrombosis of the caudal vena cava (see below) Several animals will show signs but the degree will vary would usually involve a single animal and eventually widely and often the farmer only notices one to be ill haemoptysis would occur. Brassica spp. (p. 941) poisoning at the start. The cattle tend to be much quieter and soning would have a different history of feeding and more approachable than normal and to have a sleepy

would usually be later in the autumn.

or tranquil expression. The respiratory signs are usually of distress but vary in degree. Coughing is normally

Treatment and control

little heard.

In the severe form there is the sudden onset of dys-

Treatment is to remove the cattle from the incriminated

pnocoea with a loud respiratory grunt, mouth breathing,

pasture. Most other treatment tends to be empirical.

and often the animal froths at the mouth. Auscultation

Interference with a severely distressed animal may

reveals little considering the severity of the illness,

result in its death. Atropine at 1 g/450 kg (990 lb) body

but it may produce soft, moist sounds and a few

weight intravenously acts as a bronchodilator and cor-

crackles. Death can occur as the result of excitement.

ticosteroids may be useful. Flunixin meglumine has

Less severely affected animals show tachypnoea (rate

been beneficial in experimentally produced acute

50–80/minute) with hyperpnoea and usually there is no

bovine pulmonary emphysema and in the field.

dyspnoea. The rectal temperature tends to be normal. Control means that if animals are hungry when they enter a new pasture in the autumn, restrict their feed by only heard occasionally and in some recovering animals only allowing grazing for short periods during the first two weeks. This should be for about 2 hours on the first day, increasing by an hour a day so that the cattle can be left out for the whole day after about 12 days. Otherwise the area can be strip-grazed or initially grazed with a less susceptible species such as sheep. If monensin sodium is given at the rate of 200 mg/head per day before and after and bronchial mucosae. The lungs tend to be swollen, entering the pasture, this can stop problems.

Necropsy

Dead animals have haemorrhages in the larynx, tracheal given at the rate of 200 mg/head per day before and after and bronchial mucosae. The lungs tend to be swollen, entering the pasture, this can stop problems.

Necropsy

susceptible species such as sheep. If monensin sodium is given at the rate of 200 mg/head per day before and after and bronchial mucosae. The lungs tend to be swollen, entering the pasture, this can stop problems.

heavy and dark red in colour. The cut surface glistens, is smooth and has a red appearance. Emphysema may be

Thrombosis of the caudal vena cava

present in the interlobular septa and pleura. Histological examination reveals severe congestion and oedema

Aetiology

of the pulmonary tissue, hyaline membrane formation, severe interstitial emphysema and moderate epithelial

The cause is a septic focus, usually in the liver, resulting hyperplasia of type 2 pneumocytes.

in a septic thrombus in the caudal vena cava, from

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which there is the haematogenous spread of infection normally present within the pulmonary artery. In the to the lungs.

lung itself there is usually embolic suppurative pneumonia, intrapulmonary haemorrhage, often concentric and globular in shape, and multiple red areas where

Epidemiology

blood has been aspirated. When there is obstruction of

This is an uncommon condition affecting single animals

the hepatic veins then there is marked hepatomegaly

over one year old, although many cases occur in the

and ascites.

growing animal. A few cases of thrombosis of the cranial vena cava have been recorded with similar signs.

Most cases result from a liver abscess. This causes a

Diagnosis

localized phlebitis, usually in the area of the vena cava,

Diagnosis involves the history of loss of condition and

adjacent to the liver. Septic emboli pass to the lungs

respiratory signs in a single animal. The signs help, par-

where they can produce chronic suppurative pneu-

ticularly haemoptysis with thoracic pain, and are almost

monia and multiple lung abscesses, or they can cause

pathognomonic. Post-mortem findings are relatively

pulmonary arterial lesions. Endarteritis, arteritis and

diagnostic with thrombosis of the vena cava, emboli

thromboembolism occur, resulting in aneurysms of the

in the pulmonary artery and intrapulmonary haemor-

pulmonary artery, which then rupture causing haemor-

rhage. Haematological examination shows the packed

rhage in the bronchi and alveoli. Usually, there is a

cell volume is often low (11.0–22.5 per cent).

history of sudden onset of respiratory disease, although

Differential diagnosis includes an accident, but signs in some cases there is a history of chronic loss of weight would be highly unlikely unless there is an immediate and coughing. A few cases show obstruction of the history of trauma. Tuberculosis (p. 863) could also give hepatic venous return with chronic venous congestion rise to some of the signs but is usually much slower and of the liver, its enlargement and no access to the col- the tuberculin test would reveal this.

lateral venous drainage. Bacteriological examination often reveals little because of previous therapy. However, some cases reveal staphylococci, A. pyogenes and Treatment and control Fusobacterium necrophorum spp.

There is no effective therapy. Cattle can be casualty slaughtered if necessary after a course of four or five

Signs

days' antibiotic therapy using a broad-spectrum compound and then leaving the required withdrawal time.

Peracute signs result in an animal dying suddenly with

Control is not possible, but any septic focus should be

no premonitory signs but usually there is a pool of

treated adequately as soon as it occurs. Make sure that blood in front of it. In the acute case, cattle with the all changes in feeding are undertaken slowly so as to condition show respiratory disease for a few days or avoid the possibility of acidosis (p. 829).

some months, with tachypnoea and shallow breathing. The animal develops haemoptysis and frothy blood can be found in the nasal passages and mouth. There are often blood stains around the animal and in many cases

Contagious bovine

there is melaena. There is a variable amount of thoracic pleuropneumonia (CBPP)

pain with abduction of the elbows. On auscultation there is a widespread whistle, with wheezing sounds.

Mycoplasma mycoides is the cause of contagious bovine The chronic form involves animals developing con-pleuropneumonia (CBPP), which now that rinderpest

gestive cardiac failure and ascites with an enlarged has been controlled, is the cattle disease of greatest economic importance on the continent of Africa. CBPP fossa. This often occurs some time before haemoptysis

*affects only cattle and the water buffalo (Bubalis
is present.*

*bubalis). Claude Bourgelat, the great French veterinary Once animals start to
show haemoptysis then death*

*surgeon and founder of the Lyons Veterinary School,
will ensue, usually within a week or two but occasion-
was the first person to differentiate rinderpest from
ally it may take up to 40 days.*

*CBPP (in the eighteenth century). It was thought the
disease was introduced into Europe from Asia in the
seventeenth century and that the wars of the eighteenth
Necropsy*

*and nineteenth centuries resulted in its spread through-
Following death, often one or more abscesses are found
out the continent. From Europe it was taken to the rest
in the liver, and usually the caudal vena cava thrombo-
of the world; South America is the only continent that
sis is in the area of the liver. Multiple septic emboli are
has never experienced the disease.*

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New techniques (PCR, DNA fingerprinting, ELISA,

among others) in manipulating mycoplasmas have led to new thinking about the phylogeny of the group. Where there were individual species, the organisms now have been grouped into 'clusters'. Economically the *Mycoplasma mycoides* cluster is the most important, containing as it does, *Mycoplasma mycoides* subspecies *mycoides*, small colony type (*M. mycoides*), the large colony type that infects goats and to a lesser extent sheep and *M. mycoides capri*, another of the agents that causes contagious caprine pleuropneumonia.

It has been possible to trace the introduction of the infection into many countries. Indeed, in some cases the identity of the infected animal is known! There is, however, some dispute about the entry of the disease into Africa. French workers believe that the infection was introduced when *Bos indicus* crossed into the continent from Asia; it did not get to southern Africa in this way and there is evidence that it did not exist in East Africa before the invasion of General Napier into Ethiopia in 1868.

The Dutch Ambassador to the Cape Colony, with the

Fig. 49.1

*In 1995 most countries in Africa south of the Sahara
intention of improving the local stock, imported a bull
Desert reported CBPP.*

*from his country in 1853. The infection was rapidly dis-
seminated by trek oxen, particularly to the Transvaal
where in the space of two years it killed more than
100 000 animals. From South Africa the infection was
obic conditions. The organism requires a complex
taken to Namibia, Zimbabwe and Botswana. From
medium for growth, containing serum and yeast extract,
Namibia it was taken to Angola and from there to
and growth is slow taking up to 72 hours to reach a
Zambia and the Congo. By 1940, with the exception of
maximum. In broth cultures a swirling mucoid mist is
Namibia and Angola, the southern African countries
produced from the galactan which is attached to the cell
had eradicated the infection. The development of an
membrane.*

*efficient vaccine at Muguga (the T1 broth vaccine) and
Joint Project 16 (a research project to improve knowl-
Epidemiology*

edge of epidemiology, diagnostic techniques and the vaccine) resulted in CBPP being brought under control. Only cattle and water buffalo are susceptible to CBPP; throughout west, central and east Africa by 1970. The African buffalo has been infected under experimental conditions, but that same study found that natural transmission did not occur. There is some debate as to whether *Bos taurus* or *Bos indicus* is the disease throughout the continent; by 1995 most countries more susceptible to infection with *M. mycoides*. An

tries of Africa south of the Sahara reported the disease. Understanding of the epidemiology has been bedevilled (Fig. 49.1). It is said that the vaccine no longer works.

by an inability to reproduce the disease in cattle other than by the cumbersome 'in contact' method, in which cattle are artificially infected by having a culture ino-

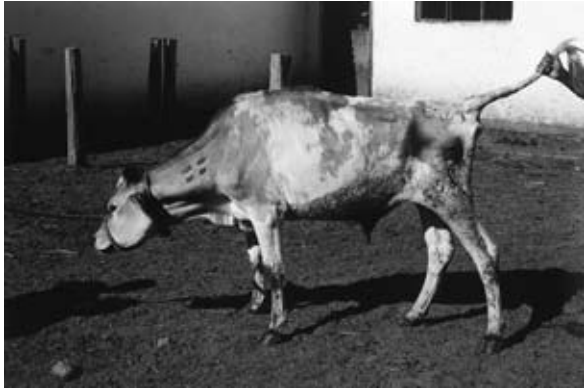
Aetiology

culated directly into the bronchi and then these

Two types of *M. mycoides* subspecies *mycoides* are re-artificially infected

animals are mixed with healthy susceptible animals. Normal transmission is by droplet in cattle and the large colony type which occurs in goats infection from actively infected animals to susceptible and rarely in sheep. They cannot be differentiated in animals in close proximity. However, indirect trans-culture, biochemical or immunological tests; however, mission has been demonstrated under experimental they can be separated by PCR. They have a different conditions.

pathogenicity in cattle, because only the SC types A second factor that hinders the understanding of causes CBPP. *M. mycoides* is a micro-organism that this disease is the potentiation of myths and statements lacks a cell wall and will grow under aerobic or anaerobic fact that have no basis in experimentation, e.g. 'There



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is a prolonged incubation period'; 'lungers break down infection passes down the respiratory passages there is and the animal again becomes infective'. There is no a bronchiolitis and alveolitis leading to pneumonia and evidence for either of these statements but they are pleurisy. It is thought that the galactan plays a part in held to be true. In all the vaccine trials carried out attaching the organism to the mucous membrane and at the East African Veterinary Research Institute, so preventing the body defences from eliminating the Muguga, Kenya, control animals put in contact with dis-organism. Some workers believe that hypersensitivity eased animals showed signs of infection (either clinical or autoimmune reactions are responsible for the lesions or serological) at about six weeks (plus or minus 10

and there is some evidence for this. It is hoped that days). Under field conditions it may be that the animal by use of modern molecular techniques this will be has avoided infection for a long period of time. Work resolved. It has also been suggested that there is a dif- at Muguga failed to reactivate disease in recovered fusable toxin which stimulates the formation of the animals: stress, corticosteroid treatment and even fibrous capsule around the necrotic tissue. Some removal of the spleen were unsuccessful. Until proven workers believe that the galactan acts as an endotoxin otherwise, it is the actively diseased or the animal incu- and causes systemic reactions similar to those seen in bating the disease which must be considered as the infection by Gram-negative bacteria; this is not proven. main risk to susceptible animals.

Until recently it was believed that only one lung was CBPP is a disease of the older animal and calves rarely affected, which would support the suggestion of an show pneumonic disease. Infection in young animals autoimmune phenomenon. However, in recent out-

normally results in lesions in the joints. In consequence, breaks lesions have been seen, in a very few animals, in calves play little or no role in the spread of disease.

both lungs. This may be the result of antibiotic treat-

Although there is no experimental evidence to

ment. As can be seen from the foregoing, much needs confirm the observation, it is generally believed that it to be done to give a clear understanding of the patho- is the weight of infection in a herd that determines the genesis of CBPP.

clinical picture. Under natural conditions, there might be a single animal infected in a herd and this animal will

Clinical signs

show only mild signs which may well pass unnoticed.

Nevertheless, the animal infects several more animals

At least these are clearly understood! In the hyperacute and they in turn yet more. In this way the weight of form the animal may be found dead, without premoni- infection builds within a herd until widespread clinical tory signs, but this is not common. The acute disease is disease is seen. This picture may explain the long incu-

*characterized by fever, lethargy, loss of appetite and
bation periods referred to in the literature.*

pneumonic signs. The animal stands with elbows

*When the infection is introduced into a susceptible
abducted and the neck stretched out, the mouth open
herd there can be a great variation in response, from a
and the tongue protruding (Fig. 49.2). From time to
severe, massive, acute outbreak of disease involving up
time it may emit a soft, moist cough. The pleurisy causes
to 60 per cent of the animals in the herd with 40–50 per
severe pain and the animal will grunt if the chest is
cent of the affected animals dying, to a much less severe
problem, involving less than half of the herd with few
animals severely affected and a low mortality. Recov-
ered animals are resistant to further infection. In many
of these recovered animals sequestra may be seen: a
sequestrum is a piece of diseased lung that has become
separated from neighbouring healthy tissue and sur-
rounded by a fibrous capsule. In a closed herd, in which
no action is taken, it is not uncommon for the disease
to die out. Recently there has been a move towards*

treating clinically sick animals. The effects of this treatment on sequestrum formation are not known but it is thought that it might prevent the animals from developing a proper sequestrum and so prolong the period in which the animal can pass on the infection.

Pathogenesis

Fig. 49.2

Animal showing signs of acute CBPP – The animal

If little is known of the epidemiology of CBPP, even less stands with elbows abducted and the neck stretched out, the is known of the pathogenesis of the disease. When mouth open and the tongue protruding.





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touched. Because the chest is so painful it is uncommon state that relapses occur, but there are no published for an animal with the acute disease to lie down and it reports of this actually happening.

will remain on its feet until almost the end. Filthy mucus Although calves can develop the classical lung lesions runs from the nostrils and saliva may drool from the and show the same signs as the adult, this is uncommon. mouth. Pregnant cows may abort and the fetal fluids

The more typical picture is one of swollen joints caused contain vast numbers of mycoplasmas. Despite the size by a fibrinous bursitis (Fig. 49.3). For some unknown and nature of the lung lesions, it is often difficult to reason it is more commonly the forelegs that are affected. identify the site of the lesions by percussion or auscultation. Although there may be massive kidney infarcts

Post-mortem findings

present it is rare for an animal to show signs of renal disease. If the animal survives the signs grow progres- In the animal that has died from acute disease, the first sively less pronounced, the nasal discharge may well thing seen is the vast quantities of straw-coloured become purulent, the animal will lose a considerable pleural fluid in the chest cavity; 10 litres or more have amount of flesh and become emaciated. The larger the been reported (Fig. 49.4). The fluid may contain pieces lesions, the slower the recovery.

of fibrin that have broken off from the pleural adhe- There is a great variety of clinical manifestation from sions, and up to 109 mycoplasmas per ml. A localized or

this dramatic picture down to almost no clinical signs at diffuse pleurisy may be present, appearing like an all. What determines the size of the lesions and hence omelette.

the clinical picture is not known. In any outbreak it is Acute lesions vary in size from 1 to 2 cm in diameter possible to see acute, subacute and chronic disease, to those affecting the whole of the lobe. More than one often all three pictures at the same time. Many workers lesion may be present but they are usually restricted to

Fig. 49.3

A young calf with CBPP, showing swollen joints

Fig. 49.4

Carcass of an animal with acute CBPP, vast quantities of straw-coloured pleural fluid in the chest cavity; 10 litres or more have been reported. Note that one lung is diseased but the other appears normal.



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Fig. 49.5

Lungs of an animal with acute CBPP. The char-

Fig. 49.6

Lung of animal 27 months after infection, showing a

acteristic lesion is ‘marbling’, identical to that seen in acute walled-off sequestrum. Thomas Walley, likened the sequestrum

pasteurellosis.

to an ‘Egyptian mummy in its case’.

one lung. The characteristic lesion is ‘marbling’, iden-

thought to be caused by emboli from the lung settling in

tical to that seen in acute pasteurellosis (Fig. 49.5).

the kidney. They can be of any size and number and one

Interlobular oedema and fibrin deposits highlight the

or both kidneys may be infected; the colour of the infarcts

structure of the lung. The lung tissue itself may be dark

varies from white to red to black. Lesions affecting

red or changing to grey and thrombi may be seen in the

the joints of calves are normally bilateral, serofibrinous

vessels. These thrombi result in infarction of the portion

tendosynovitis or occasionally arthritis. Lesions are

of the lung supplied. The tissue dies, separates from the

mostly seen in knees and lower joints of the forelegs. A

healthy surrounding tissue and becomes walled off

valvular endocarditis has been reported in calves.

into a sequestrum. The nineteenth century Dean of

the Edinburgh School, Thomas Walley, likened the

Diagnosis

sequestrum to an 'Egyptian mummy in its case' (Fig.

49.6). Small sequestra will disappear over time, to be

A presumptive diagnosis should be based on the

replaced by scar tissue, but the larger ones can remain

history, the clinical signs and the post-mortem findings.

for life. M. mycoides has been isolated from the tissue However, it is essential that the presence of M.

in a sequestrum up to 27 months after the animal was

mycoides be confirmed. The isolation and identification infected. It was rare for these sequestra to become sec-of the mycoplasma gives a certain diagnosis; growth

ondarily infected by other bacteria, but in recent out-

inhibition or immunofluorescent antibody tests can be

breaks such infection has been seen. This may be a

used to identify the organism once it has been isolated.

result of antibiotic treatment.

Under African conditions this may not always be pos-

Histological examination is not very useful in making

sible, particularly if the outbreak occurs at a great dis-

a diagnosis as the lesions are not pathognomonic. The

tance from the laboratory. In such cases samples of

lesion commences with a severe hyperaemia of the

pleural fluid can be taken onto blotting paper and then

affected lung and associated pleura with effusion of ery-

dried. These papers can then be examined by an agar

throcytes, neutrophils and macrophages. There is a

gel diffusion test (AGT) or the newer polymerase chain massive serofibrinous effusion into the alveoli and the reaction (PCR).

interlobular septa. Vasculitis and thrombosis of arteries

In the live animal serological tests are required to occur in the affected parts of the lung. This results in confirm a diagnosis. The simplest crush-side test is the necrosis of that area supplied by the vessel and the the slide agglutination serum or blood test (SAST or onset of sequestration. This commences with a layer of SAST) using a stained antigen. It is cheap to produce, inflammatory cells separating the infarcted area from easy to carry out and has therefore been discarded! the surrounding healthy tissue. Granulation and fibro- False positive results to this test occur, but they do to all sis separate the infarct from the healthy tissue.

serological tests. The complement fixation (CF) test

Lesions resembling sequestra may be seen in the is the test approved by the Office International des mediastinal and bronchial lymph nodes and they are Épizooties (OIE) as the definitive test for the confir-

*always enlarged and oedematous. Infarcts in the kidney
mation of CBPP. This workhorse was developed in Aus-
are common in animals with acute disease, and they are
tralia and has been used successfully throughout Africa
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*for the control of the disease. It does not require expen-
but caused many adverse reactions and some deaths;
sive equipment or reagents but it is a complicated test
however, the broth vaccine gave good protection
to perform, although once mastered, the technique can
without producing too many adverse reactions. Labora-
produce excellent results on a regular basis. The com-
tory and fieldwork showed that it produced a solid
plaint is that CF antibodies do not persist in animals
immunity for more than a year. The T1 broth vaccine was
with sequestra. If these animals play no part in spread-
then lyophilized, but it was too expensive to test this new
ing the disease, then this is of no importance. The tech-
vaccine and so it was assumed that since it was a T1
nique has been modified to be carried out in the African
vaccine it would work! By passage in the presence of*

bush, but apart from Zambia, this field test is no longer streptomycin, a streptomycin-resistant variety of the T1 in use in Africa. After vaccination some cattle develop strain was produced. This enabled the vaccine to be CF antibodies but by 12 weeks these antibodies have mixed with the rinderpest vaccine and so reduce the gone and at present there is no satisfactory way of determining whether or not an animal has been vaccinated. Perhaps this is why the new T1 does not work very well! The new enzyme-linked immunosorbent assay (ELISA) suffers from the same deficiencies. In the absence of Control post-mortem evidence, it would be a brave veterinarian who diagnosed CBPP on serological findings alone. CBPP is a disease of cattle movement; stop movement and the spread of disease is halted. Many countries freed themselves from infection by prohibiting movement and slaughtering infected animals or herds. When Acute pasteurellosis (p. 286) is the condition with which

CBPP was introduced into Botswana in 1995, the CBPP is most likely to be confused, but the former reg-disease was eradicated by preventing animals from ularly affects both lungs. A cultural examination of the moving and a stamping-out policy. The price of freedom lesions should identify the cause. Pneumonic lesions of from CBPP was in excess of \$200 million and there are east coast fever (ECF) (p. 750) could be confused with few African countries that can afford such an expense. those of CBPP, although pleurisy is not normally a For Botswana this was an economically sound policy feature of ECF and there are concurrent lesions in the because it protected their markets in Europe. spleen and lymph nodes of animals with ECF. Exami- In almost all African countries CBPP is a notifiable nation of Giemsa-stained preparations of smears from disease and there are official controls on the import of spleen or lymph node should confirm the diagnosis of cattle. However, in many countries there are nomadic EFC. people who have moved from country to country before

Sequestra can be mistaken for the cysts of parasitic the borders existed, e.g. the Fulani in west Africa and infestations, particularly echinococcus (p. 281), or aber-the Maasai in east Africa. A recent outbreak of CBPP rant liver flukes (p. 276).

in Tanzania resulted from the theft of two animals in Kenya by Tanzanian Maasai who moved the animals across the unmarked 'border'. Lack of appropriate Vaccination (see p. 1011)

action by the Tanzanian authorities has resulted in the There is no treatment for this disease and so vaccination disease spreading the length and breadth of the country. is an important means of protection of the animals. At Wars, famine and inadequate financing of veterinary the turn of the last century, pleural fluid or diseased lung departments have resulted in CBPP running riot in east was injected under the skin of the tail to protect the and central Africa. Unless something is done to stop the animals. Rider Haggard in his novel 'King Solomon's spread it will not be long before the disease topples over Mines' refers to this technique for protecting cattle from

into Malawi and Mozambique, two of the countries the 'lungsick'. He points out that this often resulted in sub Saharan Africa which have never experienced the animal losing its tail, which he considered preferable CBPP. From there the whole of southern Africa is at to losing its life. Throughout the twentieth century risk. Early warning of the arrival of the disease is imperative for adequate control. To this end, it is essential that live vaccines produce protection. The Australian all animals that are slaughtered, be it in an abattoir, on workers developed the V5 vaccine, which was acceptable in Australia, because there farmers considered that trained member of the veterinary department to ensure if a few animals did not die after vaccination, then the that they are not carrying lesions of CBPP. vaccine had not worked. Such a vaccine could not be Rinderpest has almost been eradicated from Africa; used in Africa. In the Sudan the KH3J vaccine was used; it is time that the international community turned its

although it caused no reactions it also failed to protect attention to the control of CBPP on the continent. To cattle. The T1 vaccine was first used as an 'egg vaccine', this end research into vaccines, epidemiology and diagnostic methods are required. Strangely enough these

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Further reading

were the topics of investigation for Joint Project 16 over thirty years ago.

Bygrave, A.C., Moulton, J.E. & Shifrine, M. (1968) Clinical, serological and pathological findings in an outbreak of contagious bovine pleuropneumonia. *Bulletin of Epizootic*

References

Diseases of Africa, **16**, 21–46.

Currason, G. (1936) *Traité de Pathologie exotique vétérinaire*

Blood, D.C., Radostits, O.M. & Henderson, J.A. (1983) *Veteri-et comparée*, Vol II, pp. 500–639. Vigot Freres, Paris.

nary Medicine, 6th edn, pp. 692–6. Baillière Tindall, London.

Henning, M.W. (1956) *Animal Diseases in South Africa*, 3rd

Harwood, D.G., Otter, A. & Gunning, R. (1995) *Peracute* edn. Central News Agency, Pretoria.

pleuropneumonia – adult cattle. Cattle Practice, **3**, (Part 2).

Hudson, J.R. (1971) *Contagious bovine pleuropneumonia*.
149–51.

FAO Agricultural Studies No. 86. FAO of the United

Monies, R.J. (2000) *Tuberculous pneumonia and BVD in*
Nations Organisation, Rome.

housed calves. Cattle Practice, 8, (Part 2), 119–25.

Masiga, W.N., Domenech, J. & Windsor, R.S. (1996) *Manifes-*

Pirie, H.M. (ed.) (1979) Respiratory Diseases of Animals.

tation and epidemiology of contagious bovine pleuropneu-

Notes for a Postgraduate Course, Glasgow University,

monia in Africa. Scientific and Technical Review, 15, Glasgow, pp. 41–2.

1283–308. Office International des Épizooties, Paris.

Chapter 50

Skin Conditions

L.R. Thomsett

Warble fly

875

Bacteria and viruses play a minor role except when skin

Dermatophytosis (ringworm)

878

signs are associated with systemic infection by these

Parasitic skin disease

880

organisms. Allergic disorders are also uncommon and

Lice (pediculosis)

880

genetic diseases rare.

Mite infestations: mange, scabies

881

Warts (viral papillomatosis)

882

Urticaria

883

The effect of skin disease on the livestock industry

Pruritis/pyrexia/haemorrhagic syndrome (PPH)

884

Photosensitization

884

Where animals are reared to provide food and other

Bovine farcy

885

byproducts, skin disease, although clinically not in itself

Atypical mycobacteriosis

886

serious and rarely life-threatening, may cause signifi-

Dermatophilosis (bovine streptothricosis)

886

cant losses to the agricultural industry through the

Horn cancer

887

following effects:

Lumpy skin disease and pseudo-lumpy skin disease

887

Lumpy skin disease

887

- *The debilitating effect of pruritus on the affected*

Pseudo-lumpy skin disease

888

animals. Heavy louse infestations or infestation by

Other conditions having skin signs

888

sarcoptic mites causes irritation, restlessness and weight loss.

The skin of the ox shows general conformity with the

- *Damage to hides from self-trauma or the migration of parasitic larvae.*

(Fig. 50.1). Approximately 7 mm in thickness, it consists

- *Damage to tissue from bacterial infection or larval of epidermis and dermis and their adnexa. The hair migration resulting in condemnation or trimming of follicles are simple and carry a single hair, the colour of meat at slaughter.*

which, depending on body site and breed, may be black,

- *Limitation of sale value or show potential of white or a wide variety of variants of brown or grey.*

infected animals and their danger as vectors of

Single hairs leave the skin surface at an angle, each disease to other stock.

follicle having an erector pili muscle allowing the hair to be raised to a more upright position.

Hair growth and replacement is a cyclic process of

Warble fly

active growth (anagen) when the hair follicle is pro-

ducing a new hair, and a period of rest (telogen) when
The insect is also known as warbles, cattle grubs, gad fly
the mature hair, which now has a constricted bulb and
or Hypoderma infestation and is due to the migrating
is referred to as a 'club' hair, is held in the hair follicle
stages of a parasitic insect (see p. 740). Two species of
before being shed.

warble fly are recognized in many countries of the
Sweat and sebaceous glands are distributed over the
northern Hemisphere, namely *Hypoderma lineatum*
body surface and show specialization in certain areas,
and *Hypoderma bovis* and they differ little in their
e.g. the mammary gland, naso-labial glands of the
territorial distribution. These parasites are not found in
muzzle. At the extremities of the limbs and on the head
the southern hemisphere as they have not become
of horned breeds the skin is specially modified to form
established, despite importation of infected cattle.
the hooves and horns.

Cattle, particularly young stock at pasture, are the
definitive host for the parasitic stages of the life cycle

although other species are recorded as occasionally

Skin diseases of cattle

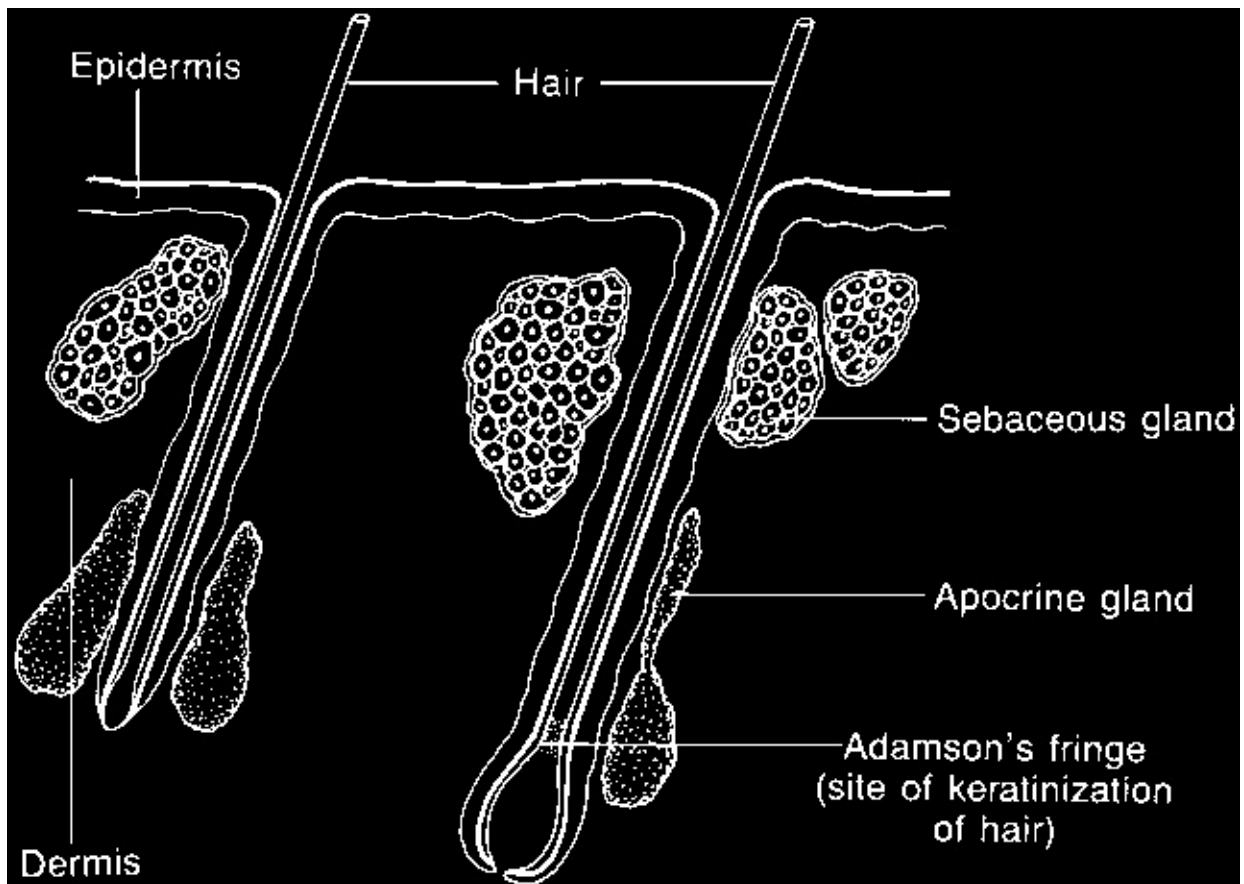
being infested, such as horses, deer, goats and even man.

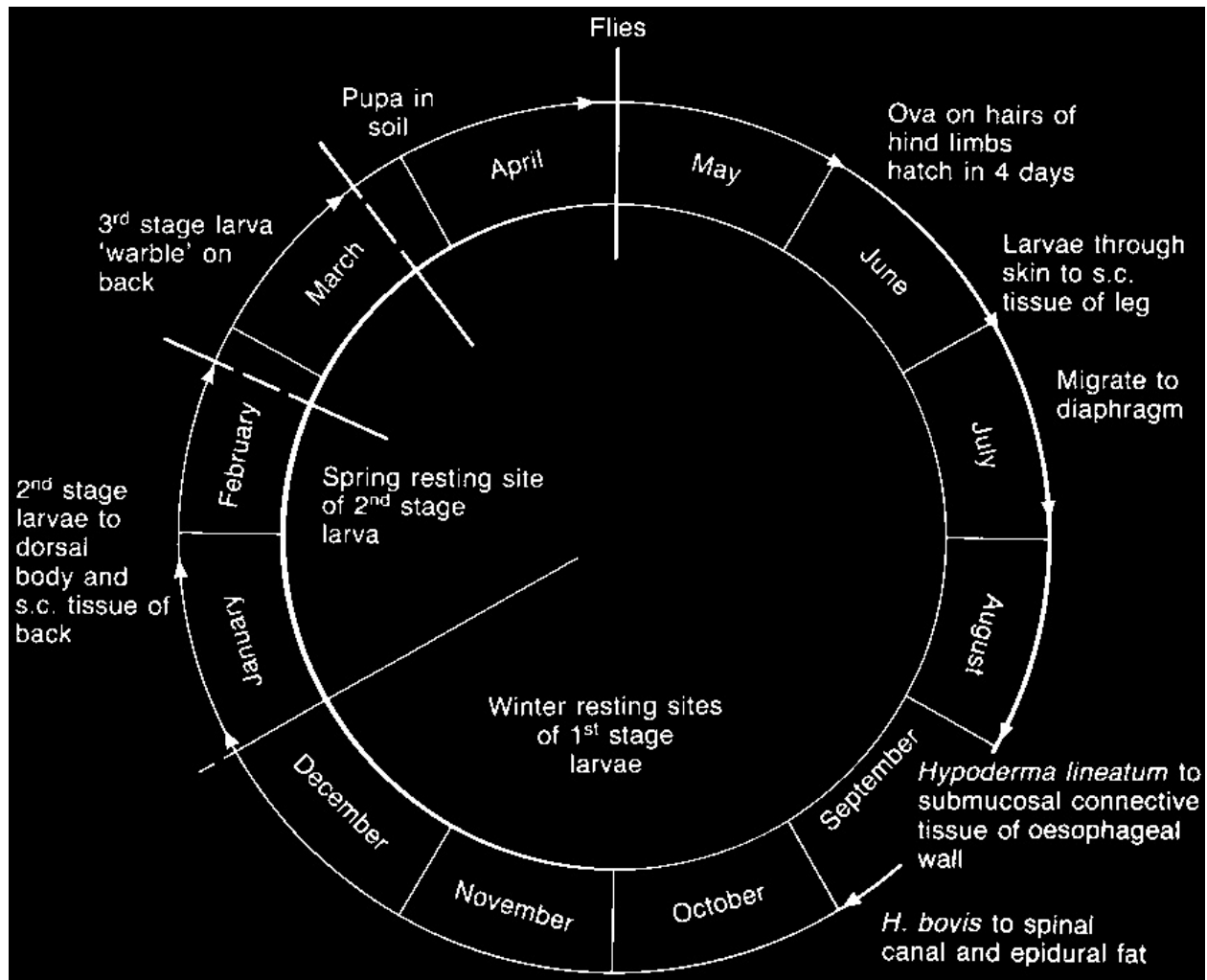
Primary disease of the skin of cattle is more commonly

In species other than cattle the life cycle is rarely, if ever, attributable to parasite infestation or fungal infection.

completed.

875





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apprehension among a group or an individual animal,

Life cycle (see Figs 50.2 and 50.3)

followed by suddenly taking flight at a gallop, tail in the

Warble flies become active in the spring and on warm

air, suddenly turning and repeating the movement in an

days in the summer months adult females (up to 15 mm

effort to shake off the pursuing flies. This is known as

long) home in on grazing cattle and alight on the hairs
'gadding'.

of the lower limbs, on which they lay their eggs. Hypo-

Once ova are attached to the hairs they hatch in four

derma bovis lays its eggs singly while *H. lineatum* lays days and larvae crawl to the skin surface, through which

a row of six or more on a hair. The egg-laying behav-

they penetrate to the connective tissue and wander for

four of the flies causes irritation and restlessness to

four to five months. Migratory patterns within the host

cattle and attempts are made to avoid the flies by

differ: *H. lineatum* moves to the submucosal connective running away.
Characteristically, this is seen as initial

tissue of the oesophageal wall while *H. bovis* goes to the region of the spinal canal and epidural fat. At these

sites they remain for the autumn and winter. As second-

stage larvae they migrate towards the back of the host

where further maturation takes place.

Large domed nodules are formed under the skin

within 30–45 cm (12–18 inches) on either side of the

spine, in which the now third-stage larva produces

a ventral breathing pore. Grubs within the nodules

progressively increase in size to 25–28 mm in length, depending on species. In the spring the larva emerges from its cyst, falls to the ground and pupates. After a period of four to six weeks the adult fly emerges.

Effect of Hypoderma larvae on the host

Fly attacks: Considerable ‘worry’ is caused to cattle when approached by these flies; this results in restlessness that interferes with grazing and may result in poor

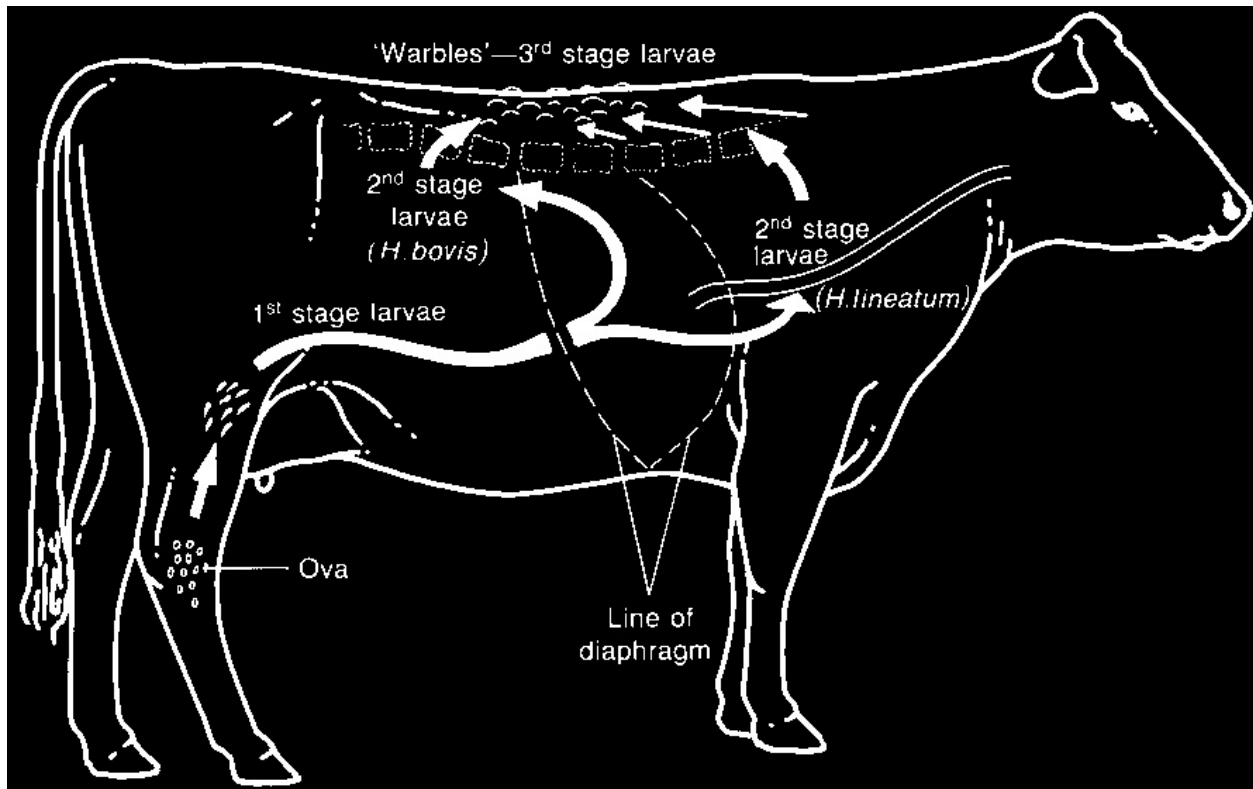
Fig. 50.1

Diagrammatic representation of bovine skin.

weight gain. Milking cattle show a fall in milk yield.

Fig. 50.2

Hypoderma spp. life cycle.



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Fig. 50.3

*Migration of warble fly larvae
within cattle.*

Damage by larvae

Table 50.1

*Dose rate for pour-on treatment using Phosmet
13 per cent w/v.*

• *Carcasses. The presence of grubs may necessitate
trimming of meat at slaughter or, in some cases,*

Weight of animal (kg)

Dose (ml)

condemnation. Trimming makes the carcass less aesthetically acceptable and so it can dispropor-

Up to 130

20

tionately reduce the price when meat is plentiful.

131–200

30

• Hides. Maturation of warble fly grubs to the stage

201–260

40

where they make breathing pores causes serious

261–330

50

damage to the dermis and results in downgrading

Over 330

60

of affected hides.

• Rupture of larvae. This occasionally precipitates an immunological reaction.

When migrating in the earlier stage of the life cycle,

Preparations used include phosmet, trichlorfon, H. lineatum larvae may cause an oesophagitis when fenchlorphos, coumaphos and fenthion. All are they reach the oesophageal wall; H. bovis larvae can cholinesterase inhibitors and knowledge of their result in posterior paralysis.

actions, in particular their toxic effects and antidotes, is essential before using them for warble treatment (see p. 940).

Treatment and control (see p. 1031)

Organophosphorus preparations have been

Treatment of lesions associated with the maturation of employed by dip, spray and wash. The most satisfactory method of application has been shown to be the preventive measures.

‘pour-on’ procedure. An example of pour-on treatment, Since the range of activity of Hypoderma flies is using Phosmet 13.3 per cent w/v solution, is shown in limited to 5–14 km (3–9 miles), control of infestation by Table 50.1.

*eradication is feasible provided neighbouring stock-
Cattle should be treated in autumn (15 September to
keepers treat their animals.*

30 November) or in the spring (15 March to 31 June).

Organophosphorus preparations. Organophosphate

*Best results are obtained by autumn treatment and
systemic insecticides have been found to be the most
prevent skin nodule formation by the third-stage larvae
effective agents for the eradication of Hypoderma
in the spring. No treatment should be given between 1
larvae, the aim being to destroy them early in the in-
December and the following 14 March because of
festation before they reach resting sites near the neural
possible reactions at the winter resting sites due to
canal or are themselves very large.*

the death of the warbles.

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Animals being treated on the above regimen should

*The incubation period of the disease is generally cannot be given
organophosphorus anthelmintics, lev-*

sidered to be approximately one week although four

amisole or diethylcarbamazine citrate at the same time. weeks has been suggested as the period in some out-Special precautions regarding the use and handling breaks. Once in contact with the skin surface of a susceptible animal, the fungus invades the anagen hairs regard to wearing protective clothing, are applicable (telogen hairs are not affected) by enzymatic destruction of keratin. Hyphal growth extends only as far as essential before their use.

the point at which keratinization of the hair takes place In Great Britain warble fly infestation is a notifiable (Adamson's fringe).

disease. The efficacy of control measures aimed at The hair, so weakened, breaks off, leading to the eliminating warble infestation can now be monitored partial alopecia seen on clinical lesions of dermatophy-serologically.

tosis. A generally mild inflammatory reaction accompanies the infection; only occasionally is this severe

Ivermectin: The systemic parasiticides based on ivermectin in cattle, leading to excess production of skin scale, ivermectin are also effective in destroying warble grubs. One folliculitis or furunculosis.

per cent ivermectin injection may be used according to the manufacturer's recommendations after the end of Signs

the period of fly activity and before the larvae reach their resting sites. The milk of treated cattle may not be used

The disease is usually non-pruritic in cattle. Lesions are for human consumption, or manufacturing purposes or characteristically greyish-white and have an ash-like within 60 days prior to parturition. Meat animals must surface. Their outline is circular and they are slightly not be slaughtered within 35 days of their last treatment.

raised due to the accumulation of many layers of scale and the swelling of tissues beneath due to a moderate inflammatory reaction. Some lesions may show areas of

Dermatophytosis (ringworm)

mild exudation and yellow crust formation where the skin reaction is more severe. Removal of hair tufts or

*The infection of hair and skin keratin with the der-
some of the accumulated crust will often leave a raw
matophytes Trichophyton verrucosum, and less com-
bleeding surface. Broken hairs remain as hair stubble
monly T. mentagrophytes, causes lesions commonly
encased in scale and crust (Plate 50.1).*

*referred to as ringworm. This disease has a worldwide
The size of lesions varies, 3–5 cm (1–2 inches) dia-
distribution, the incidence of which is considered to be
meter being common; in the more severely affected
high although an accurate figure for its occurrence is
animals lesions become confluent to form extensive
not known. It is particularly common in young stock
areas of infection (Plate 50.2).*

*between two and seven months of age and during the
The distribution of lesions in calves commonly
autumn and winter months of the year. Adult cattle are
involves the periorbital skin, ears and back, while in
also quite frequently affected.*

*adult cattle the thorax and limbs are the more favoured
Animals kept in close contact with one another, e.g.*

sites. In show cattle subjected to grooming, multiple under intensive management systems, are particularly small lesions develop over the whole of the body and at risk. Other species may also be infected, including limbs following the spread of infective spores by con-horses, sheep and also man in whom it may cause taminated grooming brushes. Very occasionally the serious skin lesions. Although not giving rise to serious udder can be affected (Plate 27.29).

systemic debilitating symptoms, the effect of ringworm is on the value of the animal or its hide.

Duration of the disease

Show animals with the disease may not be shown or Ringworm infection is generally considered to be self-sold, infected stock carry a depreciated market value limiting and the course of the disease to be one to four when offered for sale and the hides of animals slaugh-months, although in some cases a period as long as nine tered show defects that render them less valuable for months has been necessary for resolution to take place. top-quality leather manufacture.

These periods may be shortened by implementing the appropriate therapeutic measures.

Epidemiology

Diagnosis

*The spores of ringworm fungi survive for many months and in some cases years in the farm environment. They
Diagnosis is made on the clinical signs of classic lesions
may be transmitted either by fomites or by asymptomatic carrier animals to susceptible hosts.*

ination of hair and crust samples.

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Collection of samples

*may only succeed in moderately reducing the duration of the disease) has brought the therapy of ringworm in
(1)*

*By forceps epilation of hair from areas of active
cattle into question. Scott (1988) refers to 'a sea of anti-infection.*

*fungals' for the treatment of animal ringworm, many of
(2)*

Scrapings of crust, hair and scale using a scalpel which are of questionable efficacy. The choice of blade.

therapy depends on the availability of appropriate antifungal agents.

Using either of these procedures the material obtained is collected in a paper envelope (ensuring that the quantity of sample is adequate for the diagnostic procedures

Systemic treatment

to be carried out), sealed and labelled for transmission to the laboratory.

(1)

Griseofulvin. An oral feed supplement given at

If culture is contemplated, swabbing the area with

a dose of 10 mg/kg body weight for 7–14 days. In

70 per cent alcohol prior to collection of material may

the USA an alternative routine of 15–35 mg per kg

reduce contaminants in the specimen.

body weight for 18–30 days is considered more

effective. Pregnant animals should not be treated

Laboratory diagnosis: Arthrosporic hyphae on hairs

with this preparation. Milk from treated animals can be demonstrated using the microscope. should not be used for human consumption until 48 hours after the last treatment. Cattle should not

(1)

Wet preparation of suspect material on a microscope slide with coverglass mounted in 20 per cent days after the last treatment. It is no longer available for cattle in the UK.

(2)

Wet preparation in lactophenol cotton blue on a

(2)

Sodium iodide. A dose of 1 g/14 kg body weight as microscope slide under a coverglass.

a 10–20 per cent aqueous solution is given by

(3)

Wet preparation on a microscope slide under a intravenous injection, followed by a repeat injection coverglass mounted in potassium hydroxide/Super

tion seven days later. Pregnant cattle should not
Quink solution.

be treated with iodide therapy. Cattle so treated

In all procedures using the microscope for the exami-
may show signs of iodism (pp. 261, 302, 823).

nation of wet preparations care is necessary in adjust-
ing the microscope illumination in order to visualize
arthrosporic hyphae. This is equally applicable to the
Topicals

examination of portions of cultured material.

Imidazoles: A wide range of imidazole preparations

Definitive diagnosis of the species of fungus may be
have been formulated for the treatment of fungal skin
obtained by culture techniques using Sabouraud's agar,
infections.

mycobiotic agar, or dermatophyte test medium (DTM),

Enilconazole (Imaverol, Janssen Animal Health):

and observing the characteristics of the organism, i.e.

Cattle may be treated by spray or wash using a 0.2 per
colonial morphology and that of the hyphal growth. In
cent w/v emulsion of enilconazole in water. It is rec-

order to arrive at a conclusive answer culture time may

ommended that excess scab and crust be removed by

need to be extended, i.e. for as long as three weeks.

scrubbing with a stiff brush soaked in the antifungal

Using Sabouraud's agar, rapid identification of *T. ver-*

emulsion. Subsequent treatments by spraying or wash

rucosum may be achieved by culture at 30–37°C (34°C), are made on three to four occasions at three-day

when the colony shows long chains of chlamydospores

intervals.

characteristic of the organism.

The DTM may be used to suggest a positive diag-

Other preparations:

nosis of a pathogen by observation of the indicator

Natamycin (Mycophyt, Intervet UK Ltd): A dry

colour change.

powder for the preparation of a 0.01 per cent suspen-

Definitive diagnosis is only by identification of cul-

sion in water is available for application to cattle by

tural characteristics.

spray or wash. Special care is necessary in reconstitut-

ing the powder, for the final suspension reference should be made to the manufacturer's data sheet. Fol-

Treatment

Following an initial treatment a repeat application may be Owing to the difficulty in eliminating the organism from made four to five days later. Subsequent treatments can the environment in which many cattle are kept, and the follow at 14 day intervals. Following treatment with number of animals involved, the reward for treating the natamycin suspension animals should be sheltered from disease with such preparations as are suitable (these direct sunlight for several hours.

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Copper naphthenate (Kopertox): A topical preparation of formaldehyde. The wearing of appropriate protective clothing in conformation with health and safety regulations is essential. Lesions to be treated should

be thoroughly soaked with a solution. A repeat treatment may be given after an interval of 10 days if

necessary. The product is now not available in some

Parasitic skin disease

countries.

Iodine: A liquid made up of equal parts of tincture

Lice (pediculosis) (Plate 50.3)

of iodine and glycerin has been used for the treatment of individual lesions of ringworm on cattle.

Lice are somewhat dorso-ventrally flattened insects up

Note: Special precautions are necessary in the appli-

to 6 mm in length that are host-specific parasites.

cation of topical preparations. Care should be taken to

The distribution of lice is world-wide. The species

avoid sensitive areas, the eyes in particular and also the

infecting cattle are the biting louse *Bovicola* (*Dama-*

transfer of topical applications to other animals in

linia) *bovis*, and the sucking lice *Haematopinus eurys-*contact with those under treatment.

ternus, *Linognathus vituli*, and *Solenopotes capillatus*.

The biting lice feed on tissue debris while the sucking

species suck blood and tissue fluid (see p. 741).

Prophylaxis

Griseofulvin at a dose of 7.5–60 mg/kg per day for five

Life cycle

weeks has been suggested as a prophylactic against der-

matophyte infection. Pregnant animals should not be

The entire life cycle of approximately three to six

treated. However, animals so treated will not be

weeks' duration is spent on the host. Adult females lay

immune to disease and so may become infected at a

their eggs and attach them singly to hairs. These appear

later date.

as pearly or opalescent bodies (nits) 1–2 mm long. From

the egg immature nymphs emerge, and pass through

several moults before becoming adult. Survival off the

Immunity

host is short, usually less than one week. However,

The role of immunity in the epidemiology of outbreaks

under certain conditions lice have been recorded to

of cattle ringworm remains unclear. Re-infection after

survive for periods up to three weeks.

natural infection appears to be uncommon.

On the host, populations of lice vary seasonally, being

highest in colder seasons when the coat is long and

Vaccination (see p. 1017)

transmission by contact from animal to animal readily

occurs. Lower populations in the warmer seasons are

*The immunization of cattle against *Trichophyton ver-**

due to the higher environmental temperature of the

rocosum infection has been practised in Russia and

skin and coat in which lice cannot survive.

Norway and a vaccine is now available in the United

In spite of these factors, small populations may

Kingdom (Ringvac Bovis LTF-130, Intervet Ltd).

survive in protected areas, i.e. the ears, axillae, jowl, tail, Freeze dried material prepared from an attenuated

ready to multiply when environmental conditions

*non-pathogenic stain of *T. verrucosum* is reconstituted become beneficial.*

for the inoculation of young calves at two weeks of age.

Two inoculations are recommended at an interval of

Distribution of infestation on the host

10–14 days by intramuscular injection into the neck.

Biting lice occur mainly on the neck, withers and tail

Disinfection

head. Sucking lice are commonly found as a more generalized infestation, occurring on the head, neck, Viable spores of ringworm fungi may remain in build-withers, down the brisket, tail, axillae and groin. ings and particularly on porous surfaces, e.g. wood, brick, for months or years, making disinfection difficult.

Signs

Cleansing with high pressure water jets, scrubbing down with hot detergent solutions or disinfectants (e.g. The cardinal sign is pruritus, the resulting restlessness benzalkonium chloride) or alternatively, disinfection leading to poor feeding and poor weight gain. The coat with 2 per cent formaldehyde solution after prior scrubbing is poor in condition, often with alopecia and excoriation due to self-trauma, with loss of coat lustre and excess procedures, i.e. spraying and scrubbing walls, doors, etc., dandruff as well as hide damage. Animals in milk may special precautions should be taken to prevent contact show lowered production. Infestations with sucking lice with the preparation used, e.g. on the skin, in the eyes

may give rise to anaemia and consequent increased susceptibility to concurrent disease.

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Diagnosis

panied by secondary infection with bacteria (Plate 50.6).

The persistent intense pruritus causes debility, loss of

This depends on the demonstration of lice and eggs

condition, poor food conversion, hide and skin damage

within the coat. Because of pruritis, the only major

and in dairy cattle a reduction in milk yield. Workers in

disease to be differentiated is mange.

close contact with affected animals may show papular

skin lesions.

Treatment (see p. 1030 onwards)

Diagnosis: Physical examination,

history,

possible

Louse infestations of cattle may be controlled by the

*human involvement, demonstration of *S. scabiei* in skin application of antiparasitic sprays, powders, washes,*

scraping material examined microscopically all help 'pour-ons' and certain injectable preparations. The use of the diagnosis. In exceptional cases skin biopsy may be of these should be complemented by attention to man-helpful.

agement and husbandry aimed at eliminating reservoirs

The main differential diagnosis is with of infection and improving nutrition.

psoroptic/chorioptic mange.

Antiparasitics for louse control, of which there are many, are based mainly on organochlorine, organophosphorus and synthetic pyrethroid active principles, e.g.

Treatment and control (see also p. 1030)

Gamma BHC powder (0.625 per cent), Coumaphos (1)

Organophosphorus pour-ons, dips, sprays.

powder (1 per cent), Diazinon wash (2 per cent), (2)

Gamma BHC 7.5 per cent (not milking cows) 5–

Phosmet 'pour-on' (13.3 per cent) and Permethrin 25 ml in 6 l water.

‘pour-on’ (4 per cent).

(3)

Diazinon 2 per cent wash, 28 ml in 4.5 l water.

Ivermectin 200–300 mg/kg subcutaneously is effective

(4)

Phosmet 20 per cent pour-on.

against sucking lice but of variable efficacy against the

(5)

Permethrin 4 per cent pour on.

biting species. Before use reference should be made to

(6)

Ivermectin 200–300 mg/kg by subcutaneous

the manufacturer’s data sheet for details of special pre-injection.

cautions to be observed, e.g. milk withholding times of

(7)

Amitraz (Tactic, Intervet Ltd). A 0.025 per cent

dairy cattle, permissible intervals between treatment

solution in water is applied by spray at 5 to 10

and slaughter of meat animals.

litres per animal to wet the whole body. This

should be repeated after an interval of 7 to 10 days

Mite infestations: mange, scabies

in severe cases. Further applications if necessary

Infestation of the skin of cattle with parasitic mites may

may be made at two to three month intervals.

be by any of four species: *Sarcoptes scabiei*, *Psoroptes* Special precautions apply to operatives using this

communis, *Chorioptes bovis* or *Demodex*. The inci-preparation in respect of the wearing of protective

dence of disease due to these shows variation in differ-

clothing for which reference should be made to

ent parts of the world in respect of the species of mite

the manufacturer's data sheet. The milk withhold-

and pathogenicity (p. 742).

ing time for milking cattle treated with amitraz

solution is 48 hours after the last treatment.

(8)

In circumstances where the above preparations

Sarcoptic mange, sarcoptic scabies

are not readily available, lime/sulphur dips can be

Sarcoptes scabiei (bovis) (Plate 50.4) has a life cycle of effective. The disadvantage associated with this

10–17 days and may infest cattle of any age, breed or sex. Infection takes place by direct contact with infected cattle or with environmental fomites, although survival intervals may be required on as many as six occasions.

of the mites in the environment is limited to a few days.

(9)

Synthetic pyrethroids.

These are examples of preparations available. Applica-

Signs: *Sarcoptes scabiei* causes an intensely pruritic response to treatment will

popular dermatitis due to the burrowing activities of determine how many applications may be required.

the female mites within the superficial epidermis and a

Reference to the appropriate data sheet is essential for concurrent hypersensitivity reaction. This results in a

information on special precautions and withdrawal

non-follicular popular response, exudation and crusting times to be observed (see for lice, above).

with self-trauma in an attempt to relieve the itching.

This produces excoriation, alopecia and thickening of the skin (Plate 50.5).

Psoroptic mange, psoroptic scabies

Lesions commonly commence about the head and spread to become generalized with gross thickening of

Psoroptes mites are non-burrowing and have a two-skin folds. In some animals these lesions may be accom-
week life cycle. Survival off the host may be as long as

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three weeks. Transmission of infection is by direct or

Three species of Demodex mites have been identified
indirect contact.

in cattle, the life cycle of which, as in other hosts, is
obscure. Transmission of infection occurs from dam to

Signs: Infection commences over the withers and
calf by contact during suckling. No age or sex predilec-
spreads to the rest of the body. Lesions are papules
tion has been established, nor is it clear why an organ-
accompanied by severe pruritus leading to extensive
ism living as a commensal should suddenly become
serous exudation and crusting with alopecia and

a pathogen by its rapid unpredicted multiplication; lichenification. Irritability and consequent debility may immunodeficiency has been suggested as one cause. result in unthriftiness, poor weight gain, hide damage and reduced milk yield in lactating animals.

Signs (Plate 50.7): Multiple nodules, sometimes secondarily infected, result in folliculitis and furunculosis

Diagnosis: The diagnosis is by demonstration of mites of the face, neck and shoulders, occasionally becoming by microscopic examination of wet preparations of skin generalized. The disease is usually non-pruritic but never scrapings. Differential diagnosis is with sarcoptic ertheless results in severe hide damage and consequent scabies, and chorioptic scabies.

economic loss.

Treatment: Treatment is the same as for sarcoptic

Diagnosis: With skin scrapings, wet preparations of scabies, although care is needed to ensure that infection expressed nodule contents examined microscopically has been eradicated. Resistance to treatment has been show caseous sebaceous masses and large numbers of

recorded.

mites.

Histology of skin biopsy shows distended hair folli-

Chorioptic mange, chorioptic scabies

cles packed with mites. Secondarily infected lesions

show folliculitis, furunculosis and dermal granuloma

Chorioptes bovis is a superficial, non-invasive inhabi-

tant of cattle skin. Living on epidermal debris, it has a

Differential diagnosis is with dermatophytosis (p.

life cycle on the host of two to three weeks. It survives

878) and dermatophilosis (p. 886).

only a few days off the host under natural environ-

mental conditions although under experimental condi-

Treatment: Demodicosis is often difficult to treat and tions it may live as long as 10 weeks.

prognosis is always guarded. Organophosphorus 'pour

Transmission of infestation is usually by contact. Out-

ons' have in some cases been found to be beneficial.

breaks among housed dairy cattle are higher in the

winter time when mite populations are greatest.

Warts (viral papillomatosis)

Signs: Non-follicular pruritic papules result in self-trauma accompanied by erythema, excoriation, exudation, alopecia and the formation of crusts. These mostly infectious disease of the skin of mainly young cattle, involve the limbs, particularly the hindlimbs, the udder, usually self-limiting, widely distributed about the world, scrotum, perineum and tail. The neck and flanks may posing little difficulty in diagnosis, prognosis or treatment also be involved.

ment. The causal agent of the condition is a virus.

This is not, however, the whole story of bovine in-

Diagnosis: Diagnosis is by the microscopical demonstration with DNA papovaviruses of which the bovine papilloma virus has certainly five, if not more, strains. The differential diagnosis is with infestations with lice (p. 880) and other parasitic mites. Cross-reactivity between strains does not occur.

Papilloma virus appears to be host specific and transmission of bovine warts to humans handling infected

Treatment: Treatment is the same as for sarcoptic cattle and cattle products has not been proven. scabies. Apparent spontaneous regression may occur Of the strains of virus isolated, it appears that each in the summer months when infestation appears to be has an anatomically determined predilection site of confined to the distal limbs. infection.

(1)

BPV type I causes frond-like lesions on the nose,

Demodicosis

teats or penis: fibropapillomas in young cattle

Demodectic mites are considered to be normal resi-

(Plate 27.31).

dents of the skin of many species of wild and domestic

(2)

BPV type II causes papillomatosis of the skin of

animals. They are apparently host specific and live

the face, head, neck and dewlap, eyelids and occa-

mainly within the hair follicles and sebaceous glands.

sionally legs of young cattle.

(3)

BPV type III causes atypical warts, small smooth rule. Occasional cases of generalization may be en-white sessile lesions on the udder and teats (p. 367; countered and are likely to be due to a failure in cell-Plate 27.32).

mediated immunity.

(4)

BPV type IV causes alimentary tract and urinary Autogenous vaccines (p. 1014) for the treatment of bladder papillomas and ocular lesions possibly bovine papillomatosis are of doubtful value. Where progressing to squamous cell carcinoma.

large, persistent growths are present, e.g. on the neck,

(5)

BPV type V causes non-regressing rice-grain warts removal by cryosurgery or cold steel surgery may be on the teats.

attempted.

For the prevention of infection with BPV I and II,

Interdigital fibropapillomatosis, although wart-like in commercial and autogenous vaccines are claimed to be character, has no proven viral aetiology.

effective. The use of lithium antimony thiomalate 6 per cent w/v solution by deep intramuscular injection, 15 ml

Epidemiology

on each of four to six occasions at 48-hour intervals is claimed to aid enucleation and necrosis of peduncu-

Infection may be spread either by direct contact with lated warts.

infected animals or indirectly by fomites, e.g. from fences by trauma through minor abrasions or by direct or indirect effects of ectoparasitism. Following infection

Disinfection

lesions appear after a two to six-month incubation

While control of infection by disinfection is not period.

commonly undertaken, where recurrent outbreaks of disease have occurred in housed cattle attempts at dis-

Signs

infection may be justified. Solutions of formaldehyde

or caustic soda in appropriate dilution may be used, Viral papillomatosis is commonly seen in young cattle hosing down all treated surfaces well with water after and once established lesions will resolve in a period of these solutions have been allowed to act and prior to one to twelve months. Commencing as small, smooth, restocking.

hairless, firm, button-like elevations projecting slightly above the skin surface, they may be from 1 mm to several centimetres in diameter (Plate 50.8). They may

Urticaria

develop to become coarse and cauliflower-like (Plate 50.9), sessile or pedunculated and single or multiple in

The physical manifestation of urticaria in the form of number. Lesions are commonly sited on the head, neck oedematous skin plaques or of angioneurotic oedema and brisket and extensive generalization may occur.

may be caused by a wide variety of factors. These may be immunological, as in Coombs' type I and II reac-

Diagnosis

tions, or non-immunological associated with injected

irritants or non-immunological histamine release.

On clinical grounds the lesions are characteristic as is

*Some non-immunologic factors that may precipitate
their distribution pattern on the skin and also the age
urticarial responses or intensify an already established
group affected.*

reaction are pressure, sunlight, heat, exercise, drugs,

*Histological examination shows epithelial prolifera-
chemicals, even psychological stress. Certain genetic
tion with or without connective tissue proliferation:*

*disorders have also been implicated in the aetiology of
BPV I and II produce epithelial and fibrous prolifera-
urticarial reactions.*

tion; BPV III, IV and V produce epithelial proliferation.

*Urticaria is usually seen as an acute onset disorder
having no age, breed or sex predilection, which runs an*

Prognosis

*acute or chronic course. It mostly affects a single indi-
vidual or, on occasion, a small group.*

*In infection with BPV I and II, spontaneous regression
within one to twelve months is the rule, while in the case*

Signs

of BPV III, IV and V and 'interdigital papillomatosis', lesions tend to be persistent.

In cattle, urticarial reactions are associated with insect and arthropod bites and stings, infection, the administration of antibiotics, vaccines or other biological products, some feedingstuffs as well as stinging plants.

With uncomplicated cases little treatment is required

Lesions of urticaria may vary in size, often being since spontaneous regression in young animals is the plaque-like wheals, localized or generalized over the

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neck, body and upper limbs in particular. They are

Signs

usually cold to touch and may pit on pressure. Pruritus

The systemic response is of a high fever (40–41°C; 104–106°F) with petechiation of the conjunctiva and skin surface or for there to be haemorrhage. Urticarial visible mucous membranes, with general dullness. An

lesions commonly show a well-defined shape or pattern, extensive papular to exudative dermatitis with pruritus e.g. being ovular, serpiginous or arciform, or in the case of variable intensity develops over the head, neck, perineum, udder, back and tail-head (Plate 50.10). These extensive oedematous swellings.

signs are accompanied by self-trauma (rubbing, kicking and licking), which leads to excoriation with bleeding on turning out housed cattle onto spring pasture, manure and hair loss over a period of days to several weeks.

fest as oedematous swelling of the periorbital skin, Although the dermatitis may subside, the febrile muzzle, perianal and perivulval tissue and occasionally response may persist for four to seven weeks.

the udder and teats. The condition is usually transient Seriously affected animals may die, while those and apparently causes little distress. More severely less so are unthrifty and ultimately have to be affected animals may show evidence of respiratory

destroyed.

distress.

In the USA a form of auto-allergic urticaria has been described in Jersey and Guernsey cattle. Affected indi-

Diagnosis

viduals are believed to have a genetic predisposition to The blood picture shows leucopenia followed by leuco-an allergic response when there is unusual engorgement cytosis. Skin biopsy shows superficial and deep perivas-of the udder or undue retention of milk. Clinical signs cular dermatitis with eosinophils and mononuclear cells are of allergic urticarial skin rash and, in some individ-predominating.

uals, respiratory distress.

The post mortem reveals generalized petechiation particularly subserosal, with some cases showing free

Diagnosis

blood at external orifices.

Diagnosis is mainly on history and lesions of rapid

The differential diagnosis is with other acutely febrile onset. Urticarial-like plaques with superficial exudation

diseases of dairy cattle, such as anthrax (p. 717), and
and crusting closely resemble the discrete active lesions
exudative dermatitis with severe pruritus of parasitic
of dermatophytosis (p. 878).
origin.

Treatment

Treatment

Treatment is with subcutaneous or intramuscular
This is non-specific and usually there is little response.
injection of 3–5 ml of 1/100 adrenaline solution. Cattle
A few cases have improved following prolonged use of
with milk allergy should be milked out. In urticaria
injectable corticosteroids.
spontaneous resolution is often rapid and may have
commenced or taken place by the time a visiting
veterinarian arrives to attend the case.

Photosensitization

Pruritus/pyrexia/haemorrhagic

Aetiology

syndrome (PPH)

This biophysical phenomenon occurs when skin

becomes sensitized to certain wavelengths of sunlight,
A disease of cattle, the pathophysiology of which is particularly within the ultraviolet range of the spectrum, was first reported in cattle in the UK and in The Netherlands in the late 1970s. In The Netherlands the photodynamic agents (see also pp. 370, 943).
outbreaks were associated with the feeding of concentrated Photodynamic agent in circulation & skin & irradiation
trates containing a urea compound, diureidoisobutane.
by sunlight & cell death & necrosis & sloughing
In the UK this compound was not implicated and the causal factor has not been identified. Most cases were
Substances giving rise to these reactions in cattle may
on self-fed silage, often after the introduction of a defective porphyrins originating from defective haemoglobin
ferent silage additive to the farm. All cases were in
metabolism, e.g. congenital porphyria, bovine proto-cows, with a morbidity of 10.9 per cent and up to 25 per
porphyria, or they may be substances of plant origin,
cent mortality, although almost all cases are culled due

e.g. hypericin, which is found in the plant St John's wort to loss of condition.

(Hypericum perforatum). This process of photosensi-

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tivity is often referred to as primary or direct light Lesions on the muzzle and udder may suggest a differ-sensitization.

ential diagnosis of bluetongue (see p. 691).

Alternatively, another set of circumstances can lead to photosensitization, referred to as secondary or

Prognosis

hepatogenous light sensitization. Although the final outcome, skin necrosis and sloughing, is the same as in the primary form of the disease, provided the underlying cause is not genetic, then removal to housed, about differs.

well-ventilated, shaded and cool conditions should be

Where liver damage (of diverse aetiology) interferes

adequate to allow resolution of the lesions. Where

with the metabolism of the chlorophyll metabolite,

severe liver damage is confirmed, prognosis is poor and

phylloerythrin, this latter substance enters the circulation may well be terminal.

tion. As with other porphyrins, it initiates a light sensitivity response when present in the skin.

Treatment

Both of these manifestations of photosensitivity are systemic in origin. A third, localized, form of skin reaction to sunlight may on rare occasions be induced by Cool, shaded, fly-free housing to avoid myiasis is necessary. Where necrosis and sloughing has taken place contact with the sap of certain plants containing psoralens. Localized lesions are more likely to be found on the application of powder dressing to that end is beneficial. Resolution in severely affected animals is often a long process. Antibiotics to limit secondary infection place, the following circumstances are necessary: may be justified, as may the short-term administration of corticosteroids in certain cases. In secondary photo-

lesion hygiene is essential to prevent fly strike and

(1)

The photodynamic agent must be present in the dermatitis, the justification of therapy will be determined by the acuteness or chronicity of the underlying

(2)

*The exposed skin will be non-pigmented (the disease.
greater the density of melanin pigmentation, the greater is the protection against ultraviolet solar radiation).*

Bovine farcy

(3)

*The density of hair cover should be such that sunlight can penetrate to the skin surface.
A chronic nodular and ulcerative disease of the skin of Photosensitization leading to photodermatitis is cattle, the causal organisms of which may be either essentially a physical process. The activation of por-Nocardia farcinica or Mycobacterium farcinogenes. phyrins within skin cells by ultraviolet irradiation*

The disease is only found in Africa, Asia and South America. releases energy that causes cell death, the clinical signs

of which are erythema, oedema and necrosis of exposed non-pigmented areas of skin. In some animals vesica-

Aetiology

tion is present prior to necrosis, sloughing and ulceration of affected areas. Pruritus and pain may also be

Nocardia farcinica is a soil saprophyte that becomes shown.

inoculated into wounds by contamination or enters the

Sites most likely to be involved in cattle are those

skin by inoculation via tick vectors, which themselves most exposed to direct sunlight, e.g. the head, neck, carry the organism for long periods of time.

back and lateral aspects of the body, udder and teats (more extensively when the photosensitive animal is

Signs

lying down) (Plates 27.23, 50.11 and 50.12).

Once inoculated, slowly developing painless nodules appear more commonly over the head, neck, shoulders,

Diagnosis

limbs, perineum and groin. Infection may give rise to The dramatic lesions of skin necrosis and sloughing mastitis and whilst following the lymphatics cause should not present diagnostic difficulty in the establishing of these vessels as well as lymphadenopathy. Ulceration of lesions occurs with the discharge of thick, of illness, e.g. jaundice, positive blood screens for grey/yellow material.

hepatic disease, should determine whether the condi-

Infection may be protracted and confined to the skin tion is primary or secondary.

but generalization, particularly to the lungs and viscera,

Very localized lesions on distal limbs, ventrum or can occur with a progressive loss of condition leading muzzle would suggest a topical plant contact aetiology. to death.

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Diagnosis

tous bacterium. Infection with D. congolensis is con-

fined to the epidermis where the motile zoospore stage

Diagnosis is dependent on the clinical signs. The organ-

of the organism, activated under suitable conditions of

ism may be demonstrated in smears of exudate (acid-

climate and skin damage, grows to form a filamentous

fast filaments, which are beaded). Culture of Nocardia

mycelium within the epidermis. In dry conditions the

farcinica is also possible.

spore stage of the bacterium may remain dormant in

Histopathology shows the presence of pyogranulo-

dry crust, scab and hyperkeratotic skin debris for many

matous dermatitis.

months.

The differential diagnosis is with so-called skin tuber-

A number of factors influence the incidence of the

culosis due to acid-fast bacilli, particularly nodules

disease, of which high temperature and humidity play

found on the lower limbs or cording of lymphatics of

an important role. Similarly, breed susceptibility is of

forelimbs (see below).

importance, indigenous cattle being more resistant to

infection than imported exotic breeds.

Treatment

The initiation of lesions requires skin damage, albeit superficial, by abrasion, e.g. thorn scratches, insect bites

Inorganic iodides have been used but the advice given (biting flies or ticks may themselves carry the infective is usually to have the affected animals destroyed. organism).

The incubation period may be as short as two weeks.

Atypical mycobacteriosis

Signs

Skin infections with environmental mycobacteriaceae

Bovine cutaneous streptothricosis commences as a circumscribed moist patch, often with raised or matted edges, and is well recognized worldwide. Lesions arise from the introduction of organisms through sites of skin trauma.

hairs, giving a characteristic ‘paint brush’ appearance.

Infection gives rise to nodular lesions on the distal

Discrete lesions occur in the initial stages which coalesce, sometimes discrete, otherwise in chains following

the course of the superficial lymphatics. Nodules occur crust (Plates 50.13 and 50.14). In severe infections occasionally rupture to exude creamy pus. The condition is much as half the skin surface may be involved. Scab commonly referred to as 'skin tuberculosis'.

may be of variable thickness and on removal show a Diagnosis is by the demonstration of acid-fast organisms in the smears of pus or by histopathology of biopsies leaving a raw, bleeding epidermis.

sied lesions. The importance of this infection is its role in inducing a non-specific reaction to the intradermal tuberculin test (see p. 863). There is no treatment.

Diagnosis

Impression smears of the exudate, fixed and stained by Gram's method or methylene blue, reveal numerous

Dermatophilosis

rows of cocci formed into branching filaments. Organisms (bovine streptothricosis)

isms may not be readily demonstrable in material taken from old lesions or those affected by secondary infec-

*This is a chronic or acute exudative dermatitis that may
tion. Dermatophilus congolensis may be cultured on
affect many species of animals and is of world-wide
blood agar under microaerophilic conditions.*

distribution. It also occurs in humans.

Differential diagnosis is with dermatophytosis (p.

*The disease in cattle is of particular importance in
878) and sarcoptic scabies (p. 891).*

*Africa, where it is thought to have been known since
the nineteenth century, in America, the Middle East and*

Treatment

Mediterranean Europe.

*In cattle the disease is of importance for its role in
Antibiotic therapy using penicillin and streptomycin as
causing economic loss through down-grading of hides
a single dose regimen (70 000 iu penicillin and 70 mg/kg
and skins, reduced milk yield, marked debility in
body weight streptomycin intramuscularly) will prevent
severely affected animals with dehydration and death.
epidermal invasion by zoospores as well as re-infection.
Alternatively, five daily doses of 5000 iu penicillin and*

5 mg/kg body weight streptomycin may be given (Lloyd,

Aetiology

1981). Long-acting oxytetracycline by deep intramus-

The agent responsible for infection is the actinomycete,

cular injection at 20 mg/kg body weight has also been

Dermatophilus congolensis, a Gram-positive filamen-

found to be effective (Lloyd et al., 1990). Topical appli-

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cation of solutions of cresols and copper salts in appro-

has a world-wide distribution amongst all types of

priate dilution may have a preventive effect.

cattle.

Prevention

Lumpy skin disease (see p. 768)

Since predisposition to the disease is due to skin trauma

A seasonal disease that occurs particularly at times of

from vegetation, ectoparasite infestation with poor hus-

high insect population and can affect cattle of any age,

bandry resulting in malnutrition and/or the presence of

breed or type.

concurrent disease, many of these factors are difficult to

The mode of transmission of infection has not been control. Efforts directed towards the establishment of fully determined although the association with periods herds of breeds resistant to D. congolensis may be of of high insect population and proof that Stomoxys

long-term benefit.

calcitrans can carry the virus suggests that the organism The quest for an effective vaccine continues but is

is insect-borne. The incubation period is one to four still in the experimental stage of development (Lloyd, weeks.

1984). A combination of improved husbandry with Lumpy skin disease causes high economic loss vaccine therapy may eventually be the effective means through decreased milk production, abortion, loss of of control.

condition and hide damage.

Signs

Horn cancer

The initial viraemia is associated with a febrile

This disease, which results in neoplasia of the horn core, response, nasal discharge, excess salivation and possibly

is usually an extension of squamous cell carcinoma that lameness. After a period of one week the first signs of arises from the mucosa of the frontal sinus. Involve-skin lesions may be seen as papules/nodules with ment of the horn core causes loosening of the horn, enlargement of the superficial lymph nodes.

which drops off leaving the tumour exposed. While Skin lesions can be localized or generalized, circum-metastases occur in such conditions as squamous cell scribed firm and flattened intradermal nodules up to carcinoma of the eye, they do not occur in horn core 5 cm in diameter. While lesions can be confined to the lesions.

skin of the chest, neck, back, limbs, perineum, udder and scrotum, more severely affected animals may show involvement of the nasal and turbinate bones. Oedema

Diagnosis

of the ventral chest and abdomen, also the limbs, may This is by biopsy and clinical examination. occur.

Skin lesions may well ulcerate and be slow to resolve,

persisting for months to years in some cases. In most

Treatment

instances the skin lesions necrose, passing through

Treatment has been found to have variable degrees of phases of ulceration and scarring before resolution in a success and recurrence is common following surgery. period of one to three months.

Cryotherapy, surgical excision, radiodiathermy and Morbidity is usually around 50 per cent but may be immunotherapy have all been tried.

as high as 90 per cent with mortality in the region of 10 per cent.

Lumpy skin disease and

Prognosis

pseudo-lumpy skin disease

This is always guarded. Those animals that progress to (see also p. 768)

respiratory tract involvement may well die.

Two forms of lumpy skin disease are recognized:

Diagnosis

- *True 'lumpy skin' disease, the causal agent of which*

History, clinical examination, skin biopsy and virus isolation are all helpful. Apart from other histopathological changes present, eosinophilic intracytoplasmic inclusion bodies may be found in keratinocytes,

gives rise to acute or subacute disease among cattle in parts of Africa.

- *Pseudo-lumpy skin disease due to infection with Allerton virus, bovine herpes virus type 2, which and glandular and ductal epithelium of the skin glands.*

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Table 50.2

Other conditions referred to in the text which also show skin signs.

Actinobacillosis

Chronic granulomatous lesions as an extension from infected soft tissues and lymph nodes (see p. 823) Actinomycosis

Chronic granulomatous lesions eroding skin from underlying bone infections (see p. 824) Alopecia

Congenital baldness. Baldy calf syndrome (see p. 181)

Albinism

Congenital oculocutaneous absence of pigment (see p. 182)

Bacterial infections

- *of the teat – blackspot; F. necrophorum (see p. 370)*
- *of the udder – folliculitis, impetigo, Staph. aureus (see p. 371) Besnoitiosis*

*Gross thickening of skin, lichenification, hair loss (see p. 756) Claw conditions
(See Chapter 31)*

Congenital

Epitheliogenesis imperfecta (see p. 181)

Epitheliokeratogenesis imperfecta (see p. 181)

Copper deficiency

Poor coat, change in black coat colour (see pp. 254, 298)

Digital disorders see also Claw disorders (p. 427)

Ichthyosis

Congenital hair loss, accumulations of scaly epithelium (see p. 182) Interdigital dermatitis

See also Claw disorders

Onchocerciasis

Multiple parasitic nodules within the skin (see p. 774)

Selenium poisoning

Hoof cracking, rough coat, hair loss (see p. 305)

Stephanofilariasis

Thickened skin, papules, crusts, ulcers possibly on the udder and teats (see p. 774) Viral diseases

Cowpox

Rare, erythema leading to pustule formation on teats (see p. 365) Foot-and-mouth disease

Vesicles on teats, muzzle, in addition to coronary band and mouth (see p. 700) Herpes mammillitis

Irregular vesicles on skin of udder and teats, discoloration, ulceration (see p. 363) Papillomatosis

Warts on teats (see p. 365)

Pseudocowpox

Common, erythema, papules, vesicles and scabs on teats (see p. 364) Rickettsial disease

Jewbrana disease, petechiation, bleeding through the skin (see p. 766) Treatment

neck, back, udder, perineum and scrotum showing

superficial raised plaques having a central depression

None is effective. Antibiotics may be given to combat and superficial necrosis.

secondary infection. Measures to minimize the possibility of fly strike and subsequent myiasis should also be taken.

Diagnosis

History, physical examination, virus isolation and skin

Prevention

biopsy are helpful. Skin biopsy shows, among other

changes, eosinophilic intranuclear inclusion bodies in
Some protection is gained by vaccination with modified
keratinocytes of the stratum spinosum.

Neethling virus vaccine. Vaccination with sheep pox
virus has also conferred some protection.

Other conditions having skin signs

Pseudo-lumpy skin disease (see p. 768)

See Table 50.2.

A much milder condition than ‘true’ Neethling virus
lumpy skin disease and due to infection with bovine
herpes virus type 2. The organism is also responsible for

References and further reading

outbreaks of herpes mammillitis (p. 363).

General

Signs

Scott, D.W. (1988) Structure and function of the skin. In Large Animal
Dermatology, pp. 2–28. W.B. Saunders & Co., There is absence of a systemic
response or superficial

Philadelphia.

lymphadenopathy with a clinical course similar to
herpes mammillitis, resolution occurring in two to three

Warble fly

weeks. In exceptional cases lesions may persist for considerably longer.

Fadok, V.A. (1984) Parasitic diseases of large animals. Veterinary-Skin lesions have a similar distribution and appear-

nary Clinics of North America: Large Animal Practice, 6, and to those of lumpy skin disease, with limbs, body,

3–26.

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Scott, D.W. (1988) Parasitic diseases. In Large Animal Der-

Bovine farcy

matology, pp. 245–8. W.B. Saunders & Co., Philadelphia.

Soulsby, E.J.L. (1982) Helminths, Arthropods and Protozoa of Lloyd, D.H. (1981) Bovine farcy. In Current Veterinary

Domestic Animals, 7th edn, pp. 432–7. Baillière Tindall, Therapy, Food Animal Practice (ed. by J.L. Howard), London.

p. 1136. W.B. Saunders & Co., Philadelphia.

Dermatophytosis

Dermatophilosis

Scott, D.W. (1988) Large Animal Dermatology, pp. 179–82.

Lloyd, D.H. (1981) Streptothricosis. In Current Veterinary W.B. Saunders & Co., Philadelphia.

Therapy, Food Animal Practice (ed. by J.L. Howard), pp. 1134–5. W.B. Saunders & Co., Philadelphia.

Lloyd, D.H. (1984) Immunology of dermatophilosis: recent

Parasitic skin diseases

developments and prospects for control. *Preventive Medicine*, **2**, 93–102.

Blood, D.C., Radostits, O.M. & Henderson, J.A. (1983) *Veterinary Medicine*, 6th edn, p. 964. Baillière Tindall, London.

animals and man. *Symposium Proceedings*, Ibadan. Academic Press.

Fadok, V.A. (1984) *Parasitic skin diseases of large animals*. Vet-mic Press.

Veterinary Clinics of North America, Large Animal Practice, **6**, Lloyd, D.H., Hawkins, J.P. & Pragnell, J. (1990) Efficacy of 3–26.

long-acting oxytetracycline in the treatment and control of

Linklater, K.A. & Gillespie, I.D. (1984) Outbreak of psoroptic-bovine dermatophilosis. *Veterinary Dermatology*, **1**, 78–

tic mange in cattle. *Veterinary Record*, **115**, 211–12.

82.

Scott, D.W. (1988) Parasitic diseases. In *Large Animal Dermatology*, pp. 207–30. W.B. Saunders & Co., Philadelphia.

Horn cancer

Warts

Bastianello, S.S. (1982) A survey of neoplasia in domestic species over a 40-year period from 1935 to 1974 in the

Gibbs, E.P.J. (1984) Viral diseases of the skin of the bovine teat Republic of South Africa. I. Tumours occurring in cattle.

and udder. *Veterinary Clinics of North America: Large*

Onderstepoort Journal of Veterinary Research, **49**, 195–

Animal Practice, **6**, 187–202.

204.

Hunt, E. (1984) *Infectious skin diseases of cattle*. Veterinary Pachauri, S.P. & Pathak, R.C. (1969) *Bovine horn cancer*.

Clinics of North America: Large Animal Practice, **6**, 155–74.

Therapeutic experiments with autogenous vaccine.

Scott, D.W. (1988) *Epithelial neoplasms*. In *Large Animal Der-American Journal of Veterinary Research*, **30**, 475–7.

matology, p. 420. W.B. Saunders & Co., Philadelphia.

Lumpy skin disease and pseudo-lumpy

Pruritis/pyrexia/haemorrhagic syndrome

skin disease

References to outbreaks are all prior to 1980, since which time Martin, W.B. (1986) Bovine mamillitis. In Current Veterinary there have been no reports in the scientific literature.

Therapy: Food Animal Practice II (ed. by J.L. Howard).

However, a few outbreaks still occur each year.

pp. 472–3. W.B. Saunders & Co., Philadelphia.

Blood, D.C. & Radostits, O.M. (1989) *Veterinary Medicine*, 7th Njoku, C.O. (1986) *Lumpy skin disease. In Current Veterinary edn*, p. 1300. Baillière Tindall, London.

Therapy: Food Animal Practice II (ed. by J.L. Howard).

pp. 481–2. W.B. Saunders & Co., Philadelphia.

Scott, D.W. (1988) *Large Animal Dermatology*, p. 108. W.B.

Photosensitization

Saunders & Co., Philadelphia.

Manning, T.O. (1984) *Non-infectious skin diseases of cattle*.

St George, T.D., Uren, M.F. & Melville, L.F. (1980) *A general Veterinary Clinics of North America: Large Animal Practice*, alised infection of cattle with bovine herpes virus 2. Aus-6, 176–9.

tralian Veterinary Journal, **56**, 42.

Scott, D.W. (1988) *In Large Animal Dermatology*, pp. 76–83.

W.B. Saunders & Co., Philadelphia.

Chapter 51

Neurological Disorders

P.R. Scott

Introduction

890

ical examination. A methodical neurological examina-

Development, structure and function of the nervous system 891

tion allows the lesion to be localized to specific areas of

Development

891

the brain (Braund, 1985). The common diseases and

Structure and function

891

conditions which affect those areas of the brain can

The nature and causes of signs of neurological disorder

891

then be listed and further investigated. This approach

Endogenous biochemical causes

891

is much more preferable than the undergraduate

Exogenous biochemical causes

892

student or practitioner attempting to memorize long

Genetic causes

892

Micro-organisms

892

lists of clinical signs for every neurological disease. A

Neurological examination

895

logical approach to neurological examination of cattle

Cerebral syndrome

895

highlights discrepancies when new diseases arise

Cerebellar syndrome

896

because it does not limit the differential diagnoses to

Vestibular syndrome

896

known diseases.

Pontomedullary (brainstem) syndrome

896

Such a methodical approach (Scott et al. , 1989a, 1995) Collection of CSF

896

was not adopted when a vague neurological disease,

General principles of treatment for CNS infections

897

bovine spongiform encephalopathy (BSE),

first

Summary

900

appeared in United Kingdom dairy herds during the

Specific diseases

900

mid 1980s. The clinical findings were not consistent with

Cerebellar hypoplasia/hydranencephaly

900

any recognized bovine neurological disorder and led to

Bacterial meningoencephalitis

901

Brain abscess

902

spurious diagnoses such as brain tumours, chronic

Cerebellar abiotrophy (postnatal degeneration)

903

hypomagnesaemia, listeriosis, hepatic encephalopathy,

Unilateral middle ear infections/vestibular syndrome

903

etc. The lack of a methodical approach to suspected

Polioencephalomalacia (cerebrocortical necrosis, CCN)

903

cases of BSE resulted in a correct clinical diagnosis in

Listerial encephalitis

904

only 85 per cent of slaughtered cattle. Indeed, many of

Lead poisoning

906

these cattle were examined twice some weeks apart,

Thromboembolic meningoencephalitis (TEME)

907

which should have excluded many acute neurological

Aujeszky's disease

907

diseases. Despite over 15 years' experience of BSE, the

Rabies

908

clinical diagnosis remains only 85 per cent correct.

Bovine spongiform encephalopathy (BSE)

909

Despite data collected from over 20 000 suspected cases

Idiopathic brainstem neuronal chromatolysis of cattle

911

of BSE which were negative on histopathological ex-

Sporadic bovine encephalomyelitis (SBE, Buss disease)

911

Spinal cord lesions

amination of brain tissue, no summary of these clinical cases has appeared in the veterinary literature. This lack of data indicates the absence of a standard logical

Introduction

approach to neurological disease by many veterinarians. Too often the clinical signs are fitted to a limited number of known diseases and it is therefore inevitable that new diseases will not be recognized.

Many of the neurological disorders of cattle such as congenital bovine viral diarrhoea/mucosal diarrhoea (BVD/MD) or Akabane virus infections present as laboratory test in the clinical investigation of neurological outbreaks. It is essential therefore that an accurate

Cerebrospinal fluid (CSF) analysis is the most useful order to expedite preventive

1996) but such samples are rarely undertaken in practice. However, lumbar CSF samples can be obtained

central nervous system (CNS) involvement in a disease from affected animals under local anaesthesia on the process necessitates a detailed assessment of the farm with immediate results available on visual examination, animal's history and a complete physical and neurolog-
nation, ensuring an accurate diagnosis and specific
890

Neurological Disorders • 891

treatment without further delay (Scott & Penny, 1993).
the conducting system for neural impulses. The neu-
For example, animals may present with neurological
roglia are also of two types; the oligodendroglia are
signs indicative of cerebellar dysfunction where a neu-
responsible for the formation and maintenance of the
trophilic pleocytosis and increased CSF protein con-
myelin sheaths of the axons and astrocytes which
centration indicate focal meningitis (Scott et al. , 1994a) provide the supporting
scaffold of the CNS and also
while normal CSF parameters in another case do not
contribute to the blood–brain barrier. In the peripheral
indicate an infectious cause and indirectly support the

nervous system the Schwann cell is the homologue of diagnosis of a developmental abnormality causing dys- the oligodendrocyte.

function (Scott et al. , 1993a).

The nervous system is heavily dependent upon

In many situations in practice the clinical examina-

glucose and oxygen to sustain functional activity; con-

tion is incomplete and fails to yield sufficient informa-

sequently there is a rich capillary network, especially in

tion to make a specific diagnosis. Consequently, in the

regions of high neuronal density. Even brief disruption

field situation response to treatment has often been

of blood supply will interfere with membrane function

used as an adjunct to diagnosis (Morgan, 1992), despite

with accumulation of toxic metabolites, such as lipid

numerous treatments being administered simultane-

peroxides, resulting in swelling or cell death. When a

ously (Power et al. , 1985), thereby limiting interpreta-nerve cell is lost it cannot be replaced and the circuit

tion of the outcome.

containing it is diminished. If substantial numbers of

There are specific changes in antibody titre, serum nerve cells are lost, functional deficits will result. ion, metabolite or enzyme concentration in only a small Infectious agents, toxic incidents and traumatic percentage of bovine neurological diseases and a spe- events may damage not only the neurone but also the cific diagnosis is often reached only in fatal cases which glial components of the nervous system. Following are examined in detail at necropsy (Jeffrey, 1992). injury astrocytes can proliferate to produce an as- However, for cost reasons few cattle are subject to a troglial scar, which itself may distort the tissue and fur- detailed post-mortem examination. Histopathological ther interfere with neural function. Oligodendroglia can examination of the CNS is expensive and multiple sec- also be regenerated from stem cells, but in the mature tions are cost-prohibitive. Accurate lesion localization CNS the ability to replace myelin is to a large extent by the clinician is therefore essential before euthanasia dependent upon the viability of the neurone. is carried out, otherwise discrete lesions may not be sec-

tioned at necropsy and an incorrect diagnosis reached.

The nature and causes of signs

Development, structure and

of neurological disorder

function of the nervous system

Endogenous biochemical causes

Development

(see also Chapters 21, 46, 54)

Development of the nervous system of mammals takes

This group of conditions includes those metabolic disorders that are associated with the stresses of production from early embryogenesis and continue in cattle into adult life and systems of management and includes milk fever, postnatal life. During these phases cells undergoing rapid division and differentiation are susceptible to a wide variety of physical, chemical and infectious agents. These disorders produce no specific pathological changes in the CNS and the clinical condition in most instances can

The physical factors include extremes of temperature,

be readily reversed by rapid correction of the specific especially maternal hyperthermia, and direct or indirect deficiency.

trauma. Chemical factors include organic, inorganic and Inborn errors of metabolism are usually considered plant poisons, nutritional imbalances and toxic products to be heritable, mainly with an autosomal recessive of microbial activity in the maternal or external environment. A gene may control a specific enzyme or enzyme system and a defect therein will block the normal metabolic pathway. The direct invasion of the fetus by micro-organisms presents a further hazard and inherited metabolic pathway. In some cases, however, there may be defects or spontaneous errors in nucleic acid templating alternative pathways to modulate or minimize the con- may produce effects at all stages of development. sequences and these may influence the time of onset and rate of progress of clinical disease.

In inherited congenital myoclonus in the polled

Structure and function

Hereford (Harper et al. , 1986), affected calves, although Structurally, the

nervous system of higher vertebrates

bright and alert, are unable to stand from birth and comprises neurones and neuroglia. The neurones form show stimulus-responsive myoclonic spasms. A second

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genetically mediated disorder, neuraxial oedema, also teristic neuropathology. The clinical signs of hepatic occurs in Herefords and possibly other breeds. Affected encephalopathy, e.g. ragwort poisoning, may be intercalves appear normal at birth but within a day or two mittent and slowly progressive or acutely fatal, dependthey become dull and recumbent with opisthotonus.

ing upon the severity and duration of hepatic overload

The significant pathology is in the CNS and consists of by nitrogenous compounds. There are behavioural vacuolation at all levels of the neuraxis.

changes, with compulsive walking, circling, twitching of

Lysosomal storage diseases comprise an important

ears and eyelids, blindness and, in severe cases, recum-

group of genetically-mediated diseases whereby the

bency with opisthotonus (see p. 945).

products of faulty degradation accumulate in lysosomes and the cell bodies swell. The cells of the heterozygote

Genetic causes

contain approximately half the normal concentration of enzyme, but the animal itself appears normal, whilst
The plurality of genetic neurological disorders in cattle the diseased animal is homozygous for the defect and (Table 51.1) is possibly a reflection of intensive selection for particular production traits from within small appropriate enzymatic assay offers a means of control-populations of superior animals and is facilitated by
ling the disease within a population. Examples of lysosomal storage diseases include lipidosis GM1 gangliosidosis in the Friesian, glycoproteinosis man-
artificial breeding techniques.

somal storage diseases include lipidosis GM1

gangliosidosis in the Friesian, glycoproteinosis man-

Micro-organisms

nosidosis in the Angus, Murray Grey and Galloway

and ceroid lipofuscinosis, which has been described

Neurological disorders in cattle can result from the

in Beefmaster cattle. The clinical signs of GM1 gang-

activities of all types of infective agents. Some invade
liosidosis are reluctance to feed, dullness, progressive
the nervous system, usually causing inflammation of
ataxia and ill-thrift, and appear one to three months
the brain (encephalitis), spinal cord (myelitis) or the
after birth (see p. 179). Clinically, mannosidosis
membranous coverings (meningitis).

Others that

(pseudolipidosis) presents as wasting, altered behaviour
may be present in the alimentary tract or external
with aggression and progressive ataxia (see p. 179).
environment produce neurotoxins or antimetabolites,
which cause degenerative encephalopathies and
myelopathies.

Exogenous biochemical causes

There is a wide range of exogenous biochemical causes

Viruses

of neurological disorder. Some compounds, e.g.

organophosphorus compounds, organochlorides and

Viruses form an important group of neural pathogens.

carbamates, are specific neurotoxins whose mode of

The oronasal route is the most common portal of entry, action is known. However, a great many toxicants but some viruses gain access via the conjunctiva or whose primary targets appear to be gut, liver or the percutaneously by the bite of an insect vector or in-cardiovascular system also cause neurological disturbed animal. Initial replication at the site of infection bances. In the early phases these usually take the form and in local lymphoid tissues is typically followed by of excitement, muscle fasciculations, tachycardia and viraemia with fever during which the virus penetrates hyperpnoea, whilst convulsions and coma frequently the blood–brain barrier and invades the neural characterize the terminal phases. The mechanisms of parenchyma. Typical inflammatory responses are focal this toxic stimulation and depression of the nervous and diffuse proliferations of microglial cells and system are poorly understood (see Chapter 54). perivascular and meningeal infiltrations of lymphocytes. In cattle, intoxication by chemical compounds is not As the CNS is normally impermeable to circulat-

uncommon for reasons that include their innate curiosity, immunoglobulins, synthesis of antibody by B cells, considerable appetite and non-selective habits of grazing and licking. Lead, the organophosphorus compounds, the recovery of the individual. Other virus infections, pounds, ragwort, copper, urea and sodium chloride are e.g. bovine virus diarrhoea/mucosal disease (Trautwein known to cause neuropathological changes. Lead is by et al. , 1987) and Akabane disease (Inaba et al. , 1975), far the most important and is considered in detail later.

generally cause mild or inapparent disease in the adult,

Organophosphorus compounds used as insecticides

but cause severe transplacental infections of the fetus.

and herbicides are variably potent inhibitors of

Fetal meningoencephalitis or gross intracranial malfor-

cholinesterase (see p. 940). The clinical signs of acute

malignancies from destruction of the granuloprival elements

intoxication, miosis, salivation, polyuria, muscle fascic-

ulations of the developing fetal brain may be the result,

ulations, apprehension and ataxia are the consequences

depending upon the stage of fetal development at of cholinergic overstimulation, but produce no charac-infection.

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Table 51.1

Inherited neurological diseases of cattle.

Disease

Mode

Breed(s)

Age at

Clinical features

Pathology

Reference

R/D

onset

Cerebellar abiotrophy

R

Holstein

3–8 months

Progressive spastic

Degeneration of

White et al. (1975)

ataxia, dysmetria.

cerebellar neurones

(p. 178)

Loss of menace

sparing the vermis

response

Hereditary congenital

R

Jersey

Birth to 3

Ataxia and intention

Hypomyelination and

Saunders et al.

ataxia 'jittery'

months

tremor

oedema of

(1952)

cerebellar white

matter

Cerebellar ataxia

R?

Shorthorn

1–3 days

Rapidly progressive

Spongy

Hulland (1957)

Hereford

ataxia with

transformation of

(p. 178)

recumbency

cerebellar white

matter, shrinkage

and loss of neurones

Familial cerebellar

R

Hereford

Birth

Recumbent,

Narrow disorganized

Innes et al. (1940)

hypoplasia and

stuporose,

cerebellar folia.

degeneration

intermittent rigidity

Paucity of

cerebellar cortical

neurones

Familial convulsions

D

Aberdeen

Birth to 6

Recurrent seizures

Swelling and

Barlow (1980)

and ataxia

Angus

months

with gradual

vacuolation of

(p. 178)

development of

Purkinje cells and

spastic ataxia and

Purkinje cell axons

hypermetria

Doddler

R

Hereford

Birth

Muscular spasms and

Calcification of

High et al. (1958)

convulsions,

cerebellar and

nystagmus,

medullary neurones

respiratory

difficulties

GM1 gangliosidosis

R

Friesian

1 month

Reluctance to feed,

Cerebrospinal

Donnelly et al.

dullness and

lipidosis with

(1973)

progressive ataxia

ballooning of

(p. 179)

neurones by

accumulations of

glycolipid

Mannosidosis

R

Angus,

Birth

Wasting, aggression

Vacuolation of

Hocking et al.

Murray

onwards

and progressive

neurones and fixed

(1972)

Grey,

ataxia,

macrophages, renal

(p. 179)

Galloway

lymphadenopathy

epithelial cells and

exocrine pancreas

Neuraxial oedema:

R

Hereford

1–3 days

Dullness, recumbency

Raised urine ketones.

Healy et al. (1986)

maple syrup urine

and

and opisthotonus:

Vacuolation

(p. 180)

disease

polled

nystagmus. Urine

throughout neuraxis

Hereford

smells of burnt sugar

especially white

matter

Inherited congenital

R

Polled

Birth

Stimulus-responsive

Contusions of

Harper et al. (1986)

myoclonus

Hereford

myoclonic spasms

coxofemoral joint

(p. 180)

with fractures/

deformity of

articular cartilage

contd.

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Table 51.1

Continued

Disease

Mode

Breed(s)

Age at

Clinical features

Pathology

Reference

R/D

onset

Bovine generalized

R

Shorthorn

12 months

Muscular weakness,

Accumulation of PAS-

Richards et al.

glycogenosis

incoordination,

positive and Bests

(1977)

deficiency of α -1,4-

carmine plus

glucosidase or acid

granules in

maltase

neurones of

midbrain,

medullary and

cerebellar roof

nuclei

Chediak–Higashi

R

Hereford,

Young

Partial albinism (ghost

Yellow pigmented

Padgett (1968)

syndrome

Brown

adults

pattern colouring).

PAS, LFB and

Swiss

Susceptibility to

Sudan black B-

infection. Premature

positive inclusions

ageing

in nerve cells of

brain, cord,

myenteric plexuses.

Similar to lipofuscin

granules

‘Weaver syndrome’:

R

Brown

8–12

Motor dysfunction

Axonal degeneration

Stuart & Leipold

progressive

Swiss

months

with swinging gait

predominantly in

(1985)

degenerative

progressing to loss

spinal white matter,

(p. 179)

myeloencephalopathy

of movement and

axons in some brain

inability to stand

stem nuclei and

Purkinje cells of

cerebellar cortex.

Paramembranous

densities and synaptic

junctions

Neuronal lipodystrophy

R

Beefmaster

12 months

Blindness, circling,

Neuronal

Read & Bridges

recumbency and

multilamellar and

(1969)

coma

curvilinear

inclusions. Similar

structures may be

found in fixed

*macrophages in
spleen and lymph
nodes.*

*Progressive ataxia of
R?*

Charolais

8–24

Progressive weakness

Segmental

Palmer et al. (1972)

Charolais

months

and ataxia

demyelination of

(p. 179)

terminating in

CNS with retraction

recumbency

of internodes,

formation of

eosinophilic plaques

from oligodendroglial

processes

Recumbent calf

R?

Red Danish 6 weeks

Progressive ataxia,

Degeneration of

Hansen et al.

syndrome

milk

paresis and

neurones in ventral

(1988)

breed

immobility

horns of spinal cord

R, recessive; D, dominant; PAS, periodic acid shift.

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Bacteria

conditions which affect those areas of the brain can

then be listed and further investigated. The most useful

*Bacteria, with possibly *Listeria monocytogenes* as the and cost effective ancillary test is lumbar cerebrospinal*

sole exception, do not invade the neuroparenchyma as fluid analysis (Scott, 1992, 1995a, 1996). For example, a primary event. Infection of the neuraxis usually arises the clinical presentation in a young calf of depression, from a bacteraemia causing meningitis, which may be lethargy, cortical blindness and dorsomedial strabismus purulent or non-purulent depending on the organism is consistent with diffuse cerebral dysfunction; CSF involved. Infection of the meninges may also arise analysis reveals a markedly elevated protein concentration directly following local trauma. Subsequent invasion of the neuroparenchyma, however, depends upon damage are consistent with a diagnosis of bacterial meningitis to the blood–brain barrier, such as may occur following cephalitis which is then confirmed on gross CSF inspection and laboratory findings (Scott & Penny, 1993). tissue by toxic products or infarction due to infective

There is no need to consider an endless list of obscure emboli as in thromboembolic meningoencephalitis differential diagnoses when the neurological findings (Ames, 1987).

and lumbar CSF tap yield pathognomic results. The Other manifestations of bacterial involvement in methodical clinical examination explores all aspects of neurological disease include those caused by the exo- the animal's presentation rather than attempting to fit toxins of clostridial infections occurring elsewhere in the clinical signs to a recognized condition/disease. For the body, e.g. focal symmetrical encephalomalacia example, the clinician may believe the calf described

(Buxton et al. , 1981) and tetanus, which are discussed above is suffering from bacterial meningoencephalitis

elsewhere. Also in this category are the thiaminolytic because it is four days-old, dull and depressed – this organisms that proliferate in rumen contents and are description may equally apply to enterotoxigenic E. implicated in polioencephalomalacia.

coli, starvation/hypothermia, polyarthritis, etc. The

diagnosis is not confirmed until a lumbar CSF tap is

Protozoa (see p. 284)

taken and examined, when all potential diagnoses

except bacterial meningoencephalitis can be excluded.

Protozoan infections with Toxoplasma gondii and

Considerable confusion arose with the clinical recog-

Sarcocystis species have been implicated in neonatal

nition of bovine spongiform encephalopathy in the

necrotizing non-suppurative encephalomyelitis in

United Kingdom during the mid 1980s because practi-

calves (O'Toole & Jeffrey, 1987). Congenital sarcocys-

tis infestation has been recorded as a cause of recum-

known disease and spurious diagnoses such as tumour,

bency in newborn calves. Severe coccidiosis in calves is

hepatic encephalopathy and chronic hypomagnesaemia

uncommonly associated with neurological disorder in

were commonplace. The accuracy of clinical diagnosis

Britain, although more widely reported in North

was approximately 85 per cent with a wide range of dis-

America. Pelvic limb paresis in yearling cattle pro-

eases incorrectly reported as BSE, including listeriosis.

gressing to tetraparesis over two to three days has

Bovine spongiform encephalopathy presents with clinical signs that have been reported,

caused by Sarcocystis zurnii

ical signs of cerebral and cerebellar dysfunction with no evidence of encephalomyelitis.

intrathecal inflammatory response (Scott et al. , 1989b); listeriosis shows the classical presentation of the

Nematodes

pontomedullary syndrome with an elevated protein

concentration in CSF and increased white cell concentration.

The intermediate stage (Coeneurus cerebralis) of the

tration with predominance of large mononuclear cells

dog tapeworm Taenia multiceps may occasionally occur

(Scott, 1996). A methodical clinical examination should

in cattle in well-defined regions of Britain, but is much

readily differentiate these two common neurological

less common than the disease in sheep. The clinical

disorders of adult cattle in the UK.

signs are of a slowly progressive, space-occupying lesion

While the brain can be conveniently divided into six in one cerebral hemisphere, circling to the affected side, areas, each with a recognized neurological 'syndrome' proprioceptive deficits and contralateral blindness.

(Braund, 1985) only four syndromes concern the veterinary practitioner: cerebral, cerebellar, ponto-medullary (brainstem) and vestibular.

Neurological examination

Cerebral syndrome

A methodical neurological examination allows central nervous system lesions to be localized to specific area(s)

Diffuse cerebral disease is common in ruminants, associated with metabolic disease in adults and bacterial

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meningoencephalitis in neonates. The cerebrum is concerned with mental state, behaviour and, in conjunction with the eye and optic (II) nerve, vision. Clinical signs of cerebral disease include circling, ipsilateral hemiparesis and proprioceptive defects are

which suggest cerebral dysfunction include blindness, also common. Circling can be observed in diseases but with normal pupillary light reflexes (II, III), com- affecting the brainstem caused by involvement of the pulsive walking, circling, constant chewing movements, vestibulo-cochlear nucleus. Involvement of the facial severe depression, dementia, yawning, head pressing, nucleus results in ipsilateral facial nerve paralysis. hyperaesthesia to auditory and tactile stimuli, and Facial palsy is evident as drooped ear, drooped upper opisthotonus. It is important to note that the clinical eyelid (ptosis) and flaccid lip. Involvement of trigemi- signs change as the disease condition progresses; for nal nerve or the trigeminal motor nucleus results in example, calves with bacterial meningoencephalitis paralysis of the cheek muscles and decreased facial skin may present with different clinical signs depending sensation. Listeriosis is the classical brainstem lesion. upon the duration of CNS infection. Ipsilateral compulsive circling, sluggish gait, postural deficits, deviation of the head (not a head tilt) and

Collection of CSF

depression with contralateral blindness are variably seen in animals with unilateral cerebral lesions.

Collection of CSF forms an integral part of the clinician's detailed neurological examination. Lumbar CSF can readily be collected under local anaesthesia from

Cerebellar syndrome

cattle of all ages (Scott & Penny, 1993; Scott et al. , 1995).

The cerebellum is primarily concerned with fine coor-

While it may be preferable to collect cisternal CSF for dination of voluntary movement. In cerebellar dys-

a more representative sample, this technique is inher-

function limb movements are spastic (rigid), clumsy and

ently more difficult to perform and is not without risk

jerky. Initiation of movement is delayed and may be

to the patient. Numerous studies in ruminant species

accompanied by intention tremors. Cerebellar disease

(Scott, 1992, 1995a, 1996) have shown that lumbar CSF

is characterized by a wide-based stance and ataxia

samples provide meaningful data which assist the clini-

(incoordination), particularly of the pelvic limbs but

cian in formulating a more specific diagnosis. Lumbar with preservation of normal muscle strength. In addition CSF can be collected under local anaesthesia in the standing animal. Cisternal CSF collection requires appropriate restraint and, in many situations, either deep sedation or general anaesthesia.

severe lesions of the rostral cerebellum.

Collection of lumbar CSF is greatly facilitated when the animal is positioned in sternal recumbency with the hips flexed and the pelvic limbs extended alongside

Vestibular syndrome

the abdomen. In adult cattle that are ambulatory the

In growing cattle unilateral peripheral vestibular

lumbar CSF sample is collected from the standing

lesions are not uncommon. The major clinical signs

animal which must be suitably restrained in cattle

include ipsilateral (to the same side) head tilt, leaning

stocks which prevent lateral movement. The site for

and drifting sideways when walking. Circling may also be observed. Positional nystagmus may be depressed or absent in animals with a vestibular lesion. There is spontaneous horizontal nystagmus with the fast phase directed away from the side of the lesion. There is frequently ipsilateral ventral strabismus (eye drop) which is exaggerated when the head is raised. Facial nerve paralysis is common in peripheral vestibular disease. Sterile surgical gloves should be worn. A guide to needle length and diameter (Scott, 1996) is included in Table 51.2.

close to the middle ear. In central vestibular disease the nystagmus may be horizontal, vertical or rotary.

Table 51.2

Guide to needle length and gauge for lumbar CSF sampling in cattle.

Pontomedullary (brainstem) syndrome

Calves <80 kg

1 inch 20 gauge

As most of the cranial nerve nuclei are present in the

Calves 100–200 kg

2 inch 19 gauge

brainstem, dysfunction is characterized by multiple

Cattle >200 kg

4 inch 18 gauge + internal stylet

cranial nerve deficits. Depression is also a key sign of

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When the lumbosacral site has been surgically pre-

arachnoid space. The normal flow rate is one drop of

pared the needle is slowly advanced at a right angle to

CSF every 2 to 3 seconds. A steady flow of CSF is

the plane of the vertebral column or with the hub

obtained immediately following puncture of the arach-

directed 5–10° caudally. It is essential to appreciate the

noid mater in calves with meningoencephalitis, indicat-

change in tissue resistance as the needle point passes
ing increased CSF pressure. The use of a manometer
sequentially through the skin, subcutaneous tissue,
has no practical application in farm animal practice.
interarcuate ligament then the sudden 'pop' due to the
The analysis of lumbar CSF from a range of
loss of resistance as the needle point penetrates through
neurological disorders, and other conditions which
the ligamentum flavum into the epidural space. Once
present with abnormal mental state, is presented in
the needle point has penetrated the dorsal subarach-
Table 51.3.

noid space, CSF wells up in the needle hub within 2–3
seconds. There is no justification whatsoever in collect-
ing CSF from the ventral subarachnoid space. Between

General principles of treatment for

2–3 ml of CSF is sufficient for analysis and can be col-

CNS infections

lected by free flow from the needle hub or by gentle
syringe aspiration. Care must be taken not to dislodge

The important factors to consider in the treatment of

the needle point from the dorsal subarachnoid space

central nervous system infections include:

when the syringe, with the seal already broken, is

- *Bacterial pathogen;*

attached to the needle hub. Sedation of the animal is

- *Duration and extent of the disease process;*

not usually necessary but intravenous xylazine (0.05–

- *Ability of the selected antibiotic to penetrate the 0.1 mg/kg bodyweight) or diazepam (0.04 mg/kg body-*

blood–brain barrier and achieve concentrations 10

weight) can facilitate positioning of the pelvic limbs in

to 30 times the minimum bactericidal concentration

recumbent animals. It is not necessary to compress the

in order to sterilize the CSF (Prescott & Baggot,

jugular veins to facilitate CSF collection.

1988; see also Chapter 61).

Samples of CSF are collected into EDTA containers.

The specific gravity value is determined using a

Supportive treatments include corticosteroids,

hygrometer. Many cerebrospinal fluid samples have

diuretics, dimethyl sulphoxide and non-steroidal anti-

first to be concentrated before CSF protein concentration. Inflammatory drugs (NSAIDs) e.g. flunixin meglumine, can be determined using the pyrogallol method. carprofen, meloxicam and ketoprofen. In addition, White cell concentration in CSF is determined using a certain situations necessitate appropriate intravenous haemocytometer. Cytological examination of CSF is and oral fluid replacement therapy to correct second-facilitated when the sample is first concentrated by any acid–base and fluid disturbances.

cytopsin. The sample is then air-dried and stained with For an antibiotic to be effective in bacterial meningitis, it must penetrate into the CSF in sufficient concentration based on a minimum of 20 cells. Further laboratory testing, such as protein electrophoresis (Scott, 1993b), (Lambert & Wall, 1991) and achieve CSF concentration adds little additional information and is rarely undertaken 10 to 30 times the minimum bactericidal concentration for cost reasons.

tration to sterilize the CSF (Sande, 1981; Prescott & An assessment of CSF pressure can be made by Baggot, 1988). Even with successful CSF antibiotic determining the rate of CSF flow from the needle hub penetration, the intrathecal defence mechanisms are once the needle point has penetrated the dorsal sub-inadequate to control bacterial infection and treat-

Table 51.3

Lumbar CSF results (median, range) for some common bovine CNS diseases.

Disease

Number

Specific gravity

Protein (g/l)

White cells (×10⁹/l)

Control

31

1.007 (1.006–1.011)

0.28 (0.06–0.73)

0.0125 (0.012–0.25)

Meningitis

17

1.010 (1.007–1.017)

2.5 (0.5–7.1)

2.0 (0.012–12.6)

Listeriosis

12

1.009 (1.008–1.017)

1.69 (0.39–10.4)

0.3 (0.012–1.7)

Bovine viral diarrhoea

5

1.007 (1.006–1.010)

0.22 (0.09–0.24)

0.0125 (0.012–0.1)

Septicaemia

11

1.009 (1.007–1.011)

0.77 (0.19–2.04)

0.05 (0.01–0.2)

Acidosis

5

1.009 (1.007–1.011)

0.35 (0.2–1.3)

0.012 (0.012)

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ment necessitates the use of bactericidal antibiotics

monocytogenes (Tuazon et al. , 1982). It is strongly (Landesman et al. , 1981; Del Rio et al. , 1983).

recommended that the first penicillin treatment is

The blood–brain barrier can be divided into the

administered intravenously. The minimum inhibitory

blood–brain and blood–CSF barriers, although some

concentration (MIC90) of penicillin G for L. monocyto-

researchers consider them functionally synonymous

genes has been quoted as 0.02 mg/ml (Prescott &

because extracellular fluid of the brain is in equilibrium

Baggot, 1988). While lumbosacral CSF penicillin G con-

with CSF. Morphologically, the blood–brain barrier

centrations between 0.09 and 1.02 mg/ml were achieved

comprises the continuous tight junctions (zonulae occlu-

in three cases of bacterial meningitis 48 hours after the

dens) of the endothelial cells which form the brain cap-

commencement of treatment of 44 000 iu/kg procaine

illaries. The permeability of the blood–brain barrier is penicillin b.i.d. (Scott & Sutton, 1992), the median CSF dramatically increased by the inflammatory process, protein concentration in those animals was higher than whatever the cause (Oldendorf, 1975). The increased values encountered in listeriosis cases (2.5 g/l compared permeability of the blood–brain barrier in experimental to 1.7 g/l), suggesting greater disruption of the blood–meningitis models was in part caused by the separation brain barrier. However, it is possible that the CSF penicillin G concentrations achieved in brain tissue were vascular endothelium which was augmented by the considerably higher than those achieved within CSF. presence of CSF leucocytes (Quagliarello et al. , 1986). High dose penicillin G or oxytetracycline treatments Few broad-spectrum bactericidal antibiotics are have been reported to give a good prognosis for listeriosis cases provided the cattle are still ambulatory but it has generally been assumed that the disruption of

when treatment commences (Rebhun & deLahunta, 1982). High dose penicillin treatment (44 000 iu/kg pro-diseases, increases the degree of antibiotic penetration caine penicillin b.i.d.) of cases of ovine listeriosis, where (Barlow, 1991). Such increased permeability may be the clinical diagnosis was supported by characteristic sufficient to allow passage of antibiotics which achieve lumbar CSF analyses, gave a 24 per cent success rate minimum inhibitory concentrations within the CSF (Scott, 1993a). It is possible that the optimum daily dose (Rings, 1987), but few studies have been undertaken rate of penicillin G to treat acute CNS infections could in animals with naturally-occurring CNS infections to be as high as 300 000 iu/kg; approximately 15 times the support such assumptions.

normal dose rate. This dose rate is prohibitively expensive in most cattle except for the first day of treatment. depends upon lipid solubility; the greater the degree of The high cost of appropriate antibiotic therapy for

ionization of an antibiotic in plasma, hence reduced infectious diseases of the CNS further emphasizes the lipid solubility, the lower the penetration through the blood–CSF barrier. Antibiotic penetration also depends upon the degree of protein binding. Large molecular weight antibiotics, such as aminoglycosides, of bovine listeriosis. Reports of successful treatment of achieve very poor CSF concentrations even in the pres- listeriosis with certain antibiotics administered at con- ventional dose rates raise doubts over the clinical diag- As well as the selection of antimicrobial agent, there nosis and whether the cattle were indeed suffering from is considerable disagreement regarding the duration a vestibular lesion, which would have responded well to of treatment for CNS infections. Prescott and Baggot such therapy. (1988) quote the experimental findings of Tauber et al.

Active removal of penicillins from the CSF can be (1987), that the single most important factor in achieving a favourable outcome for streptococcal meningitis life of ampicillin was increased, and total body clearance is the peak concentration of antibiotic in the CSF. Conversely, Rebhun and deLahunta (1982) recommended a six week course of antibiotic treatment for bovine listeriosis. In farm animal practice the cost of antibiotic treatment is an important limiting factor in the animal's before its use can be recommended as an adjunct to treatment and high dose/short duration therapy is penicillin therapy.

perhaps the best compromise in many situations.

Aminoglycosides

Penicillins

Gentamicin is rapidly bactericidal against many poten-

In man, penicillin and ampicillin are usually considered the antibiotics of choice for meningitis caused by L.

gram-negative organisms causing neonatal meningitis but, as a polar antibiotic, is unable to cross the

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blood–brain barrier, even during meningitis. This lack

of penetration into the CSF led to intrathecal and

intraventricular administration of aminoglycoside anti-

encephalomalacia and bacterial meningoencephalitis, antibiotics for the treatment of meningitis in infants and

the control of cerebral oedema is an important factor in children, but this technique was associated with an

affecting prognosis (McGuirk, 1987). The increased mortality rate. Intrathecal injections of gentamicin in the lumbosacral region failed to result in significant concentrations in ventricular CSF (Moellering contributing to death, brain infarction and altera-

*& Fisher, 1972) and, as a consequence, this technique
tions in CSF hydrodynamics. The development of
has not been practised for the treatment of bacterial
potentially-serious increased intracranial pressure is
meningoencephalitis in cattle.*

*indicated by coma, seizures, abducens nerve palsy, per-
sistent bradycardia and respiratory depression. The rec-*

Chloramphenicol

*ommended treatment for the control of cerebral
oedema is either 1–2 mg/kg dexamethasone given*

Until withdrawal from the veterinary market in many

intravenously, 1–2 g/kg of dimethyl sulphoxide as a

countries, chloramphenicol was stated to be the anti-

10 per cent solution given intravenously or frusemide

biotic of choice for the treatment of bacterial menin-

1–2 mg/kg given intravenously (McGuirk, 1987).

goencephalitis (Rings, 1987) as it is capable of crossing

There are no comparative field investigations of cor-

the intact blood–brain barrier. In field studies 30 per cent

ticosteroid treatments in bovine CNS disease. While the

of calves responded to chloramphenicol and showed a

*potential benefits of the concurrent use of dexametha-
marked improvement within 24 hours and no relapses
sone with cephalosporin antibiotics have been demon-
occurred after five consecutive days' treatment (Scott
strated for the treatment of bacterial meningitis in
& Penny, 1993). There are no published studies of the
children, it is reported that there may be a number of
usefulness of florfenicol, a closely-related molecule, in
potential deleterious effects including a reduction in
the treatment of bacterial meningoencephalitis.*

*cellular immune function and reduced penetration of
antibiotics into CSF (Scheld & Brodeur, 1983).*

Metronidazole

*Metronidazole is highly lipid-soluble and capable of
Non-steroidal anti-inflammatory drugs (NSAIDs)
penetrating the blood–brain barrier and into brain*

*The potential application of NSAIDs such as flunixin or
abscesses. Unfortunately, this antibiotic is not licensed
ketoprofen in the treatment of LPS-induced intrathecal
for use in veterinary medicine in Britain and there are
reaction in bovine meningoencephalitis has not been*

no data regarding its use in cattle with CNS infections. reported. Bacterial meningoencephalitis is commonly In medicine, brain abscesses are frequently mixed associated with septicaemia where intravenous NSAID aerobic and anaerobic infections and in such situations metronidazole is administered in conjunction with septic shock. The analgesic properties of NSAIDs may either penicillin or chloramphenicol.

also prove of benefit in the treatment of bacterial infections of the CNS. Therefore, there are a number of indications for NSAID administration in calves with Ceftiofur

bacterial meningoencephalitis, although this statement Third generation cephalosporins are widely used for is not supported by field results. The potential benefits the treatment of bacterial meningoencephalitis in of NSAID therapy in more chronic CNS infections medicine but there are no data relating to the field use have not been investigated.

of ceftiofur in cattle. As chloramphenicol is no

*longer available to many veterinary practitioners,
ceftiofur would present a logical choice for bacterial
Dimethyl sulphoxide (DMSO)
meningoencephalitis.*

*There is no licensed DMSO product for veterinary use
in Britain, but analytical quality DMSO has been used
in the treatment of cattle with CNS dysfunction. DMSO*

*Trimethoprim–sulphonamide
has many properties, including reduction of cerebral
Trimethoprim–sulphonamide has been recommended
oedema and free radical scavenging (Brayton, 1986),
for the treatment of bacterial meningoencephalitis
but special handling precautions preclude its use in
and listeriosis in cattle but there are no published field
most situations encountered in general practice. In
studies.*

veterinary practice, DMSO administration would most

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likely be restricted to the very valuable calf suffering

Pathology

from bacterial meningoencephalitis.

In-utero BVD/MD virus infection around 90 to 130 days' gestation causes cerebellar hypoplasia and, less commonly, hydranencephaly. The BVD/MD virus invades the fetus and infects the developing germinal cells of the cerebellum and destroys the Purkinje cells in the granular layer. Microphthalmia is uncommon. Severe dehydration can be corrected by the intravenous infusion of isotonic saline. Metabolic acidosis, which is commonly encountered in listeriosis cases caused by saliva loss, can be corrected by regular administration of sodium bicarbonate by oro-gastric tube.

Signs and diagnosis

Cerebellar disease is characterized by a wide-based

Summary

stance and ataxia (incoordination), particularly of the pelvic limbs, but with preservation of normal muscle strength. In addition to the ataxia, dysmetria may be collection is a simple procedure and visual inspection

observed with hypermetria more commonly observed
can reveal much useful information, particularly in
in cerebellar disease. Coarse intention head tremors
neonatal calves with bacterial meningoencephalitis,
are frequently observed, particularly during periods of
permitting a rapid diagnosis and prompt treatment.
excitement such as feeding times. Opisthotonus can
Encephalitis lesions, such as those caused by listeriosis,
occur in calves with severe lesions of the rostral cere-
result in minor changes in the CSF and laboratory
bellum. Such calves present in lateral recumbency with
examination is necessary. Ceftiofur, a third genera-
rigid extension of the neck and thoracic limbs and
tion cephalosporin antibiotic, and trimethoprim-
flexion of the pelvic limbs with clinical signs noted soon
sulphonamide are indicated for the treatment of bacte-
after birth.
rial meningoencephalitis but field data are limited. The
In addition to the cerebellar signs, calves with hydran-
usefulness of florfenicol in the treatment of bacterial
encephaly exhibit blindness and depression/somno-

meningoencephalitis has not been reported but this mol-
lence (dummies). In extensively-managed beef herds,
ecule is sufficiently similar to chloramphenicol to justify
calves with hydranencephaly may not be detected until
its use and clinical evaluation in the treatment of
the calf is several months old. Often the demeanour
bacterial meningoencephalitis in calves. Penicillin G
of such calves is attributed to another cause and such
(>44 000 iu/kg b.i.d.) is the treatment of choice for listecalves are not presented
for veterinary examination.

riosis and peripheral vestibular lesions, with the first
Cerebellar dysfunction in young calves may be the
treatment administered intravenously. Insufficient data
first manifestation of active BVD/MD infection in the
are available to comment upon the efficacy of either cor-
herd and it is important that these clinical signs are
ticosteroids or NSAIDs for the treatment of central
identified so that control measures can be introduced
nervous system bacterial infections.

to prevent further losses.

Treatment and prevention

Specific diseases

There is no treatment for cerebellar hypoplasia and affected calves should be culled because they pose

Cerebellar hypoplasia/hydranencephaly

a risk to seronegative/unvaccinated breeding cattle

The cerebellum is the most common CNS site for in-
in the herd. Biosecurity is an important aspect of

utero insult. The cerebellum is primarily concerned with herd health which is frequently overlooked on British

fine coordination of voluntary movement. In cerebellar

farms and worldwide. Increasingly, more British

dysfunction, limb movements are spastic (rigid), clumsy

dairy and beef herds will become self-contained

and jerky. Initiation of movement is delayed and may

with high health status, but in the meantime introduc-

be accompanied by intention tremors.

tion of disease by carrier animals is a major concern.

Depending upon the health status of the herd, pur-

chased cattle should either be screened for BVD/MD

Aetiology

antibody and antigen status upon arrival or vaccinated

*Cerebellar hypoplasia/hydranencephaly can be the
during the quarantine period. Establishment of a
result of an autosomal recessive condition or caused by
BVD/MD-free herd is presently included in numerous
in-utero infection with bovine virus diarrhoea/mucosal health schemes packages
in Britain but is not without
disease (BVD/MD; see p. 855) or Akabane virus.*

risk from introduction of infection with carrier-status

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*cattle and from adjacent herds via direct contact, water
within two days or so, although this may be difficult to
courses, carrion, etc (Chapter 57).*

*visualize against the surface of the brain. Over the cere-
bral hemispheres the exudate is largely confined to
the fissures. The brain is swollen in the acute stages of
Bacterial meningoencephalitis (see p. 251)*

*disease and may be so severe to cause displacement of
Bacterial meningoencephalitis most commonly affects
the cerebellum into the foramen magnum and coning.
calves 3 to 8 days old and is associated with poor im-
munoglobulin status and high bacterial challenge in the*

Signs and diagnosis

environment (Rings, 1987). The early clinical signs of lethargy, depression and failure to suck rapidly progress. The early clinical signs include lack of suck reflex, to hyperaesthesia, opisthotonus and death (Scott & Penny, 1993). Compulsive wandering and head pressing weakness (Green & Smith, 1992; Scott & Penny, 1993). have also been reported (Jamison & Prescott, 1987) but Fever is not a consistent feature. Affected calves stand are less common. The prognosis is poor despite intensive with the neck extended and the head held lowered. antibiotic treatment and supportive care.

Flexion of the neck is painful and may result in vocalization. In the absence of appropriate treatment the calf becomes increasingly weak over the next 6 to 12 hours

Aetiology

with an altered gait, and may be unable to stand. Dili-Bacteraemia, with subsequent localization within the gent stockmanship is important when checking young meninges, results from failure of passive antibody trans-

beef calves because the early clinical signs of lethargy
fer (Jamison & Prescott, 1987) and high levels of bac-
and weakness can easily be overlooked, especially when
terial challenge in the calf's environment. The disease
the calves are lying together in a group during adverse
is more common in those calves born indoors in unhy-
weather conditions. Depression and poor appetite
gienic calving boxes than in those born of cows at
are more readily detected in individually-penned dairy
pasture. Failure of the calf to ingest colostrum equiva-
calves. As the disease progresses there is lack of menace
lent to 7 per cent of its bodyweight within the first six
response and dorsomedial strabismus. Episcleral injec-
hours of life may be caused by calf and/or dam factors.
tion is often present at this stage. Depression progresses
Dystokia, weakness, nerve damage, overcrowded
to stupor, but the calf is hyperaesthetic to auditory and
calving accommodation, udder shape, dirty teats and an
tactile stimuli which may precipitate seizure activity
overly-anxious dam may all contribute to poor/delayed
during handling for intravenous antibiotic injection.

sucking behaviour by the calf. Dystokia, nerve damage, Opisthotonus is observed during the agonal stages of hypocalcaemia and weakness may result in prolonged disease approximately 24 to 36 hours after clinical recumbency of the dam. Poor nutrition, short dry period signs are first noted. There may be evidence of infection and mastitis cause reduced colostrum accumulation in the udder. Heifers' colostrum contains 25 per cent less polyarthritis, diarrhoea and omphalophlebitis. Infarcts in the liver and kidney are frequently observed at necropsy. In a study of ten calves with meningo-gastrointestinal tract or umbilicus. *Escherichia coli* and encephalitis, three calves had polyarthritis, three *Streptococcus* spp. were the more common isolates calves showed hypopyon and two calves had omphalophlebitis, but none exhibited diarrhoea (Scott & Penny, 1993).

The important differential diagnosis for bacterial meningoencephalitis is metabolic acidosis resulting

Pathology

from diarrhoea of 2 to 3 days' duration. Septicaemia

Septic foci are prone to localize within meningeal

presents with similar clinical findings to meningo-

vessels and are either rapidly walled off or may lead to

encephalitis as the meninges are a common site

diffuse suppurative meningitis. A combination of syn-

for bacterial colonization in neonatal calves.

ovial, meningeal and intraocular localizations is almost

Lumbar CSF is readily collected under local anes-

invariably of streptococcal origin; coliform infections

thesia from depressed or stuporous calves. Calves with

rarely cause endophthalmitis (Jubb & Huxtable, 1993).

bacterial meningoencephalitis frequently have a CSF

Once bacteria gain access to the leptomeninges there is

protein concentration above 1.0 g/l (deLahunta, 1983)

little resistance to spread and the inflammatory process

and greater than 2.0 g/l (Green & Smith, 1992; Scott & becomes diffuse.

Penny, 1993). The normal CSF protein concentration is

*In the first day or so the meninges are opaque and
<0.3g/l with a white cell concentration less than 0.012 ¥
hyperaemic with pus accumulating in the basal cisterns
109/l. There is also a marked increase in CSF total white
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*cell count in the order of 100-fold and a change in the
Septicaemia
predominant white cell type from lymphocyte to neu-
Calves with septicaemia frequently present with
trophil (neutrophilic pleocytosis). These CSF changes
clinical signs similar to bacterial meningoencephalitis
cause increased turbidity of the sample which is visible
because the meninges are simply one site for localiza-
upon gross CSF inspection and affords the veterinary
tion of blood-borne pathogens. The clinical course of
surgeon an immediate diagnosis. One to two days' delay
septicaemia is generally peracute and affected calves
in detailed laboratory examination of the CSF would
may be found either dead or in extremis within 12 hours.
undoubtedly result in death of the calf if left untreated.
Other sites of infection such as eyes (hypopyon),*

However, routine antibiotic therapy of all diarrhoeic gut (diarrhoea), joints (polyarthrititis) and umbilicus calves is not recommended because many antibiotics (omphalophlebitis) may be identified. Foci in the lung, slow the mitotic rate of enterocytes, which is an important factor in recovery from viral causes of diarrhoea. necropsy.

Bacteriological culture of lumbar CSF is often unrewarding and is unnecessary because of the sporadic nature of the disease and its causation by opportunist Enteric infections causing metabolic disturbances organisms.

Sequestration of water and electrolytes in the gut lumen, often without overt signs of diarrhoea, caused

Treatment and prevention

by the heat stable enterotoxin produced by K99 strains of *E. coli*, can result in recumbency in one to four day-A successful outcome necessitates early detection of

old calves which often rapidly progresses to coma and abnormal calf behaviour by the client and prompt clin-

death. Older calves with severe metabolic acidosis ical diagnosis by the veterinary practitioner supported secondary to enteric viral infection, typically 6 to 14 by visual inspection of lumbar CSF. Any delay in veterinary diagnosis and implementation of treatment deterioration of mental state to stupor. It can prove has a hopeless prognosis, but prompt treatment with difficult to undertake a thorough neurological examination, e.g. menace response and spinal reflexes, in such (Scott & Penny, 1993). Few broad-spectrum bactericidal antibiotics are capable of penetrating the intact blood–brain barrier, although it is commonly assumed that the disruption of the blood–brain barrier, which normal clear sample which allows immediate differentiation from bacterial meningoencephalitis (Scott &

of antibiotic penetration. This increased membrane
Penny, 1993).

permeability may allow sufficient passage of antibiotics
Treatment of acidotic calves with intravenous
to achieve minimum bactericidal concentrations
isotonic saline solution, spiked with 400–600 mEq of
(MBC) within the CSF. A peak CSF antibiotic con-
bicarbonate, is successful with a rapid improvement in
centration 10 to 30 times the effective MBC may be
demeanour and ability of the calf to stand within 6 to
more important than the maintenance of CSF antibiotic
12 hours.

MBC (Prescott & Baggot, 1988) and emphasizes that
high dose antibiotic therapy is indicated as soon as pos-

Brain abscess

sible after the onset of clinical signs of bacterial CNS
infection.

Neurological signs of a brain abscess typically appear

It is reported that the best treatment for Gram-
in 4 to 12 week-old calves following localization of
negative bacillary meningitis in man is the third

neonatal bacteraemia. Extension of infection through generation cephalosporins, in particular cefotaxime the calvarium following infection of the frontal sinus (Cherubin & Eng, 1986; Feldstein et al. , 1987) but not as a consequence of dehorning, while reported in the

data could be found in the veterinary literature relating literature, is uncommon in Britain.

to the use of ceftiofur in the treatment of infectious bovine neurological diseases under field conditions.

Aetiology

Chloramphenicol is no longer permitted in food-Arcanobacterium pyogenes is the most common isolate producing animals in many countries; there are no field from brain abscesses.

studies which report the clinical efficacy of its closely-related successor florfenicol. Other antibiotics which

Pathology and pathogenesis

could be administered for bacterial meningoencephalitis include trimethoprim-sulphonamide combination or

Brain abscesses are usually of haematogenous ceftiofur.

origin. Most cerebral abscesses are small and track

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inward to the white matter rather than out to the

this condition from cervical spinal lesions which often meninges.

appear at around 6 to 12 weeks old.

Signs and diagnosis

Unilateral middle ear infections/vestibular

Affected calves are usually dull and depressed with a syndrome (see also p. 252)

sluggish gait. These calves have a poor appetite and as a consequence appear ill thriven. Approximately 90 per

The vestibular system helps to orientate the animal in cent of efferent nerve fibres cross at the optic chiasma;

its environment with respect to gravity, and maintains

therefore animals with a left-sided cerebral abscess

position of the eyes, trunk and limbs during movement.

are blind in the right eye but the pupillary light reflex

Unilateral middle ear infections (otitis media) are not

is normal. As the abscess continues to grow the animal

uncommon in growing calves and yearlings and usually

becomes increasingly depressed, sometimes over many months. Ipsilateral compulsive circling may lead to the often following respiratory disease.

calf becoming stuck with its head in the corner of the pen or fence. Once in this position, the calf may stand

Signs and diagnosis

motionless for long periods of time, sometimes hours.

The gait is often sluggish and ataxic. There is ipsilateral

The major clinical sign in unilateral peripheral vestibular disease is an ipsilateral head tilt of 5° to 10° down

head carriage must not be confused with a head tilt.

to the affected side. There may be loss of balance,

The calf may display ipsilateral postural deficits such as leaning and movement/circling toward the affected

knuckling of the fetlock joints. In most cattle there

side. When walking, cattle tend to drift toward the

are no cranial nerve deficits. There is frequently an

affected side. During the early stages of unilateral

increased protein concentration and elevated white cell

peripheral vestibular lesions there is spontaneous horizontal concentration in lumbar CSF, which reflect any accompanying suppurative meningitis.

side of the lesion. There is often ipsilateral ventral deviation of the eye (eye drop), which is exaggerated when the head is raised. The facial nerve travels close to the Treatment and prevention

middle ear and facial palsy is often seen in conjunction Treatment with high dose penicillin (minimum 44 000 with otitis media.

iu/kg b.i.d.) for 10 days may halt progression of the

In central vestibular disease the nystagmus may be abscess but the long term prognosis is very poor. Pre-horizontal, vertical or rotary, and there may be ipsilateral limb weakness. Depression indicates involvement ensuring adequate passive antibody transfer and reduction of the reticular formation.

ing environmental bacterial challenge by maintaining

It is important to differentiate vestibular lesions from

*good hygiene standards in the calving accommodation.
listeriosis because of the different treatment regimens
and control measures.*

Cerebellar abiotrophy (postnatal degeneration)

Treatment and prevention

Cerebellar abiotrophy is an inherited condition which

The bacterial infection responds well to five to seven

has been reported in Holstein calves in the UK. The

consecutive days' treatment with 44 000 iu/kg procaine

cerebellar abiotrophies are considered to be familial

penicillin s.i.d. The condition occurs sporadically and

and degenerative but not congenital. Similar clinical

there are no specific control measures.

signs have been reported in a six month-old Limousin

cross heifer caused by selected cerebellar degeneration

(Woodman et al. , 1993; see also pp. 178, 893).

Polioencephalomalacia

(cerebrocortical necrosis, CCN)

Signs and diagnosis

(see also p. 261)

*Clinical signs of pelvic limb ataxia, especially when
This is a sporadic condition that affects young growing
turning quickly, wide-base stance and hypermetria
cattle. It is characterized clinically by dullness, bilateral
appear from around six weeks of age and are slowly
lack of menace response, dorsomedial strabismus
progressive over many months, leading eventually to
and hyperaesthesia to auditory and tactile stimuli,
recumbency by one year of age. The preservation of
progressing over days to lateral recumbency and
strength and chronicity of the condition differentiates
opisthotonus.*

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Aetiology

of menace response and dorsomedial strabismus.

Affected animals are hyperaesthetic to tactile and audi-

Healthy ruminants obtain their requirements from thi-

tory stimuli. As the disease progresses animals often

amine synthesized by the rumen flora. Free thiamine is

head press into corners and there is frequent bruxism

readily absorbed and is actively phosphorylated to

(teeth grinding). Twitching, muscular tremors and intermittent opisthotonus are evident, followed by recumbency and clonic convulsions with intermittent periods of spasticity and terminal flaccidity. Untreated cattle die within three to five days.

pyrophosphate is also a coenzyme in the transketolase

Diagnosis of PEM is based primarily on the history, reaction of the hexose monophosphate shunt and the clinical signs and response to intravenous thiamine alternative, glycolytic, pentose phosphate pathway.

administration. Laboratory tests are of equivocal value

Outbreaks of PEM have been described in cattle, and response to treatment of early cases is commonly sheep (Low et al. , 1996), when fed diets high in sulphur.

employed as a diagnostic indicator by practitioners.

Changes in lumbar CSF include a slightly increased

Pathology

protein concentration, but this result is of little diagnostic value.

The brain usually appears pale and swollen with flattened gyri, which in the frontal, dorso-medial and parietal regions often show a patchy, bilaterally

Treatment and prevention

symmetrical, yellow discoloration. There is swelling of

The response to large doses (10–15 mg/kg) of thiamine

the cingulate and parahippocampal gyri, which may

hydrochloride given intravenously early in the disease

herniate beneath the tentorium cerebelli. The posterior

is usually evident within 24 hours. The thiamine should

vermis may have herniated through the foramen

be repeated within 4 to 6 hours, then twice daily for

magnum and appear necrotic. The cut surface of the

three consecutive days. Full clinical recovery may take

cerebrum reveals that the necrotic cortical tissues have

one week. Intravenous administration of dexametha-

a laminar configuration and may have separated from

sone (1.0 mg/kg) at first presentation may aid recovery.

the underlying white matter. When viewed in ultravio-

*Prevention of PEM involves the maintenance of
let light (wavelength 365 nm) affected regions of cortex
normal rumen fermentation with adequate dietary fibre
have a bright white autofluorescence, which has been
ensuring production of volatile fatty acids which curtail
attributed to ceroid lipofuscin (Little, 1978).*

*the growth of thiaminase-producing organisms. The
Histologically, there is increased prominence of cap-
efficacy of metaphylactic thiamine injections during
illary endothelium (neovascularization) and dilatation
an outbreak of dietary sulphur-induced PEM in cattle
of perivascular spaces with occasional small perivas-
remains equivocal, although this did appear to halt
cular haemorrhages. Astrocytes and neurones show
appearance of new clinical cases in housed lambs (Low
hydropic changes and nuclear pyknosis, which proceeds
et al. , 1996).*

*in time to a laminar necrosis. In well established cases
there is a massive influx of macrophages into the
necrotic areas and adjacent leptomeninges.*

Listerial encephalitis (see also p. 156)

Listerial encephalitis is the result of infection of the brain substance with Listeria monocytogenes.

Signs and diagnosis

PEM occurs sporadically, affecting weaned calves and

Aetiology

young feedlot cattle. It is associated with diets low in fibre, although cases have occurred in animals grazing

Listeria monocytogenes is a microaerophilic Gram-

lush aftermath. Under these circumstances, changes

positive, flagellated coccobacillus which is present in a

occur in the rumen flora that permit multiplication of

wide range of moist environments and may cause

micro-organisms such as Bacillus thiaminolyticus and

disease in man and a variety of domestic species.

Clostridium sporogenes, both of which synthesize thi-

Listeriosis occurs sporadically in cattle, where most

aminase type 1, thereby inducing thiamine deficiency.

cases are associated with feeding poorly fermented

During the early stages of PEM there is frequently a

silage during the winter months. Cattle show a similar

brief period of diarrhoea before nervous signs appear.

age incidence to sheep with the majority of cases affected. Affected animals are dull and may isolate themselves from others in the group. There is high head carriage and affected cattle may stagger. There is bilateral loss of vision. Cattle are less susceptible to listeriosis than sheep. Rarely are outbreaks of listeriosis encountered in cattle.

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Pathology

Parosmia is uncommon in cattle with listeriosis. Hypoglossal nerve dysfunction leading to tongue paralysis

The pathogenesis of listerial encephalitis involves centripetal passage of the organism along branches of the trigeminal nerve from minor breaches in the buccal mucosa. Intracranial pathology initially consists of a small focus of necrosis in the lateral part of the pons, progressing to coma. A moderate increase in CSF protein concentration

and protrusion is more commonly observed in ovine

listeriosis.

The course in untreated cattle lasts 10–14 days,

A moderate increase in CSF protein concentration

which is associated with activation of microglial cells in the range 0.8 to 2.0 g/l is observed in meningo- and astrocytes followed by an influx of monocytes and encephalitis caused by L. monocytogenes (Rebhun & a few neutrophils. The initial focus may be unilateral deLahunta, 1982).

but thereafter (intraneural) spread results in micro- Listeriosis is frequently associated with the feeding abscesses forming bilaterally in the mid brain and of silage and may be suspected on the basis of the lower medulla.

clinical signs and supported by findings of an elevated CSF protein concentration and monocytic pleocyto-

Signs and diagnosis

sis. Vestibular lesions (p. 896), lead poisoning (p. 944),

The rectal temperature of affected cattle is within the brain abscess, BSE (p. 909) and nervous acetonaemia range 38.5 to 39.2°C. There is reduced appetite with a (p. 795) should be considered amongst the differential gaunt appearance, marked fall in milk production in diagnosis.

lactating cattle and weight loss. Loss of saliva leads to Isolation of the causal organism from the brain may rumen impaction, causing abdominal pain manifest as require extended periods of 'cold enrichment', but the an arched-back stance and frequent bruxism.

neurohistopathology is usually sufficiently characteris- As most of the cranial nerve nuclei are present within tic to permit firm diagnosis.

the brainstem, ascending infection of the trigeminal nerve by L. monocytogenes is characterized by multiple Treatment and prevention

unilateral cranial nerve deficits, depression and, in some cases, circling to the affected side. Ipsilateral hemipare- Penicillin or trimethoprim-sulpha remain the antibi- sis may also be present and should be differentiated otics of choice for listeriosis. Oxytetracycline is not con- from ataxia observed in cattle with BSE.

sidered an appropriate antibiotic for listeriosis because Involvement of the trigeminal nucleus results in of its large molecular size, although good results have paralysis of the cheek muscles and decreased facial skin

been claimed. A minimum dose rate of 44 000 iu/kg sensation. Facial palsy is evident as drooped ear, procaine penicillin injected intramuscularly b.i.d. must drooped upper eyelid (ptosis) and flaccid lip. Occasionally, there may be paralysis and protrusion of the iu/kg penicillin G injected intravenously b.i.d. on the tongue. Exposure keratitis may result from paralysis of first day. Penicillin dose rates as high as 300 000 iu/kg the orbicularis oculi muscle. Loss of cheek and lip have been recommended for the first day of antibiotic muscle tone result in drooling of saliva from the therapy because it is essential to exceed MICs by 10 to affected side of the mouth. Depression is attributed to 30 times to achieve a successful outcome.

a lesion in the ascending reticular activating system. A Loss of saliva may lead to dehydration and metabolic head tilt toward the affected side is an inconsistent acidosis. Care must be taken when replacing fluids by finding. Circling can be observed with involvement of orogastric tube because contraction of the rumen

the vestibulocochlear nucleus. Cattle frequently display caused by anorexia of some days' duration may result a 'propulsive tendency' and may be found with the head in passive regurgitation of these fluids around the oro-forced through a gate, under a feed trough or wedged gastric tube. The amount of fluids administered in this across the front of a cubicle. Indeed, dairy cows have way should be restricted to 15 to 25 litres four to six barged through the milking parlour under the cows times daily. Transfaunation with rumen liquor from a ahead of them causing chaos and often falling into the healthy cow may promote rumen function and aid milking area; such behaviour in previously quiet cattle recovery.

led to misdiagnosis of BSE. Caution must be exercised There are no published studies which have reported when cattle are found with the head trapped under a the efficacy of prophylactic antibiotic administration in feed barrier, etc., because casual examination may the face of an outbreak of listeriosis, but such epide-attribute the facial palsy to trauma but this would not

miology is unusual in cattle.

explain the unilateral loss of jaw tone and facial skin

Listeriosis occurs sporadically in cattle and is pre-sensation.

vented by management practices which ensure the

Loss of cranial nerves IX, X and XII function results

making and storage of high quality silage. Soil contam-

in stertorous breathing and dysphagia, but this presen-

ination is limited by rolling grass fields at the beginning

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of the growing season. Good fermentation is guaran-

macrophages. In cattle with long survival periods these

teed by cutting grass at an early growth stage

cortical lesions may be extensive, and similar lesions

(digestibility value >72) when it contains a high fer-

may be present also in the thalamus and hypothalamus,

mentable sugar content, and the use of various silage

medulla and spinal cord.

additives, whether sugars, organic acids or bacterial cul-

tures. Compaction of the silage clamp is important to

Signs and diagnosis

expel all air followed by air-tight sealing to prevent aerobic bacterial multiplication. Poor quality or spoiled silage should be discarded. Irrespective of the rate of uptake of lead, the clinical signs of intoxication are sudden in onset and characterized by behavioural changes. During the early stages of lead poisoning affected cattle become isolated and depressed. These animals are hyperaesthetic to tactile and auditory stimuli and may show muscle twitching, encephalitis by vaccination is not an established procedure in ruminant species.

of lead poisoning affected cattle become isolated and depressed. These animals are hyperaesthetic to tactile and auditory stimuli and may show muscle twitching,

Lead poisoning (see also p. 944)

especially of the palpebral muscles. Affected cattle are Cattle, through their innate curiosity, indiscriminate blind and may head press forcibly into corners and feeding habits and relative susceptibility to lead, are the against walls. The disease progresses and cattle become species most commonly poisoned by lead compounds. frenzied, bellow, stagger and crash into obstacles. There Lead poisoning in cattle is characterized by an acute may be signs of abdominal pain including kicking at the

encephalopathy.

abdomen and frequent bruxism (teeth grinding). Bloat is often seen and attempts to alleviate this problem often precipitate frenzy. Death may occur suddenly or

Aetiology

within days.

The common sources of lead include discarded storage

The diagnosis of lead poisoning is suspected on the

batteries, flaking old lead-based paint, putty, asphalt

basis of clinical signs and the presence of a source of

roofing materials and used motor engine oil. Intoxica-

lead. Confirmation depends upon the histopathological

tion may result from a single large dose of lead or from

findings and the chemical determination of the concen-

ingestion of smaller amounts over a long period of time.

tration of lead in tissues. In kidney and liver concen-

In both forms the neurological signs are acute in onset

trations >4 ppm wet weight and blood values in excess

and similar in type.

of 0.3 ppm are considered diagnostic.

Pathology

Treatment and prevention

The severity of neuropathological change in lead poisoning correlates more closely with survival time than duration probably have extensive neuropathological changes and are unlikely to respond to treatment. severe in cases that survive longest.

However, if a source can be identified, in-contact Grossly, the brain appears pale and slightly swollen animals at risk from a single large dose may be with flattened gyri, but without herniation of the hippocampal gyrus beneath the tentorium or cerebellar to precipitate and remove lead from the alimentary tract and injected with calcium disodium edetate at 110 mg/kg by slow intravenous infusion on alternate days cut surface may show separation of these yellow zones for three treatments. The similarity of the pathological

of cortical tissue from the underlying white matter at changes to those of PEM has encouraged the use of the tips of the gyri with actual cavitation in the deeper thiamine (10–15 mg/kg intravenously) along with cortical laminae. In cases of longer survival these EDTA therapy and is reportedly beneficial. Control of changes may extend to the tips of almost all gyri and convulsions proves very difficult because drugs such as extend down the sides of the convolutions (Christian & diazepam have such a short half-life in cattle. Chloral Tryphonas, 1971).

hydrate sedation may be attempted but this drug is not Histologically, the earliest changes in affected gyri licensed for use in cattle in Britain. Pentobarbitone comprise swelling and prominence of capillary is frequently used to control seizure activity in cattle endothelial cells, which is sometimes referred to as with acute hypomagnesaemia and could be used in neovascularization. Swelling of astrocytes, and fine emergency situations for lead poisoning to control microvacuolation of the neuropil, also occur at an early

convulsive episodes.

*stage, advancing to spongy transformation with
Prevention is a matter of good management, not
necrosis of neurones, malacia and infiltration by
allowing access by cattle to sources of lead.*

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Thromboembolic meningoencephalitis

*tation reveals widespread wheezes. Pleural friction rubs
(TEME) (see also p. 240)*

*are described in some reports but significant pleurisy
severely restricts movement of the underlying visceral
Under conditions of stress or intercurrent disease a ful-
pleura such that no sounds are generated by this
minating *Haemophilus somnus* bacteraemia may occur
localized pathology. Localization of bacteria within
with disseminated intravascular coagulation. Throm-
joints causes joint effusion and swelling with associated
boemboli may lodge in meninges, brain, muscles and
severe lameness which may result in recumbency.
joints causing depression, blindness and recumbency
Retinal haemorrhages, hyphaema and hypopyon have*

with muscular weakness. Retinal haemorrhages may be also been described in some cattle following localization of the bacteraemia.

Aetiology

Treatment and prevention

Thromboembolic meningoencephalitis (TEME) is a

Prompt detection of early neurological signs is important to ensure early antibiotic therapy; therefore cattle is a significant problem in feedlot cattle in North

should be regularly inspected by trained stockmen

America associated with stresses of weaning, long journey during the high risk period following introduction of

neys (often thousands of miles), co-mingling in auction groups of cattle to the feedlot. H. somnus is susceptible markets and the feedlot, changes in diet, husbandry,

to a wide range of antibiotics in vitro including oxytetracycline, penicillin and ampicillin; however, oxytetracycline does penetrate the blood–brain barrier well.

describe a range of procedures undertaken on admission to the feedlot and may include castration, dehorning, anthelmintic treatment, ectoparasite treatment, and vaccination against a wide range of viral and bacterial pathogens. Penicillin offers the most cost effective treatment at a dose rate greater than 44 000 iu/kg b.i.d., which should be administered for at least five consecutive days.

Vaccination is not undertaken in Britain due to the pathogens including parainfluenza 3 virus, bovine

sporadic nature of *H. somnus* infections where respiratory syncytial virus, infectious bovine rhino-

tory disease is the more common clinical presentation.

tracheitis, *Mannheimia haemolytica* and *Pasteurella*. Prophylactic injection with long-acting oxytetracycline

multocida, implantation with hormonal growth pro-

and in-feed medication have been described in North

motors and ear tagging. These procedures and stresses

American feedlot situations, but these are of doubtful

render the weaned beef calf susceptible to a wide

use. Greater attention must be paid to the sourcing

range of respiratory tract and other infections. Infection

of feedlot cattle. Preconditioning including weaning,

with *H. somnus* probably occurs through the respiratory tract from asymptomatic carriers to non-infected concentrate feedstuffs must be undertaken at least three weeks before sale and transportation. Cattle stem, spinal cord, synovial membranes, pleurae and sourced directly from ranches have a much lower incidence of respiratory disease than those from auction respiratory disease, which is all too common in large markets or sale barns. Unfortunately, such sensible feedlots where morbidity rates may exceed 40 per cent husbandry practices will not be undertaken until a significant premium is paid for such calves. The use of processing.

prophylactic antibiotic administration on admission to feedlots in North America requires urgent review.

Signs and diagnosis

The neurological signs of TME are sudden in onset

Aujeszky's disease

and death may occur within 36 hours of onset. Affected cattle are pyrexia (40 to 42°C), anorexic and depressed.

Aujeszky's disease is a herpes virus infection principally

The depression may extend to somnolence and has led

of pigs, which can be transmitted to most other mam-

to the term 'sleeper calves'. Proprioceptive deficits are

malian species including cattle.

commonly observed and affected cattle appear ataxic.

Cattle may be found recumbent in the pen with the

Aetiology

head averted against the chest and are unable to raise

themselves. Blindness, nystagmus and strabismus are

Infections in cattle are generally sporadic and result

variably present. Terminally, there is opisthotonus and

from contact with infected pigs, foodstuffs or other

coma. Lesions are also present in the lungs and auscul-

materials contaminated with virus. The disease in cattle

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is extremely severe with self-mutilation necessitating

and transmitted by the bite of an infected animal. It is

immediate slaughter for welfare reasons.

manifested by irritability, mania, hydrophobia and paralysis. It is usually fatal, although recoveries have

Pathogenesis and pathology

been documented.

Infection in cattle is generally by the oronasal route.

Centripetal intra-axonal transport of virus causes first a

Aetiology

severe ganglioneuritis followed by meningoencephalitis

or myelitis. Lesions are most severe in the olfactory lobe,

The causal agent is a delicate rhabdovirus readily

hippocampus and cerebellum, whereas infection of a

destroyed by disinfectants and desiccation. It causes

spinal peripheral nerve results initially in a segmental

pathological changes only in nervous tissue. Rabies

myelitis. Grey matter is principally affected with degen-

occurs worldwide except in certain island territories

erative changes in nerve cells and astrocytes in which

(Australia and New Zealand) and a few countries from

multiple, small, granular, eosinophilic, intranuclear inclu-

which it has been eradicated, such as the British Isles

sion bodies may be found. The inflammatory response is and Scandinavia. In the western hemisphere the infection is endemic in dogs, foxes, wolves, skunks, raccoons and bats of several species. Infection is transmitted with the bite of a rabid animal.

Signs and diagnosis

The clinical course is short, rarely extending beyond 48

Pathology and pathogenesis

hours in adult cattle, whilst calves may die without obvious prior signs of illness. Usually, however, there is a brief period of excitement with high fever, bellowing, infected, usually on the hindquarters or limbs, from the bite of a rabid fox, dog or bat. Following local replication, virus travels centripetally in the axoplasm of a hyperpnoea, salivation and compulsive licking of the nostrils. Intense pruritus of the neck, trunk or hind legs and aggressive behaviour accompanied by trembling, peripheral nerve to reach the spinal cord and thence to

is accompanied by frantic efforts to relieve the itch to the brain. En route it replicates in neurones. From the point of self-mutilation. Affected animals may become bloated and there is incoordination and, terminally, recumbency, convulsions and coma. and lachrimal glands and is present in their secretions. Except in calves, the clinical signs are usually distinctive enough for a provisional diagnosis of minimal or absent.

Aujeszky's disease to be reached. Confirmation is dependent upon demonstration of the characteristic

Signs and diagnosis

neuropathology or virus isolation from nervous tissue.

The incubation period of rabies in cattle varies from

Treatment and prevention

about two to three weeks to several months. Clinical signs classically occur in two distinct forms, the mild There is no effective treatment and affected cattle are

paralytic or dumb form and the furious form, dependent upon the pathogenicity of the strain of virus. In paralytic rabies there may be partial loss of sensation in the hind legs, knuckling of the fetlocks, locomotor weakness and are effective in preventing disease in pigs. Attenuated and inactivated vaccines are available and paralysis of the tail. Flaccid dilatation of the anus. However, they will not protect pigs from infection with field virus, which will replicate and be shed for some time after infection. Thus control in pigs must be maintained by serological testing and slaughter. This generalized paralysis with death probably due to respiratory failure. In the furious form the animal is

in Britain. The disease in cattle and sheep is notifiable hyperaesthetic and sexually excited. It bellows hoarsely in Britain and in some other countries.

and becomes violently aggressive towards people, other animals and inanimate objects. Purposeful attacks,

Rabies (see Chapter 70)

however, are frustrated by rapidly progressive incoordination and ataxia. Death occurs quickly following

Rabies is a neurotropic viral disease that can affect all warm-blooded animals. The virus is excreted in saliva

recumbency and paralysis.

Neurological Disorders • 909

Cattle are normally 'end hosts' with respect to rabies,

Some change in exposure of cattle to infection in

although human infection may follow manual examination

1981–82 resulted in BSE appearing in the cattle population of the oral cavity in which virus in saliva is

inoculated into scratches caused by teeth.

an end to hydrocarbon fat extraction of rendered material

Clinical diagnosis of rabies is difficult, especially in

rial (NB the scrapie agent is lipotrophic) has been suggested as an important factor in BSE epidemiology.

ity of rabies should be borne in mind in the differential

Subsequent studies have demonstrated that operating diagnosis of bloat, nervous acetonemia (p. 795), listeriosis in many rendering plants during the early period of viral encephalitis (p. 904) and bovine spongiform

1980s would have been incapable of destroying the BSE agent. Rabid cattle invariably die. To date there are no experimental data which and fluorescent antibody staining of impression smears demonstrate that scrapie can be transmitted to cattle by from appropriate parts of the CNS will establish the oral route.

diagnosis rapidly.

BSE was made a notifiable disease in the United Kingdom on 21 June 1988. Compulsory slaughter with destruction of carcass (incineration since 1991) with

Treatment and prevention

compensation was introduced on 8 August 1988. On 18

No treatment of clinical cases should be attempted, nor July 1988 the 'ruminant feed ban' was introduced which should they be euthanased prematurely as this may prohibited the feeding of ruminant-derived protein to prejudice the post-mortem diagnosis. Post-exposure ruminants. Brain, spinal cord, tonsil, thymus, spleen and vaccination is a routine procedure in man, but in cattle intestine (specified bovine offals) of cattle over six clinical disease and death would probably occur before months old were no longer permitted to be sold for an effective immunity had time to develop.

human consumption. In September 1990, specified bovine offals were banned from all other animal feeds.

The introduction of a ban on the inclusion of

Bovine spongiform encephalopathy (BSE)

ruminant-derived protein in cattle rations in July 1988

Bovine spongiform encephalopathy (BSE) was first has resulted in a reduction in the incidence of BSE in reported in the United Kingdom in 1987 as a previously younger cattle since 1991 (Wilesmith & Ryan, 1993) unrecognized spongiform encephalopathy affecting

which continued throughout the 1990s and into the 2000s.

dairy cows (Wells et al. , 1987). While the first documented BSE cases were reported in 1987, retrospective

Aetiology

case record studies suggest BSE occurred as early as 1985. Some cattle practitioners suggest that clinical All the epidemiological evidence presently available cases occurred sporadically over the previous 25 years, strongly suggests that the geographically widespread but there is no supporting evidence in limited studies of incidents of BSE are not the result of cow-to-cow trans-archived pathology material.

mission but conform to the concept of a single source Bovine spongiform encephalopathy is a new member epidemic, with concentrate feedstuffs containing animal of a group of subacute transmissible spongiform protein as the probable source. Many farms have experienced only one BSE case although many cattle were properties, including long incubation period but exposed to the same feed source. In some herds

relatively short clinical course which is invariably fatal, outbreaks have involved the majority of animals in a progressive neurological signs with rapid deterioration particular cohort (Winter et al., 1989). It is possible that terminally, spongiform change visible under light

the transmissible agent was very unevenly distributed microscopy and presence of scrapie associated fibrils in the feed because, unlike sheep, host genotype on electron microscopy. All members are transmissible appears to have little effect upon either susceptibility to laboratory animals and many other species by or the length of the incubation period.

intracerebral injection and other routes. This group An alternative postulate to the feeding of scrapie- includes scrapie of sheep and goats, chronic wasting contaminated diets as the origin of BSE is that of disease of mule deer, transmissible mink encephalo-spontaneous prion mutation giving rise to histopatho-pathy of ranched mink, Kuru and Creutzfeldt–Jakob logical changes and clinical disease of a spongiform disease(CJD) of man and new variant Creutzfeldt–

encephalopathy of cattle (Scott et al. , 1995). The origin Jakob disease (v-CJD) of man.

of the BSE epidemic could, therefore, have been the

The postulate that BSE resulted from the ingestion inclusion of infected material from 'spontaneous cases'

of meat and bone meal contaminated with a scrapie-

of BSE subsequent to changes in the rendering process,

like agent (Morgan, 1988) has been supported only indi-

and not contamination of cattle rations with the scrapie

rectly by epidemiological data (Wilesmith et al. , 1988).

agent. The simultaneous widespread appearance of

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BSE in the UK could be explained, in part, by the sale

neurones of certain brain stem nuclei (Wells et al.,

and movement of meat and bone meal concentrate

1987). There is fine vacuolation of the ground substance

derived from cattle. In this respect it is interesting to

whilst neurones and neurites develop one or more well-

note that the origins of chronic wasting disease of mule

defined intracytoplasmic vacuoles, which may distend

deer and elk have not been determined. It is possible

the cell body and processes. The nuclei principally that a scrapie-like disease did occur in cattle from involved are the dorsal nucleus of the vagus, the nucleus another species (not sheep) and until transmission of the solitary tract, the reticular formation, the vestibular studies demonstrate classical BSE in cattle after feeding lar and spinal trigeminal nuclei and in the mid brain the scrapie-contaminated feedstuffs this lack of conclusive red and oculomotor nuclei.

evidence remains a serious concern.

An additional pathological characteristic of the trans-
Reports of transmissible mink encephalopathy
missible spongiform encephalopathies is the presence (TME) in ranches in Wisconsin, fed from 95 per cent downer or dead dairy cattle and a few horses in extracts of brain prepared for electron microscopy of fibrils 100–500 nm in length that are known as scrapie (Marsh & Hartsough, 1985), raise the question whether associated fibrils (SAF).

an unrecognized spongiform encephalopathy also occurs sporadically in cattle in the USA. However,

Signs and diagnosis

experimental exposure of mink to BSE produced an encephalopathy with minimal resemblance to TME

Bovine spongiform encephalopathy affects cattle 2 to

(Robinson et al. , 1994). BSE has recently (2003) been 13 years of age, with peak prevalence in the four to five

confirmed in Canada. The worldwide sporadic occur-

year age group. Early epidemiological investigations

rence of CJD, with no obvious reservoir of infection or

(Winter et al., 1989) revealed that affected cattle could mode of transmission except for iatrogenic cases, may

have had access to feedstuffs containing ruminant-

support the postulate that spontaneous changes of the

derived protein only during the first four months of life.

prion, eventually resulting in clinical disease, do occur.

The disease is less common in beef suckler herds

The existence of scrapie in many countries of the world

because most replacement heifers are reared naturally

with large sheep populations means that sporadic cases

with their dam and receive no concentrate feeding.

resulting from spontaneous changes in the prion would

Disease has been commonly reported in beef herds

not be differentiated from those scrapie cases originating where replacement heifers were sourced from dairy farms from horizontal or vertical transmission of the agent. In countries that are scrapie-free, such as New Zealand and Australia, 'spontaneous cases' of scrapie, occurring at approximately 0.1 per 100 000, appears to be a reflection of population size, the majority of cases occurring in Friesian/Holstein cattle.

Early signs include chronic weight loss and decreased milk yield over four to six weeks (Aldridge et al. , 1988; Scott et al. , 1988a, 1989a). Affected cattle are often isolated from other cattle in the field. Cows stand with an arched (roached) back and a wide-based stance.

of agriculture and unwillingness of sheep producers to employ veterinary services for single sheep. In this regard it is widely regarded that the majority of BSE

political as well as animal health reasons.

an arched (roached) back and a wide-based stance.

Experimental transmission of spongiform

There is frequent independent ear movement with the encephalopathy after intracerebral injection or oral ears often directed backwards towards the poll. The dosing with brain homogenate derived from cattle with abdomen appears drawn up with sunken sublumbar BSE has been confirmed in sheep and goats (Foster fossae consistent with reduced appetite. During the et al. , 1993) and by a combination of intravenous and latter stages, affected cattle spend very little time rumi-intracerebral injection to cattle (Dawson et al. , 1990).

nating. There is a profound change in attitude; affected

Intracerebral injection of calves with strains of scrapie

cattle become anxious, apprehensive and hyperaes-

agent from five flocks in four states in the USA pro-

thetic to tactile, auditory and visual stimuli. When trot-

duced neurological signs, brain lesions and distribution

ting there is marked pelvic limb hypermetria and ataxia,

of prion protein distinct from BSE (Cutlip et al. , 1994), but normal muscle strength. When in a group, BSE

but these differences could be explained, in part, by

cattle frequently push other cows along with vigorous

strain variation in scrapie and route of infection.

head butting. Cows have considerable difficulty when encountering obstacles such as steps, ramps and narrow gateways and will frequently attempt to jump over low

Pathology and pathogenesis

objects such as slurry scrapers and run through gate-

The pathological changes consist of bilaterally symmet-

ways, etc. As the condition progresses cattle frequently

rical degenerative changes affecting the neuropil and

slip, especially when turning on wet concrete, have great

Neurological Disorders • 911

difficulty raising themselves and excoriation of the carpi

clinical presentation is claimed to be not dissimilar to

results (Scott et al. , 1995). When confined for veterinary BSE (Jeffrey & Wilesmith, 1996). The incidence of

examination,

stimulation often provokes violent

disease appears to be more common in beef suckler

kicking (ballism) with the pelvic limbs and bellowing.

cows with a mean age of eight years old compared to

Repetitive contractions of individual muscle groups

four to five years old in BSE. The disease is more commonly reported in Scotland than England, despite region, ventral neck and proximal regions of the pelvic approximately similar numbers of beef herds.

limbs. There is rapid progression of clinical signs and Brainstem chromatolysis and degeneration of brain-cattle may become weak and recumbent within two to stem neurons are found in all cases and are generally ten weeks of clinical signs first being detected.

associated with a florid and severe lesion (Jeffrey & Differential diagnoses include listeriosis, hypomagnesaemia, space occupying lesions, lead poisoning, Wilesmith, 1996). Scrapie-associated fibrils have not been found in the brains of cattle with idiopathic brain-organophosphorous poisoning, hepatoencephalopathy stem neuronal chromatolysis nor is there evidence of and rabies. In common with the other transmissible disease specific PrP (prion protein).

spongiform encephalopathies, there is no immune The cause of this disease remains unknown but a

system response to infection; therefore routine toxic or metabolic insult has been suggested (Jeffrey & Wilesmith, 1996).

diseases from the differential diagnosis list. There is no intrathecal inflammatory response (Scott et al. , 1989b),

Sporadic bovine encephalomyelitis

which assists the clinician to exclude conventional

(SBE, Buss disease)

infectious agents from the differential diagnoses (e.g. listeriosis). Confirmation of diagnosis depends upon a Sporadic bovine encephalomyelitis is a generalized neuropathological examination.

inflammatory disorder of serous membranes, synoviae and vascular endothelium. It has no specific neurotropism, the neurological signs being a consequence of

Treatment and prevention

inflammation of the mesodermal elements in the CNS.

There is no effective treatment for BSE. Notification and elimination of affected animals have been in

Aetiology

operation in the UK since 1988. The occurrence of Sporadic bovine encephalomyelitis is a specific disease approximately 1700 BSE cases per annum 12 years of cattle and buffalo caused by a strain of Chlamydia after the implementation of the ban on inclusion of psittaci. The disease has been observed in the USA, ruminant-derived protein in ruminant rations suggests Eastern Europe, the Middle East, Japan, Australia and that vertical/maternal transmission of BSE does occur. South Africa.

A maternal effect resulting in an increased number of BSE cases in the progeny of affected cattle compared to controls was demonstrated (Wilesmith et al. , 1997), Pathology and pathogenesis

but the study design was flawed because the progeny The disease is usually fatal within four to five days. The were probably exposed to dietary BSE. While the gross post-mortem findings include a serofibrinous control measures adopted for BSE control, and human peritonitis, pleurisy and pericarditis. A serofibrinous safety, were closely modelled on data from scrapie

exudate is also found over the surface of the brain, research, regulatory authorities failed to take account especially the cerebellum and medulla. Histological of the possibility of vertical/maternal transmission and examination of the brain reveals a predominantly histiocytic and plasma cell infiltration of the meninges. Monetary expediency has proved extremely costly. Offspring of affected cattle are now culled in the UK.

Signs and diagnosis

Calves less than six months old develop a staggering,

Idiopathic brainstem neuronal

stiff gait with circling and stumbling. Affected calves are

chromatolysis of cattle

dull and depressed and may exhibit muscle tremors.

Idiopathic brainstem neuronal chromatolysis of cattle

About 70 per cent of infected animals recover slowly.

has numerous clinical features similar to BSE (Jeffrey

Clinical diagnosis of SBE may be confirmed by rising

& Wilesmith, 1992) and 8 to 27 cattle with this

titres of group-specific chlamydial antibody in comple-

condition have been mistakenly slaughtered annually
ment fixation or enzyme-linked immunosorbent assay
(Jeffrey & Wilesmith, 1996). The consistent clinical
(ELISA) tests in the live animal or by isolation of the
signs include tremor, weight loss and ataxia, but the
organism from brain and lymph nodes.

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Treatment and prevention

the lower limb joints. Stimulation of the skin over the
thoracic wall and flank with a blunt object produces a
Successful therapy depends upon treatment early in the
normal local response of muscle contraction (pannicu-
course of the disease using high dosages of tetracycline
lus reflex). In the case of a spinal lesion the skin caudal
(50 mg/kg s.i.d.) for five to seven days.

to the affected area of the cord has a reduced (hypal-
gesic) response, with a possible increased (hyperaes-
thetic) response cranial to the spinal lesion.

Spinal cord lesions

The presence of a spinal lesion at the level of the

Spinal cord lesions, whether focal or diffuse, are less

reflex arc results in a lack of muscle contraction in common in cattle than sheep but present the veterinarian response to stimulation. Denervation of the effector muscle results in flaccid paralysis with atony (lower from a wide range of aetiologies including vertebral motor neuron disease). A spinal lesion cranial to the body abscess, extradural abscess, trauma, protozoan reflex arc removes the normal controlling inputs from (encephalo-) myelitis and, in those countries where higher centres via the upper motor neurons and results enzootic bovine leucosis virus is prevalent, neoplasia. in exaggerated responses and spastic paralysis (upper Bacteraemia occurring during the neonatal period motor neuron disease).

occasionally results in vertebral body abscess and extradural abscess formation causing clinical signs in two to four month-old calves. The neurological signs are Cervical spinal cord C1–C6 frequently sudden in onset despite the chronic nature The pelvic limbs are more severely affected than the

of the compressive spinal cord lesion.

thoracic limbs. There is a range of muscle weakness pro-

Accurate localization of a focal spinal cord lesion

gressing to complete paralysis. It is important to differ-

is important to enable further investigation such as

entiate weakness from ataxia, which can be achieved by

radiography; surgery is rarely, if ever, undertaken in

pulling sideways on the tail as the animal is walking.

ruminant species. Localization of the lesion(s) relies

Weak animals can easily be pulled to the side, may

upon the assessment of simple spinal reflex arcs which

stumble and fall over. Spinal reflexes are increased

indicate the presence of either upper or lower motor

(upper motor neuron signs to all four limbs) but this

neuron signs in the affected limbs.

aspect of the neurological examination may prove dif-

The simple spinal reflex arc comprises three neurons:

ficult in adult cattle which are not recumbent. Cervical

pain may be evident as rigidity of the neck with resent-

- The sensory neuron (stretch receptor in tendon)*

ment to forced movement of the head. Typically, the

- *The internuncial neuron*

neck is extended and the head held lowered. With

- *The lower motor neuron (contraction of limb*
severe lesions the animal may be unable to maintain
muscle)

sternal recumbency but will make frequent attempts to

While the reflex motor response to sensory stimuli
raise itself from lateral recumbency.

can occur without the input of higher centres, the higher
motor centres exert control of voluntary movement via
the upper motor neurons which synapse on the lower
Cervico-thoracic spinal cord C6–T2

motor neuron. Flexor (withdrawal) reflexes can be
Spinal cord lesions involving the brachial intumescence
determined by pinching the interdigital skin or apply-
may result in equally severe deficits in both the thoracic
ing pressure across the coronary band with resultant
and pelvic limbs. There is ataxia and weakness of all
unconscious flexion and withdrawal of the stimulated
four limbs. Thoracic limb reflexes are reduced (lower
limb. Recognition of pain indicates integrity of the

motor neuron signs) with increased pelvic limb reflexes spinal cord above the reflex arc. To determine tendon (upper motor neuron signs).

‘jerk’ reflexes for the thoracic limb, the triceps tendon is tapped 2–5 cm proximal to its attachment onto the olecranon process. The normal response is extension of Thoraco-lumbar spinal cord T2–L3

the elbow joint. The pelvic limb is gently supported in Animals with a spinal cord lesion caudal to T2, but the mid femoral region and the middle patellar ligament lightly tapped. The normal reflex is extension of upper motor neuron signs affecting the pelvic limbs. the stifle joint.

Affected cattle frequently adopt a dog-sitting posture

Lesions affecting the upper motor neurons result in with normal thoracic limb function (Holmes et al. , 1989) conscious proprioceptive deficits evident as changes in but with the pelvic limbs extended alongside the flight of the foot and abnormal placement of the foot abdomen. The dog-sitting position should immediately

on the ground, resulting in stumbling and knuckling of alert the clinician because ruminants raise themselves

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using their pelvic limbs before the thoracic limbs. The Treatment and prevention

withdrawal and patellar reflexes are increased and Traumatic lesions in calves following dystokia, indi- there are conscious proprioceptive deficits and paresis cated by xanthochromic CSF collection, may improve of the pelvic limbs. The panniculus reflex may be useful within two weeks with good management and dedi- when attempting to localize a thoracolumbar spinal cated care with frequent turning of the calf. Treatment lesion.

of extradural and vertebral body abscesses is hopeless (Scott et al. , 1991) and affected calves should be euthanased for welfare reasons.

Lumbo-sacral spinal cord L4–S2

Prevention of bacteraemia in neonatal calves dictates

A lesion involving the sacral outflow results in lower high hygiene standards in the calving accomodation and

motor neuron signs of the pelvic limbs with superficial ensuring timely ingestion of adequate levels of good sensation loss,

paresis and reduced or absent

quality colostrum by the calf within the first six hours reflexes.

of life. Repeated dipping of the umbilical remnant in strong veterinary iodine BP during the first 12 hours will prevent omphalophlebitis. Control of salmonellosis

Sacrococcygeal spinal cord: cauda equina syndrome in calves from endemically-infected herds can be

The cauda equina syndrome results from lesions involving attempted by prior vaccination of the dam and feeding the sacrococcygeal spinal cord and results in hypostored colostrum during the first two weeks of life.

tonia, hypalgesia and reduced reflexes of the tail, anus and perineal region, bladder atony and dilation of the rectum.

Once a lesion has been localized to a specific section

References and further reading

of the spinal cord a number of ancillary tests can be per-

formed, including radiography and myelography, to

Aldridge, B.M., Scott, P.R., Clarke, M., Will, R. & McInnes, A.

define the lesion precisely. Before such examinations

(1988) Bovine spongiform encephalopathy: clinical signs

are undertaken, which are expensive and may require

and extended neurological investigation. In Proceedings of XV World Buiatrics Congress, Palma da Majorca, October general anaesthesia to perform, considerable useful

1988, pp. 1531–4.

information can be obtained relatively easily and

Ames, T.R. (1987) Neurologic disease caused by Haemophilus inexpensively following the collection and analysis of

somnus. Veterinary Clinics of North America, 3, 61–73.

lumbar CSF.

Baird, J.D., Johnston, K.G. & Hartley, W.J. (1974) Spastic paresis in Friesian cattle. Australian Veterinary Journal, 50, 239–49.

Lumbar CSF collection and interpretation

Barlow, R.M. (1980) Genetic cerebellar disorders in cattle. In A focal inflammatory lesion within the vertebral

Animal Models of Neurological Disease (ed. by R. Clifford column involving the leptomeninges results in leakage

& P. O'Behan), pp. 294–305. Pitman Medical Series,

of protein and some inflammatory cells into the CSF. If

Tunbridge Wells.

the lesion also causes spinal cord compression this will

Barlow, R.M. (1991) In Diseases of Sheep (ed. by W.B. Martin

& I.D. Aitken), 2nd edn. Blackwell Scientific Publications, arrest the cranial flow of CSF and prevent equilibria-London.

tion of CSF protein concentration within the lateral

Berry, P.H., Howell, J. McC., Cook, R.D., Richards, R.B. & ventricles. Such compression results in accumulation of

Peet, R.L. (1980) Central nervous system changes in

protein within the CSF caudal to the lesion (Scott et al. , sheep and cattle affected with natural or experimental

1991). In practical terms, where there is a compressive

annual ryegrass toxicity. Australian Veterinary Journal, 56, spinal lesion cranial to L5, collection of lumbar CSF will

402–403.

yield a sample with a marked increase in protein con-

Braund, K.G. (1985) Localizing lesions using neurologic syn-

centration relative to the cisternal sample which is

dromes – 1: brain syndromes. Veterinary Medicine, 80, 40–54.

usually normal (Scott & Will, 1991; Scott, 1992).

Brayton, C.F. (1986) Dimethyl sulfoxide (DMSO): a review.

Typical aetiologies include extradural and vertebral

Cornell Veterinarian, 76, 61–90.

body abscess formation in young calves from which

Buxton, D., Macleod, N.S.M. & Nicolson, T.B. (1981) Focal

symmetrical encephalomalacia in young cattle. *Veterinary*

Salmonella dublin and *Salmonella typhimurium* are frequently isolated, indicating previous bacteraemia. *The*

Salmonella dublin and *Salmonella typhimurium* are frequently isolated, indicating previous bacteraemia. *The*

Cherubin, C.E. & Eng, R.H.K. (1986) Experience with the use isolation of *Arcanobacterium pyogenes* and *Staphylo-of cefotaxime* in the treatment of bacterial meningitis.

coccus spp. from ovine vertebral body abscesses

American of Journal of Medicine, **80**, 398–404.

(Finley, 1975) has led to the treatment of such lesions

Christian, R.G. & Tryphonas, L. (1971) Lead poisoning in

with high doses rates of penicillin, but with no success

cattle; brain lesions and haematologic changes. *American*

(Scott et al., 1991).

Journal of Veterinary Research, **32**, 203–16.

914 • Chapter 51

Crick, J. & Brown, F. (1976) Rabies vaccines for animals and Haig, D.A. (1955) Tick-borne rickettsioses in South Africa.

man. *Veterinary Record*, **99**, 162–7.

Advances in Veterinary Science, **2**, 307–25.

Cutlip, R.C., Miller, J.M., Race, R.E., Jenny, A.L. & Katz, J.B.

Hansen, K.M., Krogh, H.V., Moller, J.E. & Elleby, F. (1988) (1994) Experimental inoculation of cattle with US sources

The recumbent calf syndrome in the Red Danish milk breed

of scrapie. Journal of the American Veterinary Medical Association—a new hereditary disease. Dansk Veterinærtidsskrift, 71, citation, 204, 72.

128–32.

Dawson, M., Wells, G.A.H. & Parker, B.N.J. (1990) Preliminary Harper, P.A.W., Healy, P.J. & Dennis, J.A. (1986) Inherited evidence of the experimental transmissibility of bovine

congenital myoclonus of polled Hereford calves (so-called

spongiform encephalopathy to cattle. Veterinary Record, neuraxial oedema): a clinical, pathological and biochemical

126, 112

study. Veterinary Record, 119, 59–62.

Del Rio, M.A., Chrane, D. & Shelton, S. (1983) Ceftriaxone Healy, P.J., Harper, P.A.W. & Dennis, J.A. (1986) Diagnosis versus ampicillin and chloramphenicol for treatment of

of neuraxial oedema. Australian Veterinary Journal, 63, 95.

bacterial meningitis in children. Lancet, 1, 1241–4.

High, J.W., Kincaid, C.M. & Smith, J.H. (1958) Doddler calves: Doherty, P.C. & Reid, H.W. (1971) Louping ill encephalitis in an inherited disorder of Hereford cattle. Journal of Heredity—the sheep. 11. Distribution of virus and lesions in nervous

ity, 49, 250–2.

tissue. Journal of Comparative Pathology, 81, 531–6.

Hocking, J.D., Jolly, R.D. & Bait, R.D. (1972) Deficiency of Donnelly, W.J.C., Sheahan, B.J. & Rogers, T.A. (1973) GM,

mannosidase in Angus cattle: an inherited lysosomal storage

gangliosidosis in Friesian calves. *Journal of Pathology and disease. Biochemical Journal*, **128**, 69–75.

Bacteriology, **111**, 173–9.

Holmes, L.A., Scott, P.R. & Aldridge, B.M. (1989) Thymic lymphoma in calves. *Journal of Pathology*, **157**, 173–9.
(1970) Plasma enzyme and metabolite concentrations in cerebrocortical necrosis. *Veterinary Record*, **87**, and pelvic limb paresis in a heifer. *British Veterinary*

Journal, **146**, 91–2.

Engel, D. (1970) Populationsgenetische Untersuchungen zur Hüllkrankheit der Rinder. Dissertation, Giessen.
(1957) Cerebellar ataxia in calves. *Canadian Journal of Comparative Medicine*, **21**, 72–6.

in Kurhessen. Dissertation, Giessen.

Humphreys, D.J. (1988) *Veterinary Toxicology*, 3rd edn, p. 8.

Feldstein, T.J., Uden, D.L. & Larson, T.L. (1987) Cefotaxime for Baillière Tindall, London.
treatment of Gram-negative bacterial meningitis in infants

Inaba, Y., Kurogi, H. & Omori, T. (1975) Akabane disease: and children. *Pediatrics Infectious Diseases Journal*, **6**, 471–5.

epizootic abortion, premature birth, stillbirth, and congenital

Field, A.C. & Suttle, N.F. (1979) Effect of high potassium and tal arthrogryposis-hydranencephaly in cattle, sheep and

low magnesium intakes on the mineral metabolism of

goats caused by Akabane virus. *Australian Veterinary*

monozygotic twin cows. Journal of Comparative Pathology, Journal, 51, 584–5.

89, 431–9.

Innes, J.R.M., Russell, D.S. & Wilsden, A.J. (1940) Familial Finley, G.G. (1975) A survey of vertebral abscesses in domes-cerebellar hypoplasia and degeneration in Hereford calves.

tic animals in Ontario. Canadian Veterinary Journal, 16, Journal of Pathology and Bacteriology, 50, 455–61.

114–17.

Isler, C.M., Bellamy, J.E.C. & Wobeser, G.A. (1987) A neuro-Fletcher, L.R. & Harvey, I.C. (1981) An association of a

toxin in serum of calves with ‘nervous’ coccidiosis. Canadian Lolium endophyte with ryegrass staggers. New Zealand Journal of Veterinary Research, 51, 253–60.

Veterinary Journal, 29, 185–6.

Jamison, J.M. & Prescott, J.F. (1987) Bacterial meningitis in Foster, J.D., Hope, J. & Fraser, H. (1993) Transmission of large animals. 1. Compendium of Continuing Education, 9, bovine spongiform encephalopathy to sheep and goats. 399–406.

Veterinary Record, 133, 339.

Jeffrey, M. (1992) A neuropathological survey of brains

Gallagher, R.T., White, E.P. & Mortimer, P.H. (1981) Ryegrass submitted under the Bovine Spongiform Encephalopathy

staggers: isolation of potent neurotoxins lolitrem A and

Orders in Scotland. Veterinary Record, 131, 332–6.

lolitrem B from staggers producing pastures. New Zealand

Jeffrey, M. & Wilesmith, J.W. (1992) Idiopathic brainstem neu-Veterinary Journal, **29**, 189–90.

ronal chromatolysis and hippocampal sclerosis: a novel

Geerken, C.M. & Figueroa, V. (1971) Cerebrocortical necro-

encephalopathy in clinically suspect cases of BSE. Veteri-

sis (molasses toxicity) in beef cattle: some preliminary

nary Record, 131, 359–63

biochemical parameters. Revista Cubana de Cienca Agricala

Jeffrey, M. & Wilesmith, J.W. (1996) Idiopathic brainstem

(English edn), 5, 205.

neuronal chromatolysis of cattle: a disorder with clinical

Green, S.L. & Smith, L.L. (1992) Meningitis in neonatal

similarity to BSE. Veterinary Record, 139, 398.

calves: 32 cases (1983–1990). Journal of the American

Jubb, K.V.F. & Huxtable, C.R. (1993) In Pathology of Domes-Veterinary Medical Association, **201**, 125–8.

tic Animals (ed. by K.V.F. Jubb, P.C. Palmer, & C.R.

Grundlach, A.L., Dodd, P.R., Grabara, C.S.G., Watson, W.E.J.,

Huxtable), 4th edn, Vol. 1, p. 385. Academic Press, San Diego.

Johnston, G.A.R., Harper, P.A.W., Dennis, J.A. & Healy, P.J.

Kimberlin, R.H. (1982) Scrapie agent: prions or virinos.

(1988) Deficit of spinal cord glycine/strychnine receptor in

Nature, **297**, 107–108.

inherited myoclonus of Poll Hereford calves. *Science*, **241**, deLahunta, A. (1983) *Veterinary Neuroanatomy and Clinical* 1807–10.

Neurology, 2nd edn. W.B. Saunders, Philadelphia.

Hadlow, W.J., Kennedy, R.C. & Race, R.E. (1982) *Natural*

Lambert H.P. & Wall R.A. (1991) Meningococcal meningitis:

infection of Suffolk sheep with scrapie virus. *Journal of*
treatment. In Infections of the Central Nervous System (ed.

Infectious Diseases, **146**, 657–64.

by H.P. Lambert), p. 92. Edward Arnold, London.

Neurological Disorders • 915

Landesman, S.H., Corrado, M.L. & Shah, P.M. (1981) Past and phologic
alterations of the blood–brain barrier with exper-current roles for cephalosporin
antibiotics in treatment of

infectious meningitis in the rat. Temporal sequence and role

meningitis. Emphasis on use in Gram-negative bacillary

of encapsulation. *Journal of Clinical Investigation*, **77**, 1084–

meningitis. *American Journal of Medicine*, **71**, 693–703.

95.

Little, P.B. (1978) Identity of fluorescence in polioencephalo-Read, W.K. &
Bridges, C.H. (1969) Neuronal lipodystrophy

malacia. *Veterinary Record*, **103**, 76.

occurrence in an inbred strain of cattle. *Veterinary Pathology*, J.C., Scott, P.R., Howie, F., Lewis, M., FitzSimons, J. & ogy, **6**, 235–43.

Spence, J.A. (1996) Sulphur-induced polioencephalomalacia

Rebhun, W.C. & deLaHunta, A. (1982) Diagnosis and treatment

in lambs. *Veterinary Record*, **138**, 327–9.

ment of bovine listeriosis. *Journal of the American Veterinary Medical Association*, R.M. & Dew, C. (1973) An electron microscopic

study of Aujeszky's disease. *Acta Neuropathologica*, **25**, Reid, H.W., Buxton, D., Pew, I. & Finlayson, J. (1984) Trans-207–19.

mission of louping ill virus in goat milk. *Veterinary Record*, McGuirk, S.M. (1987) Polioencephalomalacia. In *Bovine Neurological Disease* (ed. by J.C. Baker). The Veterinary Clinics of North America, **3**, 107–18.

Edwards, J.R., Cook, R.D. & White, R. (1977) of North America, **3**, 107–18.

Bovine generalised glycogenosis (type II). *Neuropathology*

Marsh, R.F. & Hartsough, G.R. (1985) Is there a scrapie-like and Applied

Neurobiology, **3**, 45–56.

disease in cattle? In *Proceedings of the 7th Annual Western Rings*, D.M. (1987) Bacterial meningitis and diseases caused by Conference Food Animal Veterinary Research, University of bacterial toxins. In *Bovine Neurological Disease* (ed. by J.C.

Arizona, p. 20.

Baker), *The Veterinary Clinics of North America*, **3**, 85–98.

Menges, R.W., Harshfield, G.S. & Wenner, H.A. (1953a)

Robinson, M.M., Hadlow, W.J., Huff, T.P. et al. (1994) Sporadic bovine encephalomyelitis. I. The natural history

Experimental infection of mink with bovine spongiform

of the disease. American Journal of Hygiene, 57, 1–14.

encephalopathy. Journal of General Virology, 75, 2151–5.

Menges, R.W., Harshfield, G.S. & Wenner, H.A. (1953b)

Sande M.A. (1981) Antibiotic therapy of bacterial meningitis:

Sporadic bovine encephalomyelitis. II. Studies on the

*lessons we've learned. American Journal of Medicine, 71, pathogenesis and
etiology of the disease. Journal of the*

507–10.

American Veterinary Medical Association, 122, 249–94.

*Sarasola, P. & McKellar, Q.A. (1992) Effect of probenecid on Merz, P.A.,
Somerville, R.A., Wisniewski, H.M. & Iqbal, K.*

disposition kinetics of ampicillin in horses. Veterinary

(1981) Abnormal fibrils from scrapie infected brain. Acta

Record, 131, 173–5.

Neuropathologica, 54, 63–74.

*Saunders, L.Z., Sweet, J.D., Martin, S.M., Fox, F.H. & Fincher, Moellering, R.C.
& Fisher, E.G. (1972) Relationship of intraM.G. (1952) Hereditary congenital
ataxia in Jersey calves.*

*ventricular gentamicin levels to cure of meningitis. Journal Cornell
Veterinarian, 42, 559–91.*

of Pediatrics, 81, 534.

Scheld, W.M. & Brodeur, J.P. (1983) Effect of methylpred-

Morgan, K.L. (1988) Bovine spongiform encephalopathy: time
nisolone on entry of ampicillin and gentamicin into
to take scrapie seriously. *Veterinary Record*, **122**, 445–6.

cerebrospinal fluid in experimental pneumococcal and

Morgan, K.L. (1992) Cerebellar ataxia and head tremor in an

Escherichia coli meningitis. *Antimicrobial Agents and Alpaca. Veterinary
Record*, **131**, 216–17.

Chemotherapy, **23**, 108–12.

Morgan, K.T. (1973) An ultrastructural study of ovine polioen-

Scott, P.R. (1992) Cerebrospinal fluid collection and analysis cephalomalacia.
Journal of Pathology, **110**, 123–30.

in some common ovine neurological conditions. *British*

Oldendorf, W.H. (1975) Permeability of the blood–brain

Veterinary Journal, **148**, 15–22.

barrier. In *The Nervous System* (ed. by D.B. Tower), Vol. 1, Scott, P.R. (1993a) A
field study of ovine listerial meningo-pp. 279–89. Raven Press, New York.

encephalitis with particular reference to cerebrospinal fluid

O'Toole, D. & Jeffrey, M. (1987) Congenital sporozoan

analysis as an aid to diagnosis and prognosis. *British Veteri-encephalomyelitis
in a calf. Veterinary Record*, **121**, 563–6.

nary Journal, **149**, 165–70.

Padgett, G.A. (1968) The Chediak–Higashi syndrome.

Scott, P.R. (1993b) Cerebrospinal fluid collection in ruminant *Advances in*

Veterinary Science, **12**, 239–84.

neurological disease. *In Practice*, **15**, 298–300.

Palmer, A.C., Blakemore, W.F., Barlow, R.M., Fraser, J.A. & Scott, P.R. (1993c) Total protein and electrophoretic pattern

Ogden, A.C. (1972) Progressive ataxia of Charolais cattle

of cerebrospinal fluid from sheep with some common ovine

associated with a myelin disorder. *Veterinary Record*, **91**, neurological disorders. *The Cornell Veterinarian*, **83**, 592–4.

199–204.

Pienaar, J.G. (1970) Electron microscopy of Cowdria (Rick-

Scott, P.R. (1994) Practical application of cerebrospinal fluid ettsia) ruminantium (Cowdry 1926) in the endothelial cells analysis in the differential diagnosis of spinal cord lesions

of the vertebrate host. *Onderstepoort Journal of Veterinary in ruminants. In Practice*, **16**, 301–303.

Research, **37**, 67–78.

Scott, P.R. (1995a) The collection and analysis of cerebrospinal Power, E.P., Crowley, J.J., Byrne, J.F., O’Keefe, M.P. & Weavers, fluid as an aid to diagnosis in ruminant neurological disease.

E.D. (1985) Cerebrocortical necrosis in dairy cows. *Irish*

British Veterinary Journal, **151**, 603–14.

Veterinary Journal, **39**, 81–3.

Scott, P.R. (1995b) Differential diagnosis of recumbency in

Prescott J.F. & Baggot, J.D. (1988) Antimicrobial Therapy in neonatal calves. *In Practice*, **17**, 162–5.

Veterinary Medicine, p. 71. Blackwell Scientific Publications, Scott, P.R. (1996)
Indications for lumbosacral cerebrospinal

Boston, Massachusetts.

*fluid sampling in ruminant species in field situations. Agri-Quagliarello, V.J.,
Long, W.J. & Scheld, W.M. (1986) Mor-*

Practice, **17**, 30–4.

916 • Chapter 51

Scott, P.R., Aldridge, B.M., Clarke, M., Will, R. & McInnes, A.

Central Nervous System (ed. by H.P. Lambert), p. 40. B.C.

(1988b) *Bovine spongiform encephalopathy: electroen-*

Decker, Philadelphia.

cephalographic studies. In Proceedings of the XV World

Stuart, L.D. & Leipold, H.W. (1985) Lesions in bovine

Buiatrics Congress, Palma da Majorca, October 1988,

progressive degenerative myeloencephalopathy. Veterinary

p. 1530.

Pathology, **22**, 13–23.

Scott, P.R., Aldridge, B.M., Clarke, M. & Will, R.G. (1989b) Sullivan, N.D.

(1985) *Cytopathology of the nervous system. In*

Cerebrospinal fluid studies in normal cows and cases of

Pathology of Domestic Animals, 3rd edn (ed. by K.V.F. Jubb, *Bovine Spongiform
Encephalopathy. British Veterinary*

P.C. Kennedy & N. Paimer), Vol. 1, p. 225. Academic Press, Journal, **146**, 88–

90.

New York.

Scott, P.R., Aldridge, B.M., Clarke, M. & Will, R.G. (1989a) Tauber, M.G., Shibl, A.M., Hackbarth, C.J., Larrick, J.W. & Bovine spongiform encephalopathy in a British Friesian

Sande, M.A. (1987) Antibiotic therapy, endotoxin concentration in cerebrospinal fluid and brain edema in experimental Escherichia coli meningitis in rabbits. *Journal of the American Veterinary Medical Association*

195, 1745–7.

Scott, P.R., Aldridge, B.M., Holmes, L.A., Milne, E.M. &

Infectious Diseases, **156**, 456–62.

Collins, D.M. (1988a) Bovine spongiform encephalopathy in

Trautwein, G., Hewicker, M., Liess, B., Orban, S. & Peters, W.

an adult British Friesian cow. *Veterinary Record*, **123**, 373–4.

(1987) Cerebellar hypoplasia and hydranencephaly in cattle

Scott, P.R., Henshaw, C.J. & Watt, N.J. (1994b) Cerebellar associated with transplacental BVD virus infection. In

abiotrophy in a pedigree Charolais ram lamb. *Veterinary*

Pestivirus Infections of Ruminants (ed. by J.W. Harkness), *Record*, **135**, 42–3.

pp. 169–78. Commission of European Communities,

Scott, P.R., Murray, L.M. & Penny, C.D. (1991) A field study Luxembourg.

of eight ovine vertebral body abscess cases. New Zealand

Tuazon, C.U., Shamsuddin, D. & Miller, H. (1982) *Antibiotic Veterinary Journal*, **39**, 105–107.

susceptibility and synergy of clinical isolates of Listeria Scott, P.R. & Penny C.D. (1993) *A field study of meningo-monocytogenes. Antimicrobial Agents and Chemotherapy, encephalitis in calves with particular reference to cere-21*, 525–7.

brospinal fluid analysis. Veterinary Record, **133**, 119–21.

Tunkel, A.R., Wispelway, B. & Scheld, W.M. (1990) *Bacterial* Scott, P.R., Penny, C.D. & Sargison, N.D. (1995) *Bovine*

meningitis: recent advances in pathophysiology and treat-

spongiform encephalopathy in a Holstein cow born in the

ment. Annals of Internal Medicine, **112**, 610–23.

United Kingdom during September 1989. Canadian Veteri-

Tyrrell, D.A.J. (1979) Aspects of slow and persistent virus

nary Journal, **36**, 310–11.

infections. In New Perspectives in Clinical Microbiology, Scott, P.R., Pyrah, I., Penny, C.D. & Clarke, C.J. (1993b) *Post Vol. 2*, p. 286. Martinus Nijhoff, The Hague.

septicaemia focal meningitis causing cerebellar dysfunction

Wells, G.A.H., Scott, A.C., Johnson, C.T., Gunning, R.F.,

in an adult ram. Veterinary Record, **133**, 623–4.

Hancock, R.D., Jeffrey, M., Dawson, M. & Bradley, R.

Scott, P.R., Sargison, N.D., Penny, C.D. & Pirie, R.S. (1994a) *A (1987) A novel progressive spongiform encephalopathy in*

field study of naturally-occurring ovine bacterial meningo-

cattle. *Veterinary Record*, **121**, 419–20.

encephalitis. *Veterinary Record*, **135**, 154–6.

White, M., Whitlock, R.H. & deLahunta, A. (1975) A

Scott, P.R. & Sutton, D. (1992) A study of some factors which cerebellar
abiotrophy of calves. *Cornell Veterinarian*, **65**, may influence the concentrations
of penicillin G in cere-476–91.

brospinal fluid in some common ovine neurological dis-

Wilesmith, J.W. & Ryan, J.B.M (1993) Bovine spongiform

eases. In *Proceedings of the XVII World Buiatrics Congress, encephalopathy:*
observation on the incidence during 1992.

St Paul, USA, Vol. 2, pp. 63–8.

Veterinary Record, **132**, 300–301.

Scott, P.R. & Will, R.G. (1991) Froin's syndrome in five cases Wilesmith, J.W.,
Wells, G.A.H., Cranwell, M.P. & Ryan, J.B.M.

of ovine epidural abscess. *British Veterinary Journal*, **147**, (1988) Bovine
spongiform encephalopathy: epidemiological

582–4.

studies. *Veterinary Record*, **123**, 638–44.

Scott, P.R., Woodman, M.P., Watt, N.J., McGorum, B.M. &

Wilesmith, J.W., Wells, G.A.H., Ryan, J.B.M. et al. (1997) A McDiarmid, A.
(1993a) Protozoan encephalo-myelitis as a

cohort study to examine maternally-associated risk factors

cause of pelvic limb paresis in a Blackface yearling sheep.

for bovine spongiform encephalopathy. *Veterinary Record*, New Zealand

Veterinary Journal, **41**, 139–41.

141, 239–43.

Stolzenberg, V. & Schonmuth, G. (1971) *Experimentelle*

Winter, M.H., Aldridge, B.M., Scott, P.R. & Clarke, M. (1989) *Untersuchungen
über die ökonomischen Auswirkungen*

Occurrence of 14 cases of bovine spongiform encephalopa-

und die Vererbung der spastischen Parese der Hinterglied-

thy in a closed dairy herd. British Veterinary Journal, **145**, massen. *Wissenschaft
zur Humboldt Universität, Berlin*, **20**, 191–4.

353–70.

Woodman, M.P., Scott, P.R., Watt, N.J., McGorum, B.M. &

Stone, J.W. & Wise, R. (1991) *Penetration of antimicrobial Penny, C.D. (1993)
Selective cerebellar degeneration in a*

*agents into the central nervous system. In Infections of the Limousin cross heifer.
Veterinary Record*, **132**, 586.

Chapter 52

Ocular Diseases

P.G.C. Bedford

Introduction 917

affected as adults and failure to produce effective

Anomalies of the orbit and globe

917

vaccines means that this disease will continue to inflict

Anophthalmia 917

severe economic loss for as long as young cattle are

Microphthalmia 918

managed intensively. Disease of epidemic proportions

Multiocular defects

918

occurs all the year round and, while the aetiological

Cyclopia 918

controversy may continue, the term IBKC generally

Strabismus 918

embraces all keratoconjunctivitis of infectious nature in

Nystagmus 918

Orbital neoplasia

918

this species. Similarly, ocular squamous cell carcinoma

Anomalies of the eyelids

918

of probable heritable nature in breeds with reduced

Congenital/paranatal defects

918

palpebral pigmentation continues to account for a high

Trauma 918

carcase condemnation rate and runs second only to

Congenital porphyria

919

lymphosarcoma in terms of condemnation for neoplas-

Diseases of the conjunctiva and cornea

919

tic reasons. At the other end of the scale there are many

Epibulbar dermoid

919

congenital, inherited and acquired defects that tend to

Infectious bovine keratoconjunctivitis (IBKC)

919

escape diagnosis on the basis of there being no unto-

Infectious bovine rhinotracheitis

921

ward effect on function, no associated discomfort or

Endothelial dystrophy

922

pain and no necessity to treat. Were cattle subjected to

Neoplasia 922

the same degree of scrutiny as the dog, then it is likely

Diseases of the uveal tract

923

Congenital anomalies

923

that the literature would indicate a similar incidence of

Uveitis 923

disease. In one study of 500 cattle of all ages, almost 20

Neoplasia 923

per cent overall demonstrated ocular anomalies of one

Disease of the lens

923

sort or another, the incidence ranging from 3 per cent

Diseases of the retina and optic nerve

924

in young cattle to in excess of 70 per cent in the older

Congenital defects

924

individuals (Amman, 1968). Examples of this kind of

Inflammation 924

survey work in cattle are few and far between, but

Hypovitaminosis A

925

general interest in ophthalmology as a refined discipline

Male fern optic neuropathy

925

in today's veterinary scenario will probably stimulate

Arthrogryposis 925

further ophthalmic studies in animals that are primarily

Progressive retinal degeneration

925

produced for food purposes in our society.

Glaucoma 925

Introduction

Anomalies of the orbit and globe

Ophthalmic disorders in cattle are more common than

Congenital and acquired defects of these structures

is generally believed, but it is only those that are of con-

both occur, some congenital anomalies being inherited

siderable economic importance that generally receive

while others are probably environmental in origin (see

much attention. Infectious bovine keratoconjunctivitis

also p. 180). The acquired defects are due to trauma, (IBKC), variously known throughout the world as New infection and neoplasia.

Forest disease, pinkeye, contagious ophthalmia and blight, is undoubtedly the commonest and most impor-

Anophthalmia

tant ocular disease that occurs in this species. In the USA alone it is estimated that IBKC is responsible for Absence of the optic vesicle means that an eye cannot an annual loss in excess of £25 million (Punch & Slatter, develop. Histologically, primordial ectodermal and 1984). Approximately three times as many calves are mesodermal tissues cannot be identified. The condition

917

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is rare and is often confused with the type of microphthalmia in which there has been some early differentiation of the optic vesicle but no subsequent maturation. as an occasional feature early in the course of BSE.

Microphthalmia

Orbital neoplasia

Variable degrees of development can be seen, ranging from minimal differentiation of ocular tissue to the development of a small but otherwise normal eye. Retrobulbar and periorbital lymphosarcoma can result in exophthalmos and possible squint. The lesions may

development of a small but otherwise normal eye. be seen unilaterally or bilaterally. Squamous cell carcinoma of the membrana nictitans may invade the orbital and persistent pupillary membrane (PPM), aniridia, tissues to produce a similar clinical picture.

cataract and neuroretinal fold and rosette formation may be seen in association with microphthalmos. Like anophthalmia, the condition is rare in cattle, but inher-

Anomalies of the eyelids

itance has been postulated (Gilmore, 1957) and it has been described in calves with vertebral column defor-

Congenital/paranatal defects

mation (Leipold & Huston, 1968).

Unlike other species, inherited and non-inherited con-

genital palpebral defects are few and far between in

Multiocular defects

cattle. Eyelid colobomata and agenesis are of rare

Bilateral congenital blindness due to multiocular defects

incidence. Although primary conditions in sheep, both

not involving microphthalmos has been recorded in

entropion and ectropion occur most commonly as

Jersey calves (Saunders & Fincher, 1951). The defects

secondary defects in cattle and are associated with

are mainly related to the lens, with microphthalmia,

microphthalmia, blepharitis, keratoconjunctivitis and

ectopia lentis and cataract being seen in association with

trauma. Correction of the cause alleviates the lid dis-

iridaemia. A recessive mode of inheritance has been

tortion except where there is palpebral or orbicularis

suggested, but subsequent literature contains no further

oculi damage and where cicatrization has occurred.

references to this condition in this breed.

Congenital supernumerary openings of the proximal

part of the nasolacrimal duct and the canaliculi at the

medial canthus have been described in calves, heredi-

Cyclopia

tary predisposition or intra-uterine dacryocystitis being

*The development of one eye in calves is extremely
the suggested possible aetiology.*

*rare (Plate 52.1), but the condition has been induced
teratogenically in sheep.*

Trauma

Laceration of the eyelid occurs infrequently, but when

Strabismus (see p. 181)

the palpebral fissure is involved repair is essential. Such

Bilateral convergent squint (esotropia) has been

wounds left to granulate or inadequately sutured may

described as a recessively inherited defect in Jersey

result in distortion of the margo-intermarginalis,

cattle, and it is probably inherited in Shorthorn and

and secondary entropion, secondary ectropion and an

Friesian cattle too (Willoughby, 1968; Bedford, pers.

incomplete blink can result in conjunctival or corneal

obs.). A degree of exophthalmos may or may not be

disease. Delayed closure is complicated by the problem

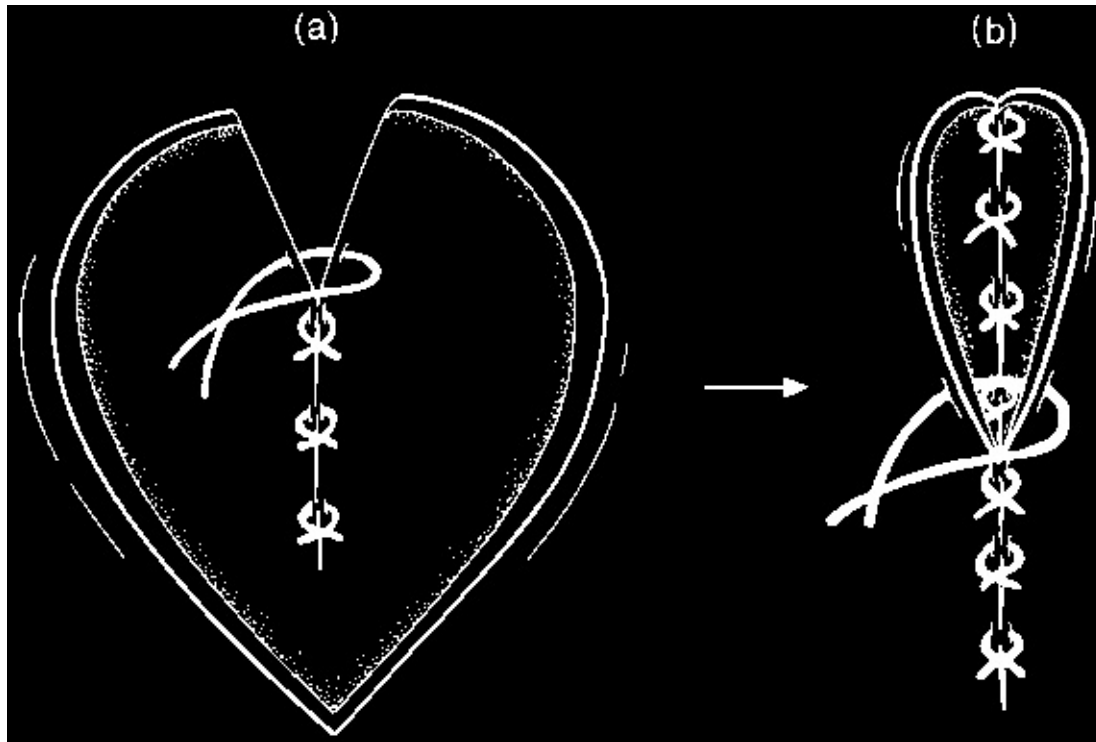
additionally present. The condition is usually noticed at

of wound contraction. Repair is easily effected using four to eight weeks of age (Plate 52.2a) and the degree manual restraint and the same seventh nerve block as of convergence increases until the sixth or seventh that used in disbudding and dehorning can be employed month when only sclera presents within the palpebral should blepharospasm be a problem. Such wounds may fissure (Plate 52.2b). Impaired vision gives way to total require debridement, but the removal of tissue should blindness, and attempts at corrective surgery may be ill be avoided whenever possible. The sutures are placed perceived in the presence of possible optic nerve, at two levels (Fig. 52.1). Tarsal plate and subconjunctival tissue are first repaired without penetrating the palpebral conjunctiva, and the knots are buried in the substance of the eyelid such that no suture material

Nystagmus

can cause corneal irritation. The margo-intermarginalis Nystagmoid movement of the globe is most commonly must be accurately reformed, the first suture ensuring

associated with congenital or paranatal blindness. It is precise apposition at this level. The second row of



Ocular Diseases • 919

and conjunctival inflammation or they physically interfere with effective blinking and lid closure. Removal is then advocated, and topical and regional anaesthesia can be complemented by using the auriculopalpebral nerve block. The lesion must be removed in entirety to prevent recurrence. While a suprascleral lesion is easily excised, care is required with the superficial keratectomy needed to remove the corneal dermoid.

Infectious bovine keratoconjunctivitis

(IBKC, New Forest eye, pinkeye)

Fig. 52.1

Repair of an eyelid wound by two-layer closure. (a)

Aetiology

Tarsal plate and subconjunctival tissue apposed; (b) subcutis

This disease has been recognized for at least 100 years and skin apposed.

(Billings, 1889) since when it has demonstrated both common incidence and worldwide distribution. It is highly contagious, and outbreaks of epidemic proportions involves the subcutis and skin, and single interrupted sutures are used to ensure accurate repair and autumn months, and housed cattle the whole year reduce the chance of wound breakdown. Absorbable round. Young animals are more severely affected, material is used for the buried sutures and nylon is dictating the development of possible local immunity preferable for the cutaneous repair.

to the disease in those previously exposed. Over the

years IBKC has been attributed to several organisms

Congenital porphyria

(Bedford, 1976), but currently *Moraxella bovis* is still regarded by most authorities to be the cause. The root

Congenital porphyria is a rare, recessively inherited defect of haemoglobin metabolism which results in the production of abnormal porphyrins. Clinical signs associated with photosensitization are seen in light-skinned cattle upon exposure to sunlight. Adenexal inflammation, infectious bovine rhinotracheitis (IBR) virus, and blepharospasm and excessive lacrimation.

Mycoplasma bovirhinitis and *M. laidlawii* have been isolated with *Moraxella bovis* from IBKC patients.

Rather than representing cause, however, these organ-

Diseases of the conjunctiva

isms may act synergistically as enhancing factors.

and cornea

Undoubtedly, other enhancing factors can be at work, and the increased incidence of IBKC during the

The occurrence of IBKC and squamous cell carcinoma

warmer months has indicated that ultraviolet light, renders this part of the eye important in economic

flies and dust play potentially important roles in the terms, and as the two disease conditions commonly

overall picture. Certainly flies of the Musca and

involve both structures it is convenient to discuss them

Stomoxys species have been incriminated as mechani-

both under the same section.

cal vectors, but the occurrence of epidemic disease

among winter-housed cattle suggest that flies and the

other environmental factors are not essential.

Epibulbar dermoid

Dermoid formation (choriostoma) is due to the poor

Epidemiology

differentiation of the palpebral tissue with the result

that plaques of hair-bearing skin and subcutis may be

Variation within the clinical picture of the disease may

found involving the lateral aspect of the globe (Plate 52.3). Both the cornea and episcleral tissues can be affected, the dermoid replacing the corneal epithelium of the animal to infection and the type of *Moraxella bovis* and bulbar conjunctiva, respectively. Bilateral involvement is present in its conjunctival sac. Older cattle are more resistant to infection, it being calves and cattle of less than two years of age that demonstrate the highest

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morbidity and the most severe disease. Such resistance may be the result of previous exposure, but local factors disease the eye is extremely painful, and a bilateral as well as antibody formation may be involved (Pugh, 1969). It has been demonstrated that two forms of the experiencing impaired vision. Inappetance, suppressed

bacterium exist: a smooth non-haemolytic form that is weight gain and reduced milk production are related to avirulent and can usually be isolated from recovering the severity of involvement. At this stage, and particularly in young animals, loss of epithelium and anterior haemolytic virulent form that causes the acute disease stroma in the central lesion may occur, and this ulcer (Pedersen, 1973). The rough form is fimbriated for may rapidly enlarge to involve deeper stromal tissue. adherence (Sandhu et al., 1974), and repeated passage Less severe forms of the disease resolve within two or has demonstrated its conversion into the smooth form. three weeks, the cornea gradually clearing from its The presence of fimbriae would appear to be necessary periphery towards the centre as the vessels cease to for disease to occur and for immunity to develop. The transmit blood to the healing ulcer site. Recovery takes difficulties experienced in the production of an effective vaccine against Moraxella bovis may be overcome

stromal scarring and keratoconus may result. In some as the result of further structural studies to determine patients descemetocoele formation may complicate deep the possible immunogenic status of the fimbriae. In the ulcers and actual rupture may lead to panophthalmitis past, alteration of the surface components of bacterial (Plate 52.4b). Blindness may result, and the eye may cells in the preparation of vaccines may have been become glaucomatous but will eventually shrink (phthi-wholly responsible for the ineffectiveness of such vac-sis bulbi). The occasional death has been attributed to cines. The economic importance of IBKC demands that meningitis following presumed ascending infection of an effective vaccine be developed, but it could be many the optic nerve.

years before the solution is finally produced.

Treatment

Signs

Moraxella bovis is susceptible to most antimicrobial

Epidemics occur as the result of the introduction of

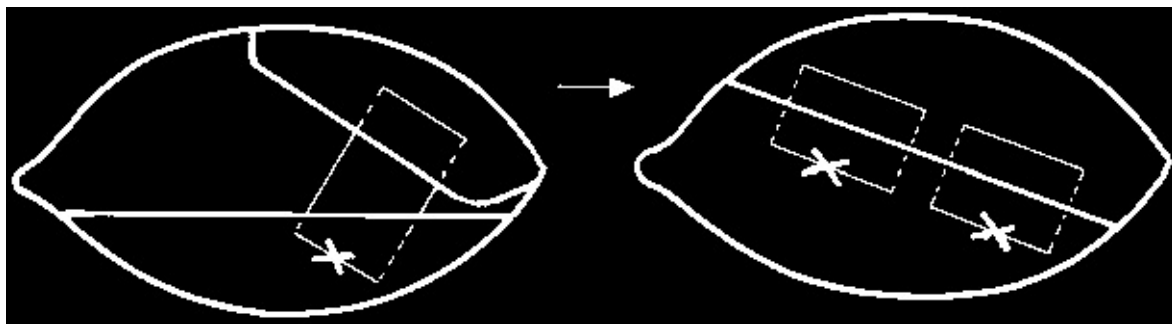
agents including many antiseptics, and this, combined

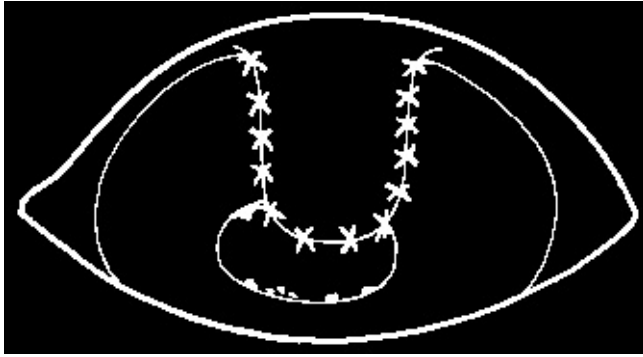
incubating, chronically affected or carrier animals into with uncertainties concerning the aetiology of the condition of the herd. The presenting clinical features may vary considerably and a tendency for spontaneous resolution in some patients, renders the accurate assessment of any individual and the level of enhancing factors present. treatment difficult. It is generally agreed, however, that This variation is not only seen on a herd-to-herd basis, the earlier the treatment the less severe the disease but between individual animals within the same herd. and the greater the chance of controlling the outbreak. Both unilateral and bilateral cases will be seen; in the In the absence of isolation facilities, it is suggested that latter a variation in the degree of severity may be noted treatment of the entire herd will protect the unaffected between the two eyes. Usually, inflammation of the cattle. Consideration of potential carrier status dictates bulbar and possibly the palpebral conjunctivae precedes the use of antibiotics in newly acquired stock before the keratitis, but occasionally the conjunctival involve-

they are introduced into the herd.

ment is not seen until after corneal inflammation has
Antimicrobial drugs may be administered topically,
made its appearance. Blepharospasm, photophobia and
by subconjunctival injection and parenterally. The ideal
copious ocular discharge herald anterior segment pain,
therapy for a herd problem demands effective one-time
the discharge, primarily clear and thin, becoming puru-
dosage and, as such, topically applied preparations,
lent quite rapidly to mat the lashes and circumorbital
which require frequent administration to maintain ther-
hair. The conjunctiva is chemotic and swelling of the
apeutically effective levels in the precorneal tear film
eyelids may occur. Corneal changes usually develop
(PCTF), cannot be very effective. Their short contact
within 48 hours, a 3-mm wide, slightly raised area of
time is reduced further by the presence of ocular
cloudiness normally making its appearance centrally.
discharge and lacrimation, and blepharospasm renders
Epithelial loss can be demonstrated using fluorescein
their application difficult. Currently, increased contact

stain, and the lesion itself may take on the yellowish blue
time as the result of specific formulation is claimed for
of pyogenic necrosis. The surrounding cornea becomes
several antibiotic preparations including cloxacillin,
oedematous and hazy, and a low-grade anterior uveitis
cephalonium and a penicillin and streptomycin combi-
with aqueous flare may be noticeable in some animals.
nation. The subconjunctival injection of antibiotics can
Corneal vascularization from the limbal blood vessels is
be used as an alternative to the topical route, but the
well established by the sixth day, the new vessels rapidly
antibiotic must be placed beneath the dorsal bulbar
progressing towards the central lesion in the anterior
conjunctiva. This is difficult to do for the unpractised





Ocular Diseases • 921

hand and a topical analgesic agent (proxmetacaine hydrochloride) must be used. From this site the antibiotic leaks into the PCTF and thus has an effect. Intrapalpebral injections of various antibiotics are used commonly, but there is evidence to demonstrate that a drug deposited in palpebral muscle gets into the lacrimal gland or the PCTF. Long-acting ampicillin,

Fig. 52.2

The membrana flap. The membrana is sutured into oxytetracycline and penicillin may be used parenterally the loose dorsal bulbar conjunctiva using two or three mattress for repeated good effect. Moraxella bovis is usually sutures.

resistant to lincomycin, tylosin and erythromycin but has variable susceptibility to cloxacillin (George, 1990). In an original study, Pedersen (1973) claimed that intra-

venous sulphadimidine was the treatment of choice for IBKC. He showed that although *Moraxella bovis* is present on the corneal and conjunctival surfaces, it colonizes the lacrimal and tarsal glands, and that any treatment must effectively penetrate these tissues. A single injection of sulphadimidine at dose rate of 100 mg/kg

Fig. 52.3

The bulbar conjunctival pedicle flap used in the repair bodyweight will do this, and the drug will remain in the of deep corneal ulceration.

RCTF at a therapeutically active concentration for 24 hours. It is still surprising that this method of treatment is not utilized to any great extent for it seems to answer both theoretical and practical aspects of therapy.

against *Moraxella bovis*, but some recent results are

However, some strains of *Moraxella bovis* have proved quite optimistic (Lepper, 1993). The research has concentrated on the fimbriae or pili found in the rough the future other delivery systems may be evaluated and form of the organism and recombinant DNA technol-

ocular inserts that allow prolonged drug release would
ogy has been involved. Certainly pilus-antigen vaccines
appear to be of potential value in this respect.

have demonstrated acceptable levels of protection in

Surgery can have a part to play in the treatment of
several studies (Lepper, 1988; Moore & Rutter, 1989)

IBKC. The membrana nictitans can be used to support

and workable protocols are now possible. Calves are

the severely ulcerated cornea or protect a ruptured

vaccinated at approximately one month of age, with

anterior chamber, and this technique is preferred to

a second vaccination three weeks later. There is no

a temporary tarsorrhaphy in which the eyelids are

evidence that colostrum derived antibodies will negate

sutured together. Using local anaesthesia together with

this procedure. Adult cattle should have yearly

an auriculopalpebral nerve block to overcome any

boosters.

blepharospasm, the membrana nictitans and the loose

bulbar conjunctiva can be apposed to cover the cornea

Infectious bovine rhinotracheitis

(Fig. 52.2). Mattress sutures of non-absorbable material (see also p. 286)

are used, and the cornea is left covered for two to three weeks. The sutures should not penetrate the mem-

The role of infectious bovine rhinotracheitis virus in brana's full thickness, otherwise corneal erosion will anterior segment disease is not fully understood. The occur. Should a more resilient method of repair be

virus is thought to enhance the effects of Moraxella required then under a general anaesthetic a pedicle of

bovis in some patients with IBKC, but it is also known bulbar conjunctiva can be sutured directly into the

to cause conjunctivitis in its own right. Respiratory corneal defect (Fig. 52.3). The pedicle is separated from disease may or may not be present, and abortion rates its bulbar conjunctival attachment several weeks later may be high in IBR-affected herds.

and the residual conjunctival tissue is usually involved

The ocular involvement may be unilateral or bilateral in the scar. This technique offers the advantages of eral. Varying degrees of chemosis may be present, and

directly strengthening the cornea and introducing a number of white plaques may be found in both the blood supply to the ulcer site.

bulbar and palpebral conjunctivae. The discharge may vary from serous in early disease to a mucopurulent type later. Anterior uveitis and corneal opacity are Vaccination (see p. 1018)

occasional findings, and this latter should not be confused with the corneal abscess or ulcer that routinely occurs in IBKC. Confirmation of diagnosis is not always

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easy, for it requires virus isolation from swabs taken and the marked geographic variation in incidence. only in the early part of the disease. Seroconversion Squamous cell carcinoma occurs in ocular tissue where from negative to positive or a rising titre for IBR anti- there is little or no pigment, and the bulbar conjunctiva body will provide the final proof. There is no specific at the lateral and medial limbi is more exposed than

treatment for the condition, but antibiotics will help conjunctiva elsewhere on the globe or lining the eyelids. control any bacterial secondary infection. Normally, the The incidence of ocular SCC is greater in those parts of disease takes three to four weeks to complete its course. the world with high levels of ultraviolet light, and at high altitude. The IBR virus and its inclusion bodies may be found in the neoplastic tissue (Taylor & Hanks,

Endothelial dystrophy

1969) and it has been postulated that the virus may play Bilateral neonatal corneal oedema has been reported a part in initiating tumour growth or be involved in the in several breeds including the Friesian (Deas, 1959). transformation of precursor lesions into carcinoma. The oedema is uniformly present throughout the whole Surprisingly, perhaps, the incidence of SCC is greater in stroma, and its persistent nature ensures permanent cattle on higher levels of nutrition, but this may be asso- impairment of sight. Treatment is not possible, but ciated with enhanced age changes induced by such diets short-term clearing of the cornea can be obtained

(Anderson, 1970).

using topically applied glycerol or hypertonic saline.

Several stages of development are described in SCC

Signs of possible precursor or accompanying anterior formation. Conjunctival plaques of hyperplastic epithelium (Plate 52.5a) or hyperkeratosis of palpebral skin and defective endothelial function alone is the

represent the initial lesion. Regression may occur, but

indicated cause. The defect is considered to be inher-

the plaques are generally replaced by papilloma for-

ited as a recessive trait, and affected animals and their

mation, and this in turn is replaced by non-invasive car-

carrier parents should be avoided in any breeding

cinoma. Eventually, most of these carcinomas become

programme.

invasive, and the whole globe can be involved (Plate

52.5b). Invasion of orbital bone can occur, and with

long-standing SCC, metastasis via the lymphatics to

Neoplasia (see p. 1126)

involve the lungs, heart, liver and kidney is possible

Ocular squamous cell carcinoma (SCC) or 'cancer eye' (Cordy, 1978).

is the commonest bovine neoplasm, and its economic Treatment is related to the extent of tumour involve- importance has already been stressed. Although SCC ment, and the possibility of metastasis may dictate a has worldwide distribution, its incidence increases in policy of early slaughter. Excision of the neoplasm, countries where cattle experience long-term exposure removal of the membrana nictitans, enucleation or to intensive sunlight. The Hereford would appear exenteration of the orbit represent possible early treat- the most commonly affected breed, and tumour ment. Alternative techniques of cryosurgery, hyper- formation is rarely seen before five years of age. A pre- thermia, radiation therapy and immunotherapy have all sumed inherited predisposition is postulated, and been tried with some success, again early in the course genetic analysis has suggested that it is a recessive trait of tumour development. Small neoplasms can be (Anderson & Chambers, 1957; Vogt & Anderson,

excised irrespective of site, but recurrence and subse-
1964). However, several contributory facts seem to be
quent extension should be expected. Large lesions of
involved, perhaps one of the most important being the
the membrana nictitans may already have invaded the
role of melanin in palpebral, conjunctival and scleral
orbit. Premalignant lesions and small carcinomas can
tissues. Pigmentation is determined genetically, and
be destroyed using liquid nitrogen sprays, but adequate
undoubtedly the presence of melanin protects tissue
freezing demands the use of thermocouple assessment.
from SCC development. As such ultraviolet light is con-
Limbal-based lesions can be difficult to treat effectively.
sidered to be a significant contributory factor in the
Again neoplastic cells can be selectively destroyed by
development of SCC, its absorption by melanin being
heating tissue to 45°C, and early SCC will respond
the significant protective factor. Much SCC finds origin
to hyperthermic therapy. Various forms of ionizing
in the bulbar conjunctiva at the lateral and medial
radiation therapy have been used successfully. Beta-

limbi, but other areas where pigmentation may be irradiation will destroy small lesions, and radioactive reduced (the palpebral conjunctiva, the membrana nictitans, cobalt or gold implants can be similarly effective and eyelid skin) can also be affected. Other positive if the dose rates can be adequately calculated. Possible contributory factors are IBR virus, dust, flies and Immunotherapy offers potentially effective treatment, high levels of nutrition.

with the parenteral use of modified or whole-cell The indications that ultraviolet light may be an tumour cell suspensions having demonstrated important aetiological factor are the actual tumour sites considerable promise (van Kampen et al., 1973).

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Diseases of the uveal tract

panuveitis may all be seen. Anterior uveitis, choroiditis and retinitis may all be present as a result of vascular (see also p. 180 onwards) necrosis and vasculitis (Plowright, 1968). A recurrent uveitis, referred to as specific ophthalmia (Marolt et al., Both congenital and

acquired disease of the uveal tract

1963) has been described for cattle. It is similar clinically to equine recurrent uveitis (periodic ophthalmia) as much difficulty in treatment as it does in the other with corneal oedema, anterior chamber and intravitreal domestic species.

exudates, and retinal haemorrhage being seen in the acute stage. A viral aetiology is suggested, with IBR and

Congenital anomalies

other respiratory viruses isolated from affected animals.

Listeria monocytogenes has been previously associated

Several congenital anomalies occur in cattle, including with outbreaks of keratoconjunctivitis and uveitis aniridia, colobomata, polycoria, persistence of the in housed cattle in the UK (Morgan, 1977), but more pupillary membrane and anterior uveal cyst formation.

recently this organism has been isolated from housed

Aniridia is seen as part of a multiocular defect in

cattle fed on silage, presenting with anterior uveitis as

Jersey calves, in which bilateral partial absence of the

the only clinical feature (Watson, 1989). It has been iris is accompanied by microphakia or lens luxation considered that the organism in the silage gains entry (Saunders & Fincher, 1951). Vision can be affected to to the eye either directly from conjunctival sac con- the point of blindness, and the defect is inherited as a tamination or from the gingival margins by trigeminal simple recessive trait. Iris colobomata, polycoria and migration. The uveitis presents clinically in the same uveal cysts are of no clinical significance, but persistence way as any other anterior segment inflammation; it is of the pupillary membrane may be associated with lens usually unilateral with marked miosis, thickening and and corneal opacities. The vascular mesoderm in which folding of the iris, keratic precipitate (KP) and the pupil develops should be resorbed by the time of hypopyon formation and vascularization of the limbal parturition, but strands of this tissue may persist to span cornea being the presenting signs (Plate 52.6). Treat- the pupil or become adherent to the anterior lens ment includes the use of systemic antibiotics, but sub-

capsule or the corneal endothelium. Cataract or corneal conjunctival corticosteroids and topical atropine are endothelial and deep stromal opacities mark the areas essential for effective resolution.

of adherence, the size of these opacities varying with the amount of the attaching remnant tissue. Heterochromia

Neoplasia

iris, due to a reduction or absence of melanin, is seen in the colour dilute breeds, and it may occasionally be

Primary uveal tract tumours in cattle are rare, but accompanied by photophobia, nystagmus and typical secondary involvement with squamous cell carcinoma papillary coloboma formation.

occurs not infrequently.

Disease of the lens

Uveitis

Inflammation within the uveal tract occurs in association with systemic disease, corneal ulceration, keratitis, genital multi-ocular defect syndrome in Jersey and trauma and intraocular neoplasia. Anterior uveitis can

Friesian cattle, and cataract probably enjoys higher incidence than the literature dictates. Intraocular examination and neonatal coliform septicaemia; panophthalmitis in tions are not commonly practised in cattle, and cataract which the whole uveal tract is involved may be seen in is only diagnosed if it is large enough to be noticed malignant catarrhal fever. Infectious bovine rhinotracheitis virus may be involved in IBKC and will cause present with other noticeable ocular anomalies.

conjunctivitis; occasionally there is an associated Congenital cataract has been recorded in several uveitis. In calves with coliform septicaemia, acute breeds (Odorfer, 1995) and in Jersey and Friesian cattle anterior uveitis characterized by episcleral congestion, its inheritance is as a simple recessive trait (p. 181). miosis and hypopyon formation may be seen in association There would appear to be a particularly significant incidence of this lesion in the Friesian and Friesian crosses

convulsions are the obvious clinical features. Occasionally, in the UK, demonstrating marked regional variation. In ally, the choroid is involved, and haemorrhage and congenital cataract the nuclear part of the lens is always exudate will be seen in the vitreous. In peracute malignant catarrhal fever severe photophobia, marked palpevision (Plate 52.7). There may also be a posterior polar bral and corneal oedema, mucopurulent discharge and cortical opacity with posterior lenticonus and possible

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hyaloid attachment. Nystagmoid movements of the maternal hypovitaminosis A induced papilloedema and globe may accompany the larger opacities. In both optic nerve atrophy are seen in the neonate. Choroidal these breeds and in the Shorthorn other ocular anomalies including buphthalmia, microphakia, lens luxation be of no clinical significance. Typical papillary colobomata in Hereford cattle with incomplete albinism are

Cataract may be secondary to uveitis, but the aetiology seen bilaterally but have no reported noticeable effect of other capsular and cortical lens opacity remains on vision. However, the same bilateral defect in Charolais (Gelatt, 1971).

lais may blind the affected animal (Plate 52.9). In both The treatment of cataract is by surgery, and two techniques are possible. In young calves of several weeks of genetic studies in the Charolais have indicated that it is age lens discission can be successful if the attendant a polygenic trait (Barnett & Ogden, 1972) (p. 181).

uveitis is minimal (P.G.C. Bedford, pers. obs.). The technique The hyaloid artery is part of the primary vitreous, but technique is a simple one in which the anterior capsule of the with the development of the ciliary vasculature it lens is disrupted using a Bowman's needle inserted into regresses and becomes non-functional. Large remnants the anterior chamber at the dorsal limbus (Plate 52.8).

of this vessel can be seen commonly in cattle (Plate Normal and cataractous lens material is released into the

52.10), but they are of no clinical importance. The vessel anterior chamber and is usually almost completely overlies the optic disc and extends forward from this resorbed, leaving the posterior capsule in situ to retain structure into the posterior vitreous. Blood is often seen the vitreous behind the pupil. Corticosteroids are used within its lumen in young calves up to eight weeks of to suppress the possible phacoanaphylactic inflammation, and remnants of the avascular vessel will persist tory reaction, but in young calves this is minimal. After throughout the animal's life.

the operation cataract reformation is unusual, despite Retinal dysplasia, in which there is typical rosette for- the retained presence of the anterior capsule and the mation and usually non-attachment of the neuroretina, lens epithelium. In older animals the attendant uveitis is seen in Shorthorn cattle with hydrocephalus and mul- renders this technique unacceptable, and extracapsular tiple ocular defects, and in Herefords with microph- lens extraction or phacoemulsification offers the best thalmia, cerebellar hypoplasia and hydrocephalus

chance of success. In the former the anterior chamber is

(Blackwell et al., 1959; Green & Leipold, 1974). Con-opened either through a corneal or limbal section, the

genital retinal degeneration and optic nerve atrophy

anterior capsule removed and the cataractous material

have been seen in association with microphthalmia,

dislocated from the posterior capsule out through the

cataract and cerebellar hypoplasia in calves born of

section. The postoperative therapy must include corti-

dams infected with bovine viral diarrhoea/mucosal

costeroids and, just as in other species, loss of pupil as

disease. Congenital optic nerve degeneration and

the result of iris spasm and posterior synechiae forma-

papilloedema can be produced by maternal hypo-

tion is the common complication that may render the eye

vitaminosis A.

blind. However, the uveitis is usually controllable, and

the overall prognosis for sight is good. In phacoemulsi-

Inflammation

fication the cataract is removed from within the confines

of its capsular bag and then the anterior capsule is

Retinal and optic nerve lesions will accompany several removed. This kind of surgery is less intrusive and the systemic infections including the neonatal pyosep- degree of associated uveitis is usually minimal.

ticaemias, bovine viral diarrhoea/mucosal disease, thromboembolic meningoencephalitis, toxoplasmosis, tuberculosis and rabies.

Diseases of the retina and

Septic chorioretinitis characterized by haemorrhages, optic nerve

exudate and bullous retinal detachment can accompany septicaemia in young calves caused by Escherichia

Both congenital anomaly and acquired disease can

coli, streptococci, Mannheimia spp. and Arcanobac-involve the bovine fundus but, as with cataract diagno-

terium pyogenes infections. Maternal infection with

sis, such lesions are not normally detected unless there

bovine viral diarrhoea/mucosal disease can cause con-

is an associated effect on sight or other more noticeable

genital optic neuritis among other ocular anomalies,

ocular defects are present.

and in thromboembolic meningoencephalitis caused by Haemophilus somnus conjunctivitis and corneal oedema may be accompanied by retinal vasculitis and

Congenital defects

detachment. Blindness is usually due to septic throm- Papillary and peripapillary choroidal colobomata, per- bosis of the visual cortex. Both anterior and posterior sistence of the hyaloid artery, retinal dysplasia and uveitis with possible retinal detachment can accompany

Ocular Diseases • 925

tuberculosis (p. 862). Focal retinitis has been reported fundus and attenuation of the superficial retinal vascu- in bovine rabies (p. 1164).

lature has been described in cattle, and compared with canine progressive retinal atrophy (PRA). It would appear to be a rare condition, and the comparison with

Hypovitaminosis A (see also p. 256)

PRA may have been somewhat premature.

Hypovitaminosis A has been described in three situa- tions with variability of the presenting clinical features.

Glaucoma

It occurs congenitally, and here either papilloedema or optic nerve atrophy is present. In the growing calf there is obvious papilloedema in which the disc becomes very swollen, and the resultant papillary vascular congestion by an elevation of the intraocular fluid pressure (IOP) may cause focal superficial haemorrhage (Plate 52.11). Optic nerve atrophy will occur if the deficiency is maintained. The papilloedema is due to compression due to the impairment of aqueous drainage through a defective or diseased iridocorneal angle, and in the canal, and the accompanying vascular occlusion results in ischaemic necrosis of the nerve (Hayes et al., 1968). In the majority of cases the glaucoma is secondary to inflammation or neoplasia. Uveitis may result in pupillary block due to extensive posterior synechiae formation. Early papilloedema is reversible and optic nerve atrophy preventable if the diet is suitably adjusted. Nyc-

This results in the forward displacement of the peripheral iris, the so-called pathological iris bombé, and is essential in the formation of the visual pigment closure of the ciliary cleft. The presence of exudation rhodopsin, and without rhodopsin the photoreceptors speeds the transformation of the trabecular meshwork degenerate. This process is also reversible should into an impervious physical barrier to aqueous outflow. vitamin A be supplemented soon enough. It is likely Alternatively, these peripheral anterior synechiae and that the sight problems experienced by the calf are ciliary synechiae may form directly to deny bulk more likely to be caused by the optic nerve lesions aqueous drainage. Tumour cells and the products of any rather than the retinal degeneration. In the adult deficient in vitamin A papilloedema is much less noticeable angle to produce secondary glaucoma. and optic nerve atrophy does not occur. Here the outer A primary glaucoma has been recorded in the

segment degeneration is probably more significant in Friesian (Carter, 1960); the condition is inherited as a terms of the effect on sight.

dominant trait and is seen in association with cataract formation and lens luxation.

Treatment of glaucoma in cattle is determined on the

Male fern optic neuropathy

basis of whether the enlarged blind eye is of nuisance

Permanent or transient blindness will accompany weak-

value, and enucleation may be necessary in the pres-

ness, malaise and constipation in the early stages of

ence of associated corneal damage or degeneration.

*male fern (*Dryopteris filix mas*) poisoning. The toxic*

effect is on the optic nerve, and varying degrees of

References

papilloedema and associated haemorrhage are seen

ophthalmoscopically. In severe cases optic nerve

Amman, K. (1968) Eye diseases in ruminants. In Veterinary

atrophy follows the papilloedema.

Encyclopaedia, Vol. II (ed. by E.A. McPherson), pp. 931–45.

Medical Book Co, Copenhagen.

Arthrogryposis

Anderson, D.E. (1970) Cancer eye in cattle. *Modern Veterinary Practice*, **51**, 43–7.
(see p. 177)

Veterinary Practice, **51**, 43–7.

The aetiology of arthrogryposis is open to speculation,

Anderson, D.E. & Chambers, D. (1957) Genetic aspects of and inheritance, fetal lupin toxicosis, manganese deficiency and maternal viral infection have all been considered to be involved. *Miscellaneous Publications of the Oklahoma Agricultural Experimental Station*, **MP48**, 28–33.

Joint deformities, syringomyelia

Barnett, K.C. & Ogden, A.L. (1972) Ocular colobomata in Charolais cattle. *Veterinary Record*, **91**, 592.

and cleft palate may be seen, and blindness is due to

Bedford, P.G.C. (1976) Infectious bovine keratoconjunctivitis. *abnormal photoreceptor development.*

Veterinary Record, **98**, 134–5.

Billings, F.S. (1889) Keratitis contagiosa in cattle. *Nebraska*

Progressive retinal degeneration

Agricultural Station Bulletin, **10**, 247.

Blackwell, R.L., Knox, J.H. & Cobb, E.H. (1959) A hydro-

A form of retinal degeneration characterized by

cephalic lethal in Hereford cattle. Journal of Heredity, 50, nyctalopia, progressive pigmentation within the tapetal

143–8.

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Carter, A.H. (1960) An inherited blindness (cataract) in

Moore, L.J. & Rutter, J.M. (1989) Attachment of Moraxella cattle . Proceedings of the New Zealand Society for Animal

bovis to calf corneal cells and inhibition by antiserum.

Production, 20, 108.

Australian Veterinary Journal, 66, 39–42.

Cordy, D.R. (1978) Tumours of the nervous system and eye.

Morgan, J.H. (1977) Infectious keratoconjunctivitis in cattle

In Tumours in Domestic Animals (ed. by J.E. Moulton), 2nd associated with Listeria monocytogenes. Veterinary Record, edn, pp. 443–53. University of California Press, Los Angeles.

100, 113–14.

Deas, D.W. (1959) A note on hereditary opacity of the dis-

Odorfer, G. (1995) Occurrence and frequency of eye diseases

eased cornea in British Friesian cattle. Veterinary Record, among cattle in Austria. Wein Tierarztliche Monatsschrift, 71, 619–20.

82, 170–8.

Gelatt, K.N. (1971) Cataracts in cattle. Journal of the

Pedersen, K.B. (1973) Infectious keratoconjunctivitis in cattle.

American Veterinary Medical Association, 159, 195–200.

PhD thesis, Royal Veterinary College, Copenhagen,

*George, L.W. (1990) Antibiotic treatment of infectious bovine
Denmark.*

keratoconjunctivitis. Cornell Veterinarian, 80, 999–1002.

*Plowright, W. (1968) Malignant catarrhal fever. Journal of Gilmore, L.O. (1957)
Inherited defects in cattle. Journal of the American Veterinary Medical
Association, 152, 795–*

Dairy Science, 40, 593–5.

804.

Green, H.J. & Leipold, H.W. (1974) Hereditary internal

*Pugh, G.W. (1969) Characterization of Moraxella bovis and its hydrocephalus
and retinal dysplasia in Shorthorn calves.*

relationship to bovine infectious keratoconjunctivitis. PhD

Cornell Veterinarian, 64, 367–75.

thesis, Iowa State University, Ames, Iowa.

*Hayes, K.C., Nielsen, S.W. & Eaton, H.D. (1968). Pathogene-Pugh, G.W. &
McDonald, T.J. (1977) Infectious bovine kera-*

sis of the optic nerve lesion in vitamin A deficient calves.

*toconjunctivitis: treatment of Moraxella bovis infections Archives of
Ophthalmology, 80, 777–87.*

*with antibiotics. Proceedings of the Annual Meeting of the Kampen van, K.R.,
Crisp, W.E., Martin, J.C.D. & Ellsworth, United States Animal Health*

Association, **81**, 120–30.

H.S. (1973) *The immunologic therapy of squamous cell car-*

Punch, P.I. & Slatter, D.H. (1984) A review of infectious

*cinoma. American Journal of Obstetrics and Gynaecology, bovine
keratoconjunctivitis, Veterinary Bulletin, 54, 193–*

116, 569–74.

208.

Leipold, H.W. & Huston, K. (1968) Congenital syndrome of

*Sandhu, T.S., White, F.H. & Simpson, C.F. (1974) Association anophthalmia–
microphthalmia with associated defects in*

of pili with the rough colony type of Moraxella bovis.

cattle. Pathologia Veterinaria, 5, 407–18.

American Journal of Veterinary Research, 35, 437–9.

Lepper, A.W.D. (1988) Vaccination against infectious bovine

*Saunders, L.Z. & Fincher, M.S. (1951) Hereditary multiple eye
keratoconjunctivitis: protective efficacy and antibody*

*defects in grade Jersey calves. Cornell Veterinarian, 41, response induced by pili
of homologous and heterologous*

351–66.

*strains of Moraxella bovis. Australian Veterinary Journal, 65, Taylor, R.L. &
Hanks, M.A. (1969) Viral isolations from*

310–16.

bovine eye tumours. American Journal of Veterinary

Lepper, A.W.D. (1993) A *Moraxella bovis* pili vaccine pro-Research, **30**, 1885–6.

duced by recombinant DNA technology for the prevention

Vogt, D.W. & Anderson, D.E. (1964) Studies on bovine ocular of infectious bovine keratoconjunctivitis.

Veterinary

squamous cell carcinoma ('cancer eye'). XV. Heritability of

Microbiology, **36**, 175–83.

susceptibility. *Journal of Heredity*, **55**, 133–5.

Marolt, J., Burdnjak, Z, Vekelic, E. & Andrasic, N. (1963) Spe-Watson, C.L. (1989) Bovine iritis? *Veterinary Record*, **124**, 411.

cific ophthalmia of cattle. *Zentralblatt Veterinarmedizin*, **10**, Willoughby, R.A. (1968) Congenital eye defects in cattle.

286–94.

Modern Veterinary Practice, **49**, 36–9.

Chapter 53

Other Conditions

A.H. Andrews

Anaphylaxis

927

Signs

Amyloidosis

928

Congestive heart failure

928

Reaction can occur about 20 minutes after the intro-

Diabetes mellitus

930

duction of the antigen. The animal exhibits pronounced

Hypothermia

930

dyspnoea with, on auscultation, bubbling and emphyse-

Lightning strike/electrocution

930

matous sounds. Muscle tremors can occur, which cause

Stray voltage

931

pyrexia to 40°C (104°F). Other reactions include bloat,

Meloidosis

932

diarrhoea, increased salivation, urticaria and rhinitis.

Mycotic diseases

932

Occasionally laminitis occurs. Death is due to anoxia.

Mucormycosis

932

Following i.v. blood transfusion, there are usually hic-

Cryptococcosis

932

coughs and then dyspnoea, muscle tremors, salivation,

Rhinosporidiasis

933

coughing, lacrimation and fever.

Candidiasis (moniliasis)

933

Aspergillosis

933

Histoplasmosis

934

Necropsy

Teat burns

934

Water availability

934

At necropsy usually only the lungs are involved in acute

Heat stress

935

cases, with marked pulmonary oedema and vascular

Acute heat stress

935

engorgement; some animals develop emphysema.

Chronic heat stress

935

Longer-standing cases show hyperaemia and oedema of

Malignant catarrhal fever

935

the abomasum and small intestines.

Anaphylaxis

Diagnosis

Diagnosis depends on the history of recent introduction

Aetiology

of an antigen to which the animal may previously have

Anaphylaxis is the result of antigen–antibody reaction

been exposed. The signs are helpful, being sudden with

and when severe it can cause anaphylactic shock.

severe dyspnoea, urticaria, etc. Haematological exami-

nation shows increased packed cell volume, leucopenia and thrombocytopenia. Biochemical changes include a

Epidemiology

hyperkalaemia and blood histamine levels are raised in

The condition is usually very uncommon. Most cases

some cases. Finally, there is a response to therapy.

follow the parenteral injection of drugs or biological

Differential diagnoses are few because of the rapid

products such as blood. Occasionally, the problem can

onset but acute pneumonia could be confused, although

arise through exposure via the alimentary tract or lungs.

usually there is toxæmia and lesions are more pro-

Signs may be seen in the system exposed, or they may

nounced in the arterio-ventral parts of the lungs.

be generalized. Most cases follow the introduction to

the blood of an antigen to which the animal has already

Treatment and control

been sensitized, but reactions can occur where the

animal is not known to have been exposed previously.

The most effective method of treatment is an intra-

More anaphylactic reactions occur in some herds and

muscular injection of adrenaline (4–5 ml of 1 in 1000 families of cattle than others. Reactions are most prevalent in Channel Island breeds. Initial signs tend to be can be given intravenously, diluted to about a 2 per cent largely of a respiratory nature, but other areas affected solution. Otherwise, corticosteroids can be administered or they may be given immediately following

927

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adrenaline as they potentiate the latter's activity. uraemic, recumbent and comatosed. Death occurs two Antihistamines give variable results, partly because to five weeks after the onset of signs. Almost all cases most histamine is released early in the reaction and will eventually die unless slaughtered. also there are other mediators of the anaphylactic reactions. Various other compounds have been shown Necropsy to alter the reaction of mediators and these in-

clude sodium meclofenamate, acetylsalicylic acid and

At necropsy there are usually one or more chronic sup-
diethylcarbazine.

purative processes present in the organs. The carcass is

Once a reaction has occurred in an animal, the

emaciated, usually with marked oedema. The affected

antigen causing the problem should not be reintro-

organs are enlarged and pale in colour. The kidney and

duced. If a blood transfusion is given, introduce up to

liver have diffuse amyloid infiltration, while in the

200 ml in a 450 kg (990 lb) animal and wait for about 10

spleen it is more localized. The amyloid can be shown

minutes before injecting the remainder.

by aqueous iodine staining.

Amyloidosis

Diagnosis

Diagnosis depends on signs, including an enlarged

Aetiology

kidney, diarrhoea and oedema. There is hypopro-

teinaemia,

proteinuria and hyperfibrinogenaemia.

The aetiology of amyloidosis is uncertain but it is the Blood biochemical examination shows hypomagne- result of a hyperglobulinaemia, probably causing an saemia and a serum calcium level at the lower end of abnormal antigen–antibody response.

the normal range. There are also a high serum urea nitrogen and high serum creatinine. The specific gravity

Epidemiology

of the urine is low and there is a prolonged brom- sulphthalein (BSP) clearance test.

Is a rare condition that is not fully understood. A few

Differential diagnoses are few but include

cases occur as the result of repeated antigen usage in pyelonephritis where there is pus and haematuria, and the production of hyperimmune sera. However, most congestive heart failure in which there is an increased animals are affected sporadically and spontaneously heart rate, respiratory embarrassment and dyspnoea.

following prolonged suppurative infections. The origin of amyloid, which is a glycoprotein, is not certain, but is thought to be due to an abnormality of the

Treatment

antigen–antibody reaction. Renal amyloidosis is the

No successful therapy: cattle should be slaughtered.

most commonly recognized condition, but amyloid can

also be deposited in the liver and spleen. When the

main organ affected is the liver, there tends to be

Congestive heart failure

proteinuria. This leads to hypoproteinaemia and then

oedema of the organ, resulting in marked anasarca. The

Aetiology

diarrhoea produced is partly due to amyloid deposition

and oedema of the intestinal wall. Many of the animals

The condition can follow diseases of the pericardium,

affected are still growing. Foci of inflammation found

myocardium or endocardium.

can include traumatic reticulo-peritonitis, traumatic

pericarditis, salpingitis, mastitis, metritis (Johnson &

Jamison, 1984) and nephritis. Cases have occurred

Epidemiology

following repeated hormonal injections in donor cows

It is relatively uncommon and mainly affects older

which are superovulated several times.

cattle. Pericarditis or hydropericardium can interfere with the normal filling of the heart during diastole.

Pulmonary hyperaemia, which may occur in diffuse

Signs

fibrosing alveolitis, can result in right-sided congestive

Usually, the affected animal is thin and emaciated. In

heart failure. In cattle, the main cause of myocardial

most cases there is marked anasarca with an enlarged

disease is foot-and-mouth disease. Endocardial diseases

liver palpated in the right sublumbar fossa and an

are the most common and are usually due to infection

enlarged kidney with loss of its lobular structure when

or inflammation.

palpated via the rectum. There is usually polydipsia and

When extra demand is placed on the heart, or the

a profuse watery diarrhoea. The animals later become

myocardial activity is reduced, then compensation can

Other Conditions • 929

occur by an increased heart rate, increased filling of the

there is pulmonary congestion and oedema. In left-

ventricles and improved cardiac performance. There sided failure there is subcutaneous oedema, hydro- are also dilatation and hypertrophy. Venous return thorax, ascites and hydropericardium.

increases in speed and blood distribution changes so that there is an increase in blood volume, a decrease in renal blood flow and sodium retention. Cardiac

Diagnosis

response is reduced and the animal is less able to cope with unusual exercise or other emergencies (Blood et

Diagnosis is by the signs, particularly those of oedema.

al. , 1983). There is a decrease in exercise tolerance and The heart rate is elevated and there is an increased area

once the compensating mechanisms have been over- of cardiac dullness; abnormal heart sounds may be come then the heart cannot cope and congestive heart present, depending on the cause. Insertion of a needle failure develops. Many of the signs of cardiac insuffi- into a vein shows a markedly increased venous pres- ciency are the result of increased venous pressure. They sure. Aspiration of fluid from oedematous areas shows

result in congestion of organs or oedema and the
it to be a transudate containing large amounts of
decreased output produces tissue hypoxia.

protein. There is also a proteinuria.

Failure of the right-sided ventricle causes congestion
of the major circulation with reduced blood flow
through the kidney, resulting in anoxic damage to the

Differential diagnosis

glomeruli and venous congestion of the portal system

Differential diagnosis includes amyloidosis but diar-

of the liver, and eventually there is transudation into

rhoea tends to be marked and the kidneys are enlarged

the intestinal lumen giving rise to diarrhoea. Left-sided

without heart abnormality. In chronic peritonitis there

ventricular failure results in engorgement and oedema

is often some pain and the aspirated abdominal fluid

of the lungs. Both left and right-sided failure can occur

contains many leucocytes. In bladder rupture there is a

together.

normal heart and urine is present in the abdomen. In

liver fibrosis the heart is normal and there is usually

Signs

jaundice or photosensitization. Parasitic gastroenteritis

occurs in a younger animal and there is oedema, mainly

Early signs are of reduced exercise tolerance with

of the submandibular area, with a high faecal egg count.

increased respiratory effort and tachypnoea following

Fascioliosis could confuse, but again there is usually

light exercise. Pulse and respiratory rates take a long

only submandibular oedema and a faecal egg count.

time to return to normal. On percussion the heart area

Fog fever (p. 866) could cause problems in diagnosis but

may be enlarged.

usually several animals are affected quite suddenly in

In left-sided failure: lungs are mainly affected so that

the autumm. Anaphylaxis is also a sudden problem with

there is a cough, tachypnoea and hyperpnoea. The heart

no cardiac signs.

rate is increased and a murmur may be present in the

region of the aorta or left atrioventricular valve. On

auscultation there are moist noises at the ventral edges

Treatment

of the pulmonary field and on percussion increased dullness on the lower borders of the lung. Later on The primary cause should be treated as effectively as there is cyanosis and dyspnoea.

possible, but with most cases the prognosis is poor.

In right-sided failure or cor pulmonale: appetite is Where oedema is present it is partly due to sodium poor and the animal is dull with a rapid loss of condition and so the salt intake should be kept as low as possible. Diuretics such as frusemide or the skin of the lower part of the body, the neck and hydrochlorothiazide are useful and they can be given the submandibular space. There is ascites, hydropericardium by injection or orally. The heart can be treated to improve contractility by use of etamiphylline camsylate, enough to be palpated in the right sublumbar fossa. The theophylline or digitalis derivatives. The use of digitalis heart rate tends to be increased and there is hyperkalemia by mouth is probably of limited value because of break-

*pnoea and often slight tachypnoea. There is a marked
down in the rumen. Parenteral administration of
jugular pulse. Prognosis usually poor in all cases.
digitalis extracts is little used for several reasons: intra-
venous use could result in toxicity whereas intramus-
cular administration gives variable results. There are no
Necropsy*

*licensed preparations. Little can be done to control the
At post-mortem examination there are abnormalities of
congestive heart failure except to treat all infections
the pericardium, myocardium or endocardium. Usually,
quickly and adequately.*

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Diabetes mellitus

*rhage and acute synovitis. Some cases show haemor-
rhages of the adrenal glands and oedema of the iliac
This is a very unusual condition in cattle compared with
and jejunal lymph nodes.*

its relatively frequent occurrence in dogs and cats.

However, lesions can occur in the pancreas resulting in

Diagnosis

this condition. It is usually seen in old cattle and often animals will have had other problems such as fatty liver. Diagnosis depends on the history and signs. Increased The signs are loss of condition with polydipsia and serum glucose and phosphorus levels occur with cooling polyuria. There is no rise in temperature and the urine but fall during recovery.

contains both ketones and glucose. Biochemical examination of the blood shows hyperglycaemia. Usually

Treatment

treatment is not contemplated.

Treatment is by immersion in warm water, which results in quicker rewarming than use of a heat pad or heat

Hypothermia

lamp. A hot air environment could be constructed with a straw bale shelter and a hot air heater. Control is

Aetiology

by ensuring that adequate shelter is provided or by housing cattle during the bad weather.

This is also called cold stress and is mainly seen in young calves in severe climatic conditions, particularly

where there is wind chilling as well as a cold ambient

Lightning strike/electrocution

temperature. The problem is not encountered anything like as commonly as in lambs.

Aetiology

This is exposure to high voltage electric currents, either

Epidemiology

natural or generated by man.

Exposure to cold conditions mainly occurs in calves of cows calving outside in the winter months with no

Epidemiology

shelter. It is particularly prevalent in suckler calves and occurs more frequently in countries such as Canada and

The condition is uncommon but it is often of interest as Australia than say Britain. Mortality is usually higher in it is one of the few problems that farmers have usually calves born in winter and early spring. There is a delay insured against. Problems arise from lightning, exposure of electrical wires or faulty wiring or earthing in from colostrum in cold-stressed calves. Primary

farm buildings. Cases may be single or a group. In many hypothermia with no associated disease can be seen in instances, damp ground or floors help to conduct the young calves, and secondary hypothermia with no other electricity. Low voltages of 110–220 V are sufficient to disease has been recorded in older calves.

kill cattle. Outside, trees such as oak, poplar, elm and conifers are all prone to lightning strike.

Signs

Signs

The rectal temperature may be low and can be defined as a temperature less than 37°C (98.5°F) and there is The signs in the severe form are such that animals die shivering. Shivering appears to be more intense during without a struggle. Burns or singeing may be seen the initial period following exposure to cold. Most because of the severity and often they involve the calves have no problems in the cold but some are muzzle and feet. Death is usually due to paralysis of the depressed and stand or walk about stiffly. A few animals medullary centre, accompanied in some cases by ven-

may remain in sternal recumbency. The heart rate tends to be raised but respiratory rate is low.

becomes unconscious for a varying time and there is usually some sign of a struggle. After regaining consciousness there may be dullness, blindness, paralysis of
Necropsy

one or more legs, and surface hyperaesthesia. The signs

At post mortem the most common lesions involve the
may persist or slowly disappear over a period of up to
limbs and include subcutaneous oedema and haemor-
two weeks. If burns are present, sloughing of the skin
rhage. Experimentally, more haemorrhages occur in the
in the area is seen. Minor problems may occur such as
hindlimbs. Some animals show oedema of the ventral
the animal may jump, be restless, show periodic con-
sternum. The joints may show mild to severe haemor-
vulsions or be knocked down.

Other Conditions • 931

In lightning strike the animal is usually close to a

Occurrence

fence, barn, trees or a pond. Often there are signs of
There are a large number of different origins for this
burning affecting these objects. The animal itself may
problem. Stray voltage is an electrical current passing
show singeing of the hair or burn marks on the muzzle
between two points. Should an animal come into
and feet. Half-chewed food may be present in the
contact with these points the current will pass through
mouth. The animal quickly becomes distended with gas
the animal. Cattle are more sensitive to electrical volt-
and decomposes rapidly. Blood often exudes from the
ages than human beings, who often do not feel any
nostrils, rectum and vulva. The pupils are dilated and
shock due to wearing clothes and boots which can insu-
the anus relaxed. There tend to be petechial haemor-
late them from the problem. While there is individual
rhages throughout the body and the viscera are
variation in the susceptibility to electricity, most cases
congested. The superficial lymph nodes are often
will only be detected when several animals show similar
haemorrhagic.

signs. The most common place for this to occur is in the milking parlour, although feed and water troughs as

Diagnosis

well as other farm building equipment can be involved.

The history may contain the fact that there has been

Diagnosis depends on the history of a storm, position

some alteration or modification of the electrical supply

of the animal near trees, etc. and sudden death of a

or there has been some building. Otherwise most prob-

group of animals. The signs may also be helpful such as

lems result from old and exposed wiring, often with

singeing of the hair, burns on the muzzle and feet and

damage to the insulation covering or vermin damage.

evidence of sudden death.

Signs

Differential diagnosis

These will vary and are related to the area of the stray

Differential diagnosis includes anaphylaxis but there is

voltage. If the source is in the parlour, the cattle are often marked pulmonary involvement. Acute heart failure

reluctant to enter, are fidgety during milking and will

can cause problems but there is engorgement of visceral
rush out of the parlour after milking. If the source is a
veins and macroscopic or microscopic myocardial
water trough it may be avoided by the cows or they may
lesions. Brain trauma can occur but usually there is a
lap rather than drink normally. Other signs include rest-
haemorrhagic lesion of the brain. Nitrate/nitrite poi-
lessness, muscular twitching, etc. Milk production may
soning results in sudden death but methaemoglobin is
suffer and feed intake as well if the feed area is affected.
present. Anthrax can be confused, but stained blood
smears assist in determination. Bloat is a possibility but

Diagnosis

on necropsy the front part of the animal is congested,
there is a distended rumen and sometimes froth
This is often difficult; any behavioural change will
present. Blackleg results in swelling of the part affected
usually only be seen in the area of the stray current.
with the causal organism present.
If several animals are showing similar discomfort or
behavioural changes then diagnosis is made more easy.

Confirmation will depend on a qualified electrician and

Treatment

will require a sensitive voltmeter. It may also be neces-

No treatment is usually given as often the cattle

sary to set up the conditions that cause the shock, i.e.

are either better or dead before any therapy can be

have the milking equipment working.

administered. Central nervous system stimulants can be

given. Artificial respiration may be helpful. Although

Differential diagnosis

nothing can be done about lightning strike, all

Behavioural changes will usually only affect a single

electrical installations should be properly fitted and

animal and will be less likely to be related to a par-

earthed.

ticular area of the farm. BSE can result in signs such as

reluctance to enter the parlour and difficulty in milking,

but would always involve the same animal. Other signs

Stray voltage

would also occur.

While not a very common problem, the signs are often

Control

difficult to determine and also the origin is hard to find.

These stray electrical currents result in mild electric

Ensure all electrical installations and equipment are

shocks to the animals and cause discomfort, often

properly earthed and maintained. Have wiring checked

resulting in apparent behavioural problems.

periodically.

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Meloidosis

Mycotic diseases

Aetiology

Mucormycosis

The cause is Pseudomonas (Malleomyces) pseudo-Aetiology

mallei. It can survive for long periods in the soil and There are various fungi of the family Mucoraceae that

water.

can cause infection, including the genera Absidia, Entomophthora, Mortierella, Mucor, Rhizopus.

Epidemiology

The condition is primarily a disease of rodents but occa-

Epidemiology

sionally it spreads to man and farm animals such as
The fungi occur usually in soil and water and are
sheep, goats, horses and pigs. It is very rare in cattle. The
often plant pathogens and food decomposers. They are
disease was first recognized in South East Asia but it is
opportunistic.

now seen in Australia, Malaysia, Papua New Guinea
and Nigeria. Infection is passed in the faeces of rodents

Signs

and is spread by ingestion of contaminated feed or
water, by insect bites, wounds and possibly inhalation.

Infection can cause a necrotic placentitis in cattle

Mortality is high and disease lasts about two to eight
resulting in abortion at three to seven months' gesta-
weeks.

tion. There is necrosis of maternal cotyledons and
yellow, raised, leathery lesions on the intercotyledonary
areas. Granulomatous lesions of the mesenteric and

Signs

mediastinal lymph nodes can occur. Calves can develop

The main signs are a marked pyrexia with anorexia and

*alimentary tract lesions after prolonged antibiotic
a thick yellow exudate from the eyes and nose. Nervous
therapy. Following acidosis the ruminal wall can be
signs may be exhibited. Some cattle have infection
invaded with Rhizopus spp.*

without signs.

Diagnosis

Pathology

*Hyphae are present on smears of cotyledons, fetal
stomach and skin. The lymph nodes need to be exam-
There are multiple abscesses in many areas of the body
ined. There may be a history of antibiotic usage and on
as well as in the subcutaneous tissues and associated
post mortem there may be abomasal ulceration. The
lymph nodes. The size of the abscesses is variable from
animal will become black tinted.*

a few millimetres to over 2.5 cm in diameter.

Prevention and control

Diagnosis

*As the fungi are ubiquitous it is impossible to control
This depends on demonstrating the causal organism by*

properly. There are no satisfactory antibiotics.

culture from pus, nasal discharges or blood.

Cryptococcosis

Treatment

Aetiology

There is little information available on satisfactory

The cause is Cryptococcus (Saccharomyces) neoformans-treatment of meloidosis. Chloramphenicol is used in

mans (synonyms C. hominis, Torula histolytica).

man and in vitro tests suggest oxytetracycline may be of use in animals, and perhaps novobiocin, chloramphenicol and sulphadiazine. Treatment is to be discour-

Epidemiology

aged because of the possibility of spread to humans.

The incidence is worldwide and is high in areas of warmth and moisture.

Control

Signs

Infected animals: slaughtered, disposed of by burning. In-contact animals: slaughtered, the premises

Latent: There is a mastitis in many cases with no visible disinfectant signs in the udder or milk.

Other Conditions • 933

Severe: The quarter(s) swells slowly and becomes firm

Treatment

and swollen. The subcutaneous tissue also develops

Polyps are best removed surgically.

oedema, which persists for several weeks. There is

often some discomfort with the legs held apart. Pyrexia

occurs up to 40°C (104°F) with anaemia. The supra-

Candidiasis (moniliasis)

mammary lymph nodes are enlarged. Milk production

is reduced but the milk itself shows few changes except

Aetiology

a few flakes. Later, in persistent cases, there is a watery

The main causal agent is Candida albicans but C. trop-secretion.

icalis, C. krusei, C. parapsilosis and C. guilliermondii can also initiate disease.

Mild: A swelling of the quarter(s) is seen with little effect on the milk.

Occasionally a granulomatous

meningoencephalitis may develop or the nasal mucosa

Epidemiology

may show nodular lesions.

The organisms are worldwide and found in animal

faeces.

Pathology

Infection is mainly confined to the udder, although

Signs

*there may also be reddening and thickening of the
brain tissue and oedema, and naso- or oropharyngeal*

*A chronic pneumonia can occur with dyspnoea plus
nodules.*

*only a moderate fever. There is a profuse stringy sali-
vation and brown-streaked nasal discharge. Abortion
can occur and there can also be mastitis with mild*

Diagnosis

transient signs that are self-terminating. Where there is

Diagnosis is by direct examination of the fluids.

*severe mastitis it may follow the use of intramammary
tubes. The udder is swollen and spongy and the animal's*

Prevention and control

temperature is 40–41.5°C (104–107°F).

Ensure proper disinfection.

Diagnosis

Rhinosporidiasis

Isolation of the organism provides the diagnosis.

Aetiology

The causative organism is Rhinosporidium seeberi.

Treatment and control

There is a limit to what can be done but amphotericin

Epidemiology

B can be successful. An iodine in liquid paraffin intra-mammary infusion can be left in the udder for 10–15

The disease is distributed worldwide.

minutes, and then stripped out. This is repeated in seven days, and oral iodides may be given if necessary.

Signs

There is the formation of large polyps in the posterior

Aspergillosis

nares and this interferes with respiration. The lesions are small, about 0.5–3 cm in diameter. They are soft,

Aetiology

friable and bleed. The surface of the polyp shows many

There are many Aspergillus spp. including Aspergillus small white specks. The nasal discharge results in dys-fumigatus, A. flavus, A. niger and A. terreus.

pnoea with mucopurulent and bloody discharge.

Pathology

Epidemiology

There is a polyp of papillomatous epithelium, which is

The organisms are widespread. Placental infection of hyperplastic and contains numerous sporangia.

cattle is thought to be via abomasal ulcers or from the respiratory tract. It also occurs following inhalation of spores from mouldy straw, hay and sugar beet pulp.

Diagnosis

Outbreaks of abortions can occur following the feeding

Isolation of the organism provides the diagnosis.

of mouldy silage.

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Signs

may lead to side-effects. The imidazoles may be satisfactory.

Aspergillosis is a quite common cause of abortion with a placentitis. Abortion is in the sixth to eighth month of pregnancy. Another form is a fatal gastroenteritis with

Teat burns

ulceration of forestomachs and oesophagus. Pulmonary

aspergillosis also occurs in animals subjected to diar-

Epidemiology

rhoea and poor ventilation. There is fever and dyspnoea.

In parts of the world where forest and grass fires are common, burns can result in damage necessitating the

Diagnosis

slaughter of cattle. However, in other cases the animals

Examine placental direct smears for fungi. Culture will survive but there may be injury to the teats or stomach contents or cotyledon.

udder. In such cases it is necessary to provide an accurate prognosis. Often several teats are affected.

Treatment and control

Signs

Nystatin can be used in the treatment of diarrhoea.

The lesions can be assessed according to the severity of

There is no effective preventive but avoid feeding the burn (Morton et al. , 1987).

mouldy silage to cows in mid to late pregnancy. Definitely mouldy straw or hay should not be used in

Mild burns: These are reddened with a loss of the

enclosed buildings, and preferably not at all.

outer, white, paper-thin tissue, which sloughs. A teat may have normal tissue interspersed with multiple

Histoplasmosis

small black areas over the teat surface or a uniform relatively thick black or red–brown surface layer. There

Aetiology

may be areas of sloughing or crusts that develop in the

There are two species causing infection, Histoplasma

tissue and milk is apparently normal. In all cases the

capsulatum and H. duboisii.

teats are pliable on palpation.

Severe burns: The teats tend to be dull brown or black, Epidemiology

dry and often are corrugated. If sloughing has occurred

Whereas H. capsulatum is found worldwide, H. duboisii then a thick layer (over 1 mm thick) of tissue is

is only found in Africa. It is a rare condition but can

sloughed. Underneath there is red haemorrhagic tissue.

occur in man and other species. Histoplasma capsula-

On palpation the teat is leather-like and lacks pliability.

tum is found in the soil. Infection follows inhalation of In some cases the teats tend to be distorted, especially

contaminated dust and entry to the lung.

in heifers.

Prognosis

Signs

With mild lesions prognosis is good. In severe lesions

Affected cattle may show chronic emaciation, dys-

prognosis is variable but tends to be better in cows than

pnoea, diarrhoea, swelling of the brisket and grinding

heifers. Some cows only show partial return to function

of the teeth. At post-mortem examination there is an

with reduced milk flow.

enlarged liver, ascites, oedematous thickening of the

large intestine and interstitial emphysema. Histologi-

cally, eosinophilic round bodies can be found in

Treatment

endothelial cells.

Treatment can increase recovery. Application of 0.5 per

cent cetrimide in lanolin daily is helpful. However,

Diagnosis

healing is slow, taking about 14 weeks. When the surface

skin starts to peel antibiotic ointment is helpful. Teat

*Diagnosis is from the signs, and samples should be
orifice restrictions often respond to surgery.*

taken for culture and histology.

Water availability

Treatment and control

Amphotericin B given intravenously at 1 mg/kg body

*Water debility need not necessarily damage cattle in the
weight daily has been effective but a prolonged course
dry season in semi-arid regions. Some breeds are obvi-*

Other Conditions • 935

*ously less susceptible. They are less affected when they
air is hot and humid, then every attempt must be made
are on pastures containing a very low crude protein
to ensure good air flow by louvred windows, fans, etc.*

level. If fed a high plane of nutrition without water,

The cattle should receive an adequate amount of water.

problems could arise, but in practice this is unlikely to

Feeding at night will also ensure maximum heat pro-

*occur. The amount of water required varies but is obvi-
duction is during the cool period.*

ously higher in a dairy cow than a beef animal. Animals

are capable of being watered only once every three days if necessary but they should not have to walk more than

Malignant catarrhal fever

about 2.5 km to it. If water deprivation occurs there is a reduction in milk or meat deposition. If dehydration

Aetiology

persists there is circulatory collapse when the level of sodium reaches 170 mEq/l.

Also known as bovine malignant catarrh and malignant head catarrh. Caused by two different agents but clinically identical. In many countries only one of the agents

Heat stress (see Chapter 8)

is present. The African strain, (alcelaphine herpes virus 1 and 2 or bovid herpes virus 3) is wildebeest-associated.

Acute heat stress

The European, North American and Asian strain has not been characterized but is sheep-associated.

If the heat produced by the animal and the heat absorbed from the atmosphere are higher than the heat lost then the animal's temperature will rise. It thus

Epidemiology

develops hyperthermia and if the rise is great and sudden the animals will pant, salivate excessively, Malignant catarrhal fever (MCF) has a sporadic world-wide distribution. It mainly affects single animals, become restless and then prostrate. Problems often occur if cattle are made to walk long distances. Heat although outbreaks have occurred. The wildebeest stroke can occur at a normal outside temperature if strain is seen in Africa and in zoological gardens. The animals are packed tightly in poorly ventilated vehicles sheep-associated strain occurs when sheep associate travelling during the hottest parts of the day.

with cattle, which are considered 'dead-end' hosts. The If action is not quick death occurs. Affected animals African form is more easily transmitted, transferred via should be sprayed with cold water. They should be kept the placenta into the fetus. Soon after birth, the wildebeest calf sheds infection which can be contracted by Clipping the hair, particularly along the mane, withers other species. Transmission of the sheep form is not

and backbone, will assist but such animals are then susceptible, although cattle have usually been in contact with pregnant or lambing sheep. The virus is fragile and the shade.

does not survive more than a day outside the host.

There may be many distinct MCF virus forms.

The incubation period is shorter for the African form.

Chronic heat stress (see Chapter 8)

Following infection, there is an eclipse phase of nine to seventeen days, then viraemia continues until death.

The chronic or subacute form occurs more frequently

After a further three to fifteen days, signs develop, fol-

lowed by death five to ten days after their onset. Trans-

mission is probably by inhalation or possibly blood

by panting and sweating. They can also reduce heat pro-

transfusion.

duction by reducing food intake, drinking more and

reducing activity. Cattle are able to accept high midday

temperatures if the evenings and nights are cool.

Signs

In chronic heat stress there is decreased milk production, reduction in weight gain and sexual maturity.

Cases are sporadic. Animals are very ill with anorexia, There is also some loss in fertility, particularly involving pyrexia (40.5–41.5°C; 105–107°F), depression and loss spermatogenesis in the bull. When animals are affected of condition. An early pathognomonic sign is an intense they should be provided with shelter or otherwise given scleral congestion with bilateral keratitis and corneal access to well-ventilated shady areas. Oestrus suppression starting at the edge of the sclera, causing blindness may also occur. When cattle are grazed in very hot ness. Early lesions also involve the buccal mucosa, with conditions grazing should be restricted to the evenings, reddening of the lips, gingivae and muzzle. Erosions nights and mornings. If necessary zero-grazing may develop, including necrosis of the tips of the labial need to be adopted. In such circumstances, where the papillae and the mouth corners.

Other signs vary. They can include nervous signs such as muscle tremors and hyperaesthesia. The superficial histological presence of vasculitis confirms diagnosis. lymph nodes are grossly enlarged. There is profuse There is leukopenia. Serological tests can be used in the mucopurulent oculo-nasal discharge which can encrust African form. Viral isolation or transmission can be around the nostrils and eyes. There is often excessive attempted.

salivation, followed by dyspnoea and stertor due to exudate accumulation. Faeces vary between profuse diarrhoea and soft, scanty faeces. Laminitis and der-Differential diagnosis

matitis can occur in the sheep-associated form.

Differential diagnosis includes mucosal disease, but

At later stages, more prominent nervous signs may ocular lesions are not severe and there is no lymph node occur, such as incoordination, leg weakness and nystag-enlargement. Rinderpest and foot-and-mouth disease

mus followed finally in some by head pressing, convulsions are herd problems with no severe ocular lesions. Infections or paralysis.

tious bovine rhinotracheitis usually affects several Uncommonly, a mild form occurs with mild, transient animals, with mainly respiratory signs. Calf diphtheria fever and some oral and nasal mucosal erosions. These does not produce ocular signs.

animals can recover.

Treatment and control

Necropsy

There is no treatment or suitable vaccine. Infected Lesions occur in many parts of the body including the animals should be separated from others. Cattle should respiratory, alimentary and urinary tracts. They include not be grazed with sheep, particularly at the time of varying degrees of haemorrhage, hyperaemia and dis-lambing, nor near wildebeest. Bought-in sheep should crete or extensive erosions. The eyes show scleral con-come from disease-free farms. gestion and keratitis. The lymph nodes show marked

enlargement. The skin around the coronets may show lesions. Histologically, the epidermis shows extensive hydropic degeneration and vesicle formation with

References

rupture. The dermis shows vasculitis with proliferation and necrosis and marked lymphoid cuffing.

Blood, D.C., Radostits, O.M. & Henderson, J.A. (1983) *Veterinary Medicine*, 6th edn, pp. 273–5. Baillière Tindall, London.

Johnson, R. & Jamison, K. (1984) Amyloidosis in six dairy

Diagnosis

cows. *Journal of the American Veterinary Medical Association*, **185**, 1538–43.

History helps with a record of other species contact

Morton, J.M., Fitzpatrick, D.H., Morris, D.C. & White, M.B.

and one animal affected. Nervous signs, lymph node

(1987) Teat burns in dairy cattle – the prognosis and effect

enlargement, scleral congestion, corneal opacity and

of treatment. *Australian Veterinary Journal*, **64**, 69–72.

Chapter 54

Major Poisonings

C.J. Giles and A.H. Andrews

Introduction

937

in general, must rely on the vigilance and safety stan-

Diagnosis of poisoning

937

dards of industry and the monitoring authorities.

Relationship between the veterinary clinician and the

Most instances of poisoning in cattle therefore are

diagnostic laboratory

939

not the result of large-scale incidents but arise from

Principal toxicoses

940

accidental access by cattle to toxic substances at the

Organophosphate and carbamate poisoning

940

local farm level either by cattle moving from a safe to

Lead poisoning

944

a toxic environment or by the inadvertent introduction

Ragwort poisoning

945

Bracken poisoning

946

by man of a toxicant into the animals' previously safe

Yew poisoning

947

environment.

Copper poisoning

948

Poisoning in cattle can vary greatly in severity, mor-

Fluoride poisoning (fluorosis)

949

bidity and mortality. In the most severe cases, where a

Nitrate/nitrite poisoning

950

large number of cattle have consumed toxic quantities

Oak poisoning

951

of poison very high morbidity and mortality can result.

In contrast, mild episodes occur where the clinical

effects are transient and few animals suffer harmful

Introduction

effects. Some of such cases are often so insignificant that they are missed or misdiagnosed.

The nature of many farming practices such as the wide-spread and increasing use of agrochemicals and the wide

Diagnosis of poisoning

variety of potentially toxic plants, coupled with the

The diagnosis of poisoning in cattle is not easy, as often

innate inquisitiveness of cattle to investigate (often by

it is the last thing a clinician will consider when pre-

licking) new or unusual substances, means that many

sented with an outbreak of disease and not infrequently

cattle live in a potentially toxic environment. It is

it is a subject about which he knows little. In many cases

perhaps surprising therefore that despite this, incidents

the evidence of intoxication is merely circumstantial,

of poisoning remain comparatively uncommon in cattle.

the materials required to establish a definitive diagno-

Most toxic dangers to cattle are well understood and

sis have long disappeared and in many cases the clini-

documented. This fact, together with the usually respon-

*cal signs are vague and confusing. However, poisoning
sible and diligent approach to the handling of toxic
incidents are often serious, potentially litigious and
chemicals and the correct and proper management of
hence must always be handled with care and vigilance.*

pasture, considerably reduces the risk of cattle gaining

*There are essentially three stages in establishing a
access to poisonous substances. Most instances of poi-
diagnosis of poisoning:*

*soning arise, therefore, by accident, usually as the result
of human error, ignorance, neglect or, rarely, malice.*

(1)

*Recognition that the incident is probably a case of
Such errors, of course, may not always be under the
intoxication.*

direct control of the stockworker, for it may be the

(2)

*Reaching a presumptive diagnosis of the toxicant
actions of a third party, often unconnected with live-
or class(es) of toxicant.*

stock that are to blame for a poisoning incident. As an

(3)

Establishing a definitive diagnosis of the intoxication.

example, the environment and feed of cattle, like all

It is not possible in many cases for all three stages to be

livestock and indeed humans, may be subjected to the

accomplished.

effects of industrial pollution. However, incidents where

there is gross contamination of the environment or

Recognition of apparent intoxication

where an accident results in the release of highly toxic

substances are very uncommon and for the maintenance

Rarely is an outbreak of disease in cattle immediately

of such a state the livestock farmer, like the population

attributable to poisoning on clinical or pathological

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grounds alone. Before the clinician comes to such an

Any recent use of agrochemicals should elicit ques-

opinion he needs to consider fully the history, epidemi-

tioning of farm staff as to their correct usage and the

ology and clinical signs presented and consider care-

disposal and storage of concentrate. Anything in the fully the differential diagnosis in order to exclude the environment of the cattle that would not normally be more common non-toxic causes of the condition.

present should be investigated with suspicion; for

Poisoning incidents should be suspected in the

example, old farm machinery or rubbish left in fields,

following circumstances:

lead from old batteries and discarded sump oil may be

the sources of various toxicants. The water supply must

- The onset of disease is clearly associated with a always be critically examined, especially if the supply is change in management of the affected animals, for provided by means other than a piped mains supply.*

example a change in feed, movement to a new envi-

Could the supply have been contaminated by local

ronment or a concurrent agricultural management

dumping of toxic rubbish, or run-off from nitrogenous

practice, e.g. spraying or dipping.

fertilizers from fields? Only when there is no known

- The epidemiology of the disease is not that*

indication of local contamination should more distant expected of an infectious disease, for example the sources, e.g. industrial sites, be considered.

condition may quite obviously arise simultaneously

The clinician's best initial guide to the type of toxin in several animals or groups rather than appear to cant involved is detailed appraisal of the clinical signs be the result of spread.

and post-mortem findings, where appropriate, and to

- *The clinical signs as presented are not typical of the correlate these with the known effects of the major more common infectious, parasitic or metabolic types of toxicant (Table 54.1). However, few toxicants diseases with broadly similar clinical pictures.*

produce a clinical picture not shared with other poisons

- *There is circumstantial evidence that animals may or non-toxic causes and several can produce a wide have had access to unusual materials.*

array of clinical signs.

- *The differential diagnosis of the disease as determined by clinical and post-mortem examination*

includes poisoning as a possible cause.

Establishing a definitive diagnosis

Having attempted to form an opinion as to the type of toxicant involved, the veterinary clinician is now in a

Presumptive diagnosis of the toxicant

position to attempt to reach a definitive diagnosis. To

Having established the incident may be a case of

achieve this, in most cases, especially when chemical

intoxication the clinician needs to consider what agent

rather than plant toxicants are involved, he will require

or type of agent might be responsible. In some cases this

the assistance of a diagnostic laboratory. In some cases

may be straightforward as the history and circums-

a routine thorough gross and histological examination

tantial evidence of poisoning may direct the clinician

by the laboratory may provide strong supportive evi-

immediately to the probable cause. More frequently,

dence of the toxicant as may the botanical examination

however, it is far from obvious and requires a detailed

of rumen contents in cases of plant poisoning, but in the

consideration of the clinical signs, the post-mortem

majority of instances a definitive diagnosis of poisoning findings (if appropriate) and the circumstantial evidence is dependent on the finding, by chemical analysis, of the toxicant in the tissues, ruminal or intestinal contents or food of the poisoned animal. The results of such chemical analyses must always be interpreted carefully and picture as observed? This inquiry needs to be wide-ranging. A full history should always be taken; the mortem findings. The question to be answered is: Would clinician should examine the animals in the housing or the observed level of chemical (as determined by analysis) in the particular tissue at the time of examination should not merely concentrate on the animals themselves as it is necessary also to inspect the environment observed? Only when this question can be answered

for possible sources of toxicants. It is vital, of course, in the affirmative can a definitive diagnosis result. This always to inspect the feed but this should not automatically be viewed as the prime suspect source, although constraints usually imposed on veterinary laboratory a recent change in feed is strong supportive circum-examination, and the clinician must view attempts at substantial evidence that it may be the source of intoxication. In grazing animals the pasture should be thoroughly inspected for the presence of known toxic suspect batch of feed may have long since disappeared plants.

and despite a diligent examination the analyst is unable

Major Poisonings • 939

Table 54.1

Initial linking between clinical abnormality and type of poisoning.

System

Abnormality

Suspected class of poison

Chemical

Plant

Alimentary

Diarrhoea

Heavy metals, organophosphates,

Solanine containing, oak, GI

ionophores

irritants, ragwort

Colic abdominal pain

Heavy metals, formaldehyde, urea

Oak, GI irritants

Hypersalivation, vomiting

Organophosphates, urea

GI irritants, rhododendron

Central nervous system Depression, coma

Ionophores

Oxalate containing, solanine

containing

Hyperexcitability, hyperaesthesia,

Heavy metals, nitrofurans,

Atropine containing

convulsions

organochlorines

Eye

Mydriasis

Lead

Atropine containing, Hemlock,

Cicuta sp.

Miosis

Organophosphates

Blindness

Lead

Respiratory

Dyspnoea

Nitrate/nitrite

Cyanide containing

Skin

Photosensitization

St John's wort, ragwort

Gangrene

Ergot

Locomotor

Lameness

Fluoride

Ergot

Urinary

Haematuria

Bracken

Haemoglobinuria

Copper

Brassica spp.

Allium spp.

All

Wasting

Pyrrolizidine containing

Sudden death

Cyanide containing, yew,

water dropwort

GI, gastrointestinal.

to determine a chemical cause of the signs observed.

Table 54.2

Specimens submitted for toxicological examination.

*The veterinarian at this stage must conclude the case,
after referral if necessary, as merely one of suspected*

Tissue

Amount usually required

intoxication.

Serum (separated from clot,

up to 10 ml

not haemolysed)

Relationship between the veterinary

Urine

50 ml

clinician and the diagnostic laboratory

Liver

100 g

Kidney

100 g

If a significant number of investigations are to be con-

Other tissues

100 g

cluded beyond the stage of ‘suspected intoxication’ by

Rumen contents

500 ml

the clinician, a working cooperation must develop

Feed

minimum 100 g

between the veterinary clinician and the diagnostic laboratory. Once intoxication is suspected the golden rules are to contact the laboratory earlier rather than later and to take too much material for examination should be packaged or sent and any special instructions. rather than too little. When the clinician suspects intoxication Accompanying all specimens must be the clinical information and has concluded the initial clinical and environmental assessments contact should be made with a given and the time of death. As a general rule, contain laboratory experienced in diagnostic toxicology. The ers should be glass or plastic and tightly sealed, different clinician should inform the laboratory of the following: ent organs should be packaged separately and no history, clinical signs, post-mortem findings (if appropriate preservative added. As tests for different toxicants

priate) and the results of the environmental assessment, require different samples the need for prior discussion indicating an opinion as to possible toxicants. The relationship between the clinician and the laboratory is paramount. The clinician must then be guided by the laboratory as to what samples to submit, the size of specimens, how they serves as a guide.

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Principal toxicoses

tion, respectively) at cholinergic nerve endings and myoneural junctions. The continued presence of acetylcholine at these sites produces continued nerve stimulation and accounts for the clinical signs observed. The least, cause poisoning in cattle due to the fact that they time of onset of clinical signs varies with the dose and contain known toxic agents and/or have been proven as route of absorption from five minutes to a few hours. toxic by experimental observation. In practice, a few

Clinical signs are similar for both organophosphate and principal toxicoses are commonly observed in a given carbamate toxicoses and are broadly threefold, namely region or country and the majority are rarely, and a few stimulation of the parasympathetic nervous system almost never, encountered.

(muscarinic effects), stimulation of the skeletal muscles Local, regional and geographic factors greatly influence the importance of the various toxicoses in different regions and countries, so much so, particularly with (nicotinic effects) and central nervous effects. The muscarinic effects in general precede the nicotinic. The first effects are excessive, followed by profuse salivation. toxic plants, that attempting to define the principal This is accompanied by nasal discharge, cough, dyspnoea, colic, diarrhoea, excessive lacrimation, frequent certain types of toxicant are encountered much more urination and miosis. These are then joined by the nicotinic signs, muscle fasciculations, stiffness, adoption of a

generally on the position in the UK, are accorded 'saw-horse posture', which then progresses to muscle detailed treatment in the text, whereas for those paralysis. Cattle usually show marked central depression although rarely they may show central nervous principal facts is given in Table 54.3.

excitation. As the condition progresses the muscarinic signs become very pronounced with profuse salivation,

Organophosphate and

severe colic, sweating and dyspnoea progressing to

carbamate poisoning

collapse and death. Death is the result of hypoxia due to severe bronchoconstriction with respiratory hyper-

The organophosphate compounds are widely-used secretion and irregular slowing of the heart.

agrochemicals in agriculture, horticulture and livestock production, and accidental overdosage or overexposure can lead to toxicosis.

Diagnosis

The history and characteristic clinical signs of parasymp-

Source of toxicant

pathetic stimulation are suggestive of poisoning. This
The principal uses of organophosphates are as insecti-
will be reinforced if there is circumstantial evidence of
cides and acaricides, and preparations are made for
exposure to organophosphates or carbamate com-
animal treatment (dusting powders, sprays, washes, dips,
pounds. Particular attention should be paid to the
pour-on, etc.), sprays for application to plants and
possibility of inadvertent therapeutic overdosage, for
granules for application to soil. Carbamates, which have
example by combinations of oral and topical exposure.
a similar mode of action, are used as molluscicides and
Chemical analysis of blood or tissue for the toxicant is
herbicides.

usually of no value due to the fast breakdown of
Cattle may become poisoned by a variety of methods,
organophosphates; the analysis of contaminated feed
principally due to human error, negligence or igno-
or rumen contents is likely to be more successful.
rance. The compounds can be absorbed orally, dermally

However, the preferred method of confirmatory diagnosis is by the determination of the reduction in cholinesterase activity. This test is often performed, for they may also consume treated seed. Containers may convenience or necessity, on whole blood, but examine used for feed or water without cleaning. Sprays for nation of brain tissue is better. The test is a specialized pest control in crops may directly contaminate animals laboratory procedure and the testing laboratory or pollute feed or water courses. Inadvertent over-should preferably be consulted before the submission dosage can occur with insecticidal sprays, dips or topical of samples.

pour-on preparations or by a combination of topical and oral therapy or by rapid retreatment.

Principal differential diagnoses

Signs

The respiratory signs may resemble fog fever (p. 866)

The organophosphates and carbamates block the action

in which the muscle fasciculations are not, however, of cholinesterases (by phosphorylation or carboxyla- usually observed. The syndrome is similar to acute urea

Major Poisonings • 941

Table 54.3

Summary of the less commonly observed toxicoses of cattle.

Poison

Usual source(s)

Clinical signs

Management guidelines

Aflatoxin

Feeds containing groundnut,

Reduced feed intake, poor weight

*No specific treatment. Remove all
cottonseed or maize*

gain (or decreased milk yield),

*suspect feed, submit samples of
contaminated with the toxigenic*

inappetence, rough hair coat,

feed for analysis (permitted

*fungi *Aspergillus flavus* and *A.**

reduced resistance to infectious levels of dietary aflatoxin parasiticus. Growth of toxigenic disease. Less commonly acute controlled).

fungi is only possible when severe signs may be observed moisture content of stored grain especially in calves less than 6 exceeds 15%, feed levels should months, including nervous signs, not exceed 50 ppb.

circling, blindness, convulsions, death.

Arsenicals

Accidental contamination of feed

Often sudden death. Acute –

Rarely successful. Adsorbents

with inorganic arsenicals or

severe colic, salivation, teeth

orally, sulphur compounds, e.g.

organic (herbicides, pesticides).

grinding, weakness,

dimercaprol (4 mg/kg im) or

Formerly in dips and weedkillers,

incoordination, rapid collapse

sodium thiosulphate 15–30 g in

industrial chemicals,

and death. Subacute – similar

100–200 ml water i.v. and up to

horticultural sprays.

with ruminal stasis, diarrhoea,

60 g orally (adult).

severe thirst, dehydration,

collapse and death.

Atropine-containing

Deadly nightshade (Atropa

Those of atropinization, dilation of

Uncommon, often little can be

plants

belladonna), black or stinking

pupils, dryness of mouth

done; pilocarpine can be given by
henbane (*Hyoscyamus niger*),
progressing to excitement,
injection but is not widely
thorn apple (*Datura stramonium*).
incoordination, convulsions and
available and can itself be
death.

dangerous.

Brassica

Conversion of S-methyl cysteine

Peracute – collapse and death;

Stop feeding kale, blood

sulphoxide in the plants to

acute – haemaglobinuria,

transfusion, vitamin injections,

dimethyl disulphide. Kale

pallor, weakness, jaundice,

iron. Feed good quality hay

(*Brassica deracea*) fed as fodder

tachycardia, diarrhoea, low

before and as well as kale.

crop, rape (B. napus) and

haematocrit. Heinz–Ehrlich

cultivated cabbages. Usually

bodies in RBCs. Can be fatal.

need to eat kale as sole fodder for

Within a group usually a high

about three weeks.

prevalence of subclinical

anaemia.

Cyanide

Cyanide release by hydrolysis from

Dyspnoea, bright red mucosae,

Inject sodium nitrite 16 mg/kg iv

cyanogenetic glycosides in plants

recumbency, convulsions,

followed by sodium thiosulphate

including cherry laurel (Prunus

opisthotonus, rapid death. Often

(40 mg/kg iv). Repeat only

laurocerasus), linseed cake,

*less acute; depression,
thiosulphate, 30 g doses of
sudan and sorghum grasses,
staggering, muscle tremor,
sodium thiosulphate orally.
couch grass, white clover.
dyspnoea, death within 2 h,
fresh blood is bright red.*

Ergot

*Ergots (sclerotia of Claviceps
Acute (convulsive) rare –
Remove from feed source, avoid
purpurea) are found on rye and
depression, staggering,
heavily ergotized pastures or
also on other cereal grains and
blindness, convulsions. Chronic
uninspected stored grain
rye grasses particularly in warm
(gangrenous) – lameness,
following warm wet summers.*

wet seasons.

abnormal gait, extremities

swollen, reddened, gangrenous,

particularly tail, ear tips, distal

limbs.

Formaldehyde

Formalin used for agricultural

Mild – salivation, inflammation of

Do not leave cattle unattended

(formalin)

purposes, e.g. foot baths.

buccal mucosae. Severe –

near source of diluted formalin.

Usually associated with a lack of

dullness abdominal pain, weak

Symptomatic therapy.

available drinking water.

pulse, coma, death.

Furazolidone

Accidental overdosage following

Acute – hyperaesthesia,

Stop administration of

(see p. 260)

therapeutic or prophylactic use,

convulsions, death. Chronic –

furazolidone, sedatives to control

improper mixing in milk

haemorrhages, necrotic lesions in

hyperaesthesia.

substitute.

mouth, dysentery.

Hemlock; spotted

Umbelliferous plant common in

Dilation of pupils, weakness,

Avoid contact with plant and

hemlock, Conium

wasteland and neglected pasture,

stagging, muscle tremor, death

overgrazing, especially in early

maculatum

characterized by permanganate-

from respiratory failure.

part of the grazing season.

coloured spots on stems and

mousey odour when crushed.

May be eaten when grazing is

sparse; palatibility decreases as

plant gets woody in late summer.

contd.

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Table 54.3

Continued

Poison

Usual source(s)

Clinical signs

Management guidelines

Hemlock; water

Plant grows in wet ditches and on

Dilated pupils, frothing at the

None, awareness of the potential

hemlock, cowbane,

edges of ponds and swamps.

mouth, abdominal pain, violent

danger.

Cicuta virosa

Contains the very toxic

convulsions, rapid death.

cicutotoxin, the roots being the

most dangerous.

Mercury

Organic mercury compounds from

May not appear for three to four

Poisoned animals can be treated

grain dressed with fungicide, no

weeks after feeding dressed

with sodium thiosulphate (20 mg/

longer common due to control of

grain; inappetence, blindness,

kg i.v. and orally) or dimercaprol

seed dressings.

staggering, weakness, nervous

(BAL) 2.5–5 mg/kg iv 4×

signs.

daily.

Metaldehyde

Molluscicide for slug/snail control.

Incoordination, hyperaesthesia,

Sedation or anaesthesia to control

salivation, tremor, dyspnoea,

hyperaesthesia and convulsions.

fever, ataxia, convulsions,

Consider rumenotomy.

opisthotonus, cyanosis, death.

Molybdenum

High soil levels on teart pastures,

Diarrhoea, often green or black

Test group for low blood copper,

exacerbated by low copper and

with offensive odour,

careful administration of copper

high sulphate levels. Induces a

depigmentation, poor condition,

supplements or injections. A

relative copper deficiency; high

stiff gait in young, anaemia,

*perennial problem on known
dietary molybdenum and low
osteoporosis, fractures, poor
pastures and prophylactic
available copper are interlinked.
milk yield.*

measures should be introduced.

*Ionophores (monensin,
Accidental overdosage or incorrect
Anorexia, depression, diarrhoea,
History of recent access to
salinomycin,
mixing in cattle diets.*

*ataxia, recumbency, dyspnoea,
changed feed useful in
lasalocid)*

*can be fatal. Signs develop in up
presumptive diagnosis. Feed
to two days or longer with lower
assays, remove ionophore from
toxic doses.*

*feed of affected group, ensure
correct levels in feed.*

Onion, wild garlic,

Large quantities of unwanted

Haemolytic anaemia,

Remove from source, feed well,

Allium sp.

onions fed to stock, access to wild

haemoglobinuria, pallor,

multivitamin and iron by

garlic in woodlands.

possibly jaundice. Can be fatal.

injection.

Organochlorine

Accidental access or overexposure

Acute and chronic syndromes

Give adsorbents orally and/or

compounds

to insecticides for animal or

occur – abdominal problems and

wash skin contamination with soap

agricultural use.

typically salivation,

and water. Control excitation with

incoordination, muscle tremor,

pentobarbitone or sedatives.

clonic spasms, convulsions,

Exposure of food-producing

possibly aimless or frenzied

animals to these chemicals may

movements, clonic–tonic

result in high and persistent

convulsions, collapse, death.

tissue residues, particularly in fat.

Oxalate-containing

Unwilted leaves of sugar beet,

Rapid ingestion of soluble oxalates

Treatment of clinical cases with

plants

fodder beet, mangels, also fat

induces hypocalcaemia, paresis,

calcium borogluconate as in milk

hen (*Chenopodium album*),
muscle tremor, recumbency,
fever. The response is not as
docks and sorrells (*Rumex sp.*).
coma, death. Ill-thrift due to
certain and is often unsuccessful.
Oxalate is detoxified by ruminal
chronic renal damage.

Affected forage should be
microflora and hence previous
introduced gradually, is best fed
sublethal exposure will increase
wilted, and not as the sole feed
tolerance to oxalate. Large
source.

quantities of unwilted leaves
need to be consumed over a short
period.

Plants causing

In addition to those described

Generally salivation, teeth

Uncommon, symptomatic
gastrointestinal signs
elsewhere there are a large
grinding, abdominal pain, severe
treatment of colic and diarrhoea,
number of hedgerow and wild
diarrhoea, may be accompanied
remove from access to suspect
plants which will induce
by nervous signs. Fatal in some
plants.

salivation, colic and diarrhoea,
cases.

these include: cuckoo pint (*Arum*
maculatum), black bryony
(*Tamus communis*), autumn
crocus (*Colchium autumnale*),
monkshood (*Aconitum napellus*),
hellebores (*Helleborus sp.*),
buttercup (*Ranunculus sp.*),
spindle tree (*Euonymus*

europaeus), white bryony

(Bryonia dioica), dog's mercury

(Mercurialis perennis), box

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Table 54.3

Continued

Poison

Usual source(s)

Clinical signs

Management guidelines

(Buxus sempervirens), greater

celandine (Chelidonium major),

charlock (Sinapis arvensis).

Plants causing

St John's wort (Hypericum

Photosensitization, erythema,

House animals out of direct

photosensitization

perforatum) is a common plant in

swelling, necrosis of skin in white

sunlight, debride raw areas,

(see p. 884)

hedgerows and rough grazing and areas only, pruritus, oedema, protect against infection and is the most frequently implicated later sloughing and self-inflicted blowfly strike.

in primary photosensitization. It injury.

contains the photodynamic hypericin which is retained when dried. Buckwheat (*Fagopyrum esculentum*) is also photodynamic. Several plants including the ragworts (*Senecio* sp.) and bog asphodel (*Narthecium ossifragium*) cause secondary photosensitization following liver damage.

Rhododendron

Rhododendron ponticum grows as a

*Salivation, staggering gait,
Symptomatic treatment,
cultivated shrub but also extensively
abdominal pain, collapse, death
purgatives, ensure adequate
in the wild on acid soils where it
in a few days. Projectile
fencing of stock.*

*can form large thickets. Poisoning
vomiting, green froth around*

Rumenotomy?

*usually occurs when hungry cattle
mouth.*

*break out into woodland or gardens
or from hedge clippings dumped in
fields.*

Selenium

Toxic levels of selenium may

Acute (rare) – blindness,

Test forage for selenium levels

(see p. 305)

*accumulate in certain plants
depression, circling, head-
and withdraw suspect feed
including certain forage plants
pressing, colic, paralysis, death.*

*(>5 ppm is suspect). Levels in
and grasses growing on high*

Chronic – loss of condition,

blood >2 ppm indicate

selenium soils. Increased levels

lameness, emaciation, hair loss

excessive exposure.

may occur accidentally in feeds; the

from base of tail, hoof damage.

diet should contain under 5 ppm.

Solanine-containing

Plants of the genus Solanum. Green

Salivation, dyspnoea, diarrhoea,

Symptomatic treatment, green

plants

tubers and leaves of the potato

depression, prostration, coma.

potatoes should not be fed to

(S. tuberosum). The woody

Can be fatal.

livestock but boiling and feeding

nightshade (S. dulcamara) is

at less than 25% of diet reduces

common in hedgerows; this plant

risk.

together with the black

nightshade (S. nigrum) are

common in neglected pasture.

All parts of the nightshades are

toxic, the berries being the most

dangerous.

Tremorgenic

Certain fungi of the genera

‘Ryegrass staggers’, usually not

Recovery is usually uneventful

mycotoxins

Penicillium and Aspergillus

*observed until animals are
when removed from suspect
which grow at the base of the
disturbed, muscular tremors,
pasture and given alternative
grass sward produce tremorgens.
stumbling, swaying; when forced
feed.*

*Toxicity is most likely to occur in
to run develop a high-stepping
hot, dry summers with cattle
gait, staggering, sternal
grazing at the bottom of the grass
recumbency, will then recover if
plant.*

left undisturbed.

Urea

Inadequate mixing or diluting of

Begin within 1 hour, excessive

Drenching with vinegar (4–7 l

dietary urea, accidental access to

salivation, frothing at mouth,
daily) together with cold water
concentrated amounts, feeding to
bellowing, bloat, muscle
(20–40 l). Ensure adequate
unadapted animals, or with high-
fasciculations particularly of the
mixing of urea, continuous access
fibre, low-energy rations.

head, colic, dyspnoea, death.
is preferred; tolerance will build
Overconsumption of urea-
up in cattle accustomed to urea
containing blocks or licks.
feeding, but is short-lived on
withdrawal.

Water dropwort

Root tubers from the plant which
Often sudden death, salivation,
No treatment, awareness of
(*Oenanthe crocata*)

*grows commonly in ditches and
dilated pupils, convulsions,
potential danger to grazing
marshes. Usually exposed tubers
death.*

*animals when ditches are cleared.
on pasture following ditching or
drainage operations.*

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*poisoning (p. 943), and it may clinically resemble nitrate
are seen. Ingestion of large quantities, particularly by
toxicosis (p. 950) but without the cyanotic mucosae. The
calves, tends to lead to an acute syndrome with the
nicotinic effects may resemble hypomagnesaemia
neurological signs predominating. Ingestion of lesser
(pp. 253, 787).*

*amounts results in a subacute pattern. In very acute
cases sudden death may be the presenting sign. Acute
cases are characterized by sudden onset of muscle fas-
Treatment and prevention
ciculations, particularly of the head and neck, frothing*

Atrophine sulphate is the specific antidote to organo- at the mouth, teeth grinding, jaw champing and abnor- phosphate or carbamate toxicosis. It acts by blocking mal movements of the head and eyelids. There is a the effects of acetylcholine at nerve endings and will staggering gait and apparent blindness with pupillary thus only counteract the muscarinic effects. The re- dilation. Colic may be observed. This may progress to commended dose is 0.1 mg/kg bodyweight by slow collapse, tonic/clonic seizures, hyperaesthesia and intravenous injection followed by 0.4 mg/kg given death. In adults, abnormal patterns of behaviour, subcutaneously. The effects are usually observed within including pushing through fences, charging or mania minutes. Treatment may need to be repeated during the can be seen.

first 48 hours depending on clinical response. Cattle Subacute cases are dull and anorexic and are appar- should be removed from the suspected source and ently blind. There may be aimless wandering, there is washed with soap and water if exposed via the skin.

muscular tremor and a staggering gait, tooth grinding, Intestinal adsorbents such as activated charcoal given signs of colic and a ruminal stasis resulting in constipation by drench or stomach tube are also useful. Prevention followed by diarrhoea. The palpebral eye reflex is is dependent on recognition of the wide diversity of absent. Rarely, a more chronic form may be observed uses of organophosphates and carbamates in the environment with poor growth and anaemia.

ronment and the possibility of accidental exposure or overdosage.

Necropsy

Lead poisoning

In very acute cases there may be no gross lesions but (see also p. 906)

the lead-containing material, such as flaked paint, oil or grease, may be found in the reticulum and rumen. In

Poisoning by lead is one of the most common intoxications of cattle, which are more susceptible to this testinal tract are frequently observed including aboma-

toxicant than other farm species.

sitis and enteritis. The liver and kidneys may be abnormally pale and show some degeneration. There

Sources of toxicant

are frequently epicardial and sometimes endocardial haemorrhages. The brain may be oedematous. Histo-

There are many potential sources of lead in the environment and accidental access by cattle to a lead-containing product is the usual predisposing cause. Histological changes may be observed in the brain depending on the length of exposure. The hepatic and renal cells may also show degeneration.

which, combined with their natural inquisitiveness and a tendency to lick foreign objects, means that a toxic dose is soon ingested. Common sources of lead include

Diagnosis

old flaking paint, batteries, discarded engine sump oil, grease, putty, plumber's materials, linoleum and mine

Often the history and circumstantial evidence (suspected lead-containing material in the environment)

also contain significant amounts of lead from leaded petrol. Lead poisoning is more frequently observed in calves than adults. Poisoning can result from a single ingestion of a toxic amount or from a continued ingestion of lead from the environment with accumulation in the body tissue.

Diagnosis in the live animal rests on a measurement of blood lead levels. A heparinized blood sample should be submitted, and levels in excess of 0.4 ppm are considered

Signs

diagnostic. From the dead animal, kidney, liver and stomach contents should be submitted. Measurement of lead from the kidney is most reliable and levels in excess of 0.4 ppm are considered diagnostic. The onset of disease and, to some extent, the clinical picture that ensues depends on the dose of lead ingested. Both neurological and gastrointestinal signs

20 ppm are diagnostic.

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Principal differential diagnoses

important single species the common or tansy ragwort

(S. jacobaea). In temperate regions the ragwort is wide- In the absence of compelling circumstantial evidence

spread in wasteland and neglected pastures and poses

of poisoning, lead intoxication can closely resemble

a serious potential toxic threat to livestock. Plants of

several other disorders of calves characterized by neu-

the genera Crotalaria, Heliotropium and Amsinckia rological signs. These include infections, e.g. listeriosis

also contain the toxic alkaloids.

(p. 904), Haemophilus somnus infection, brain abscess, rabies, coccidiosis (p. 282), together with hypomagnesaemia, tetanus, cerebrocortical necrosis (pp. 261, 903),

Circumstances of poisoning

vitamin A deficiency, other poisoning (e.g. mercury) (p.

Common ragwort is not attractive to grazing animals

942) and in older animals nervous acetonaemia (p. 794)

and cattle will usually avoid eating the fresh vegetative

and bovine spongiform encephalopathy (BSE) (p. 909).

plant. However, this is not always the case and should

never be relied upon. The plant is relatively late in

Treatment and prevention

emerging through the sward in the spring and, particu-

larly following widespread seeding from the previous

Treatment is threefold: (i) supportive and symptomatic,

year, small, young plants can become finely distributed

(ii) oral salts to precipitate soluble lead and (iii) intra-

within the grasses. This is then non-selectively grazed.

venous administration of lead-chelating agents, which

In dry conditions and where grass growth is poor, cattle

can increase by up to 50 times the rate of lead

may also be attracted to ragwort. The unattractiveness

excretion.

of the plant is lost, but its toxicity largely retained, when

Convulsions and nervous signs can be controlled

it is dried in hay or ensiled and this represents the single

by the use of tranquillizers and sedatives including

most important cause of intoxication. Cattle will readily

acepromazine and xylazine. Magnesium sulphate, egg

consume hay or silage containing ragwort.

whites and strong tea will precipitate any soluble lead

remaining in the gut as insoluble salts of lead. Calcium disodium edetate should be given by slow intravenous

Pathogenesis

injection at a dose of 110–220 mg/kg body weight. This

The pyrrolizidine alkaloids are hepatotoxic, damaging dose should preferably be divided between two or three

the hepatocytes. The speed at which this cell damage

separate injections per day. Treatment should be given

occurs and the consequences thereof are dependent on

for two to three days, withheld for two days and then

the dose of alkaloid and the duration of consumption.

repeated, repeating this pattern again if necessary.

The liver has a large functional reserve and thus can

Good nursing care and oral fluid replacement is

withstand the functional loss of many hepatocytes

required to combat dehydration. Clinical improvement

before gross dysfunction occurs. An animal would

may take two or three days to be apparent and blind-

therefore only rarely ever ingest sufficient quantities of

ness can persist for up to three weeks. Prevention of

alkaloids over a sufficiently short period to result in

poisoning by lead is largely a matter of education of acute poisoning. Much more commonly cattle will those responsible for the care of livestock. A knowledge ingest smaller amounts of the toxicant over a period of of the common lead-containing commodities and the weeks or even months resulting in a more gradual loss need to be vigilant about accidental access should be of hepatocyte function, it only being when the func-stressed.

tional reserve of the organ has been exceeded that gross hepatic dysfunction and hence clinical disease results.

Ragwort poisoning

Signs

Poisoning of cattle by the pyrrolizidine alkaloids found

The clinical picture can vary: although the principal

in certain genera of plants is a common problem in

cause is a subacute to chronic intoxication, acute clini-

many parts of the world and can result in severe losses.

cal syndromes may be observed, although more usually

a subacute pattern is seen in cattle. Affected cattle will

lose weight and usually develop a mild to moderate

Sources of toxicant

jaundice, and may show photosensitization. Diarrhoea, The principal genus of plants containing pyrrolizidine colic and straining characteristically occur. Subcutaneous oedema and ascites may be present due to 1200 species. The quantity and type of alkaloid varies hypoalbuminaemia. Affected cattle are usually dull between species, for example the widespread groundsel and depressed. They may show signs of hepatic (Senecio vulgaris) is much less harmful than the most encephalopathy, resulting from the effects of raised

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blood ammonia levels on the brain due to the inability be immediately switched to a non-contaminated food of the damaged liver adequately to remove urea arrival supply. Prevention relies upon control of the plant and ing via the portal circulation. Signs of encephalopathy an understanding of the syndrome. Hand pulling and include an unawareness of surroundings, staggering burning of plants before seeding and the use of herbi-

gait, aimless wandering, circling and apparent blindness.

cide sprays is to be recommended on affected pastures.

Head pressing and aggressive syndromes are rare in

Sheep are less susceptible than cattle and can often

cattle. Death usually occurs a few days after the com-

graze affected pasture or eat hay if not too heavily con-

mencement of such clinical signs.

taminated. Hay or silage should never be prepared

from ragwort-infested grassland.

Necropsy

The carcass may be jaundiced and in poor condition,

Bracken poisoning

and ascites may be present. The liver is characteristi-

cally shrunken, fibrosed, slate-grey or mottled. The his-

The bracken fern (Pteridium) is widely distributed in

tological picture of the liver in pyrrolizidine alkaloid

the UK, the USA and in many other temperate hilly

toxicosis is characteristic and highly suggestive of the

and forested areas. It can relatively quickly become

condition but not pathognomonic. The lesions are a

dominant in a grassland pasture. Where cattle have

fine pericellular cirrhosis, bile duct proliferation and continued access to bracken-contaminated grazing, hepatocytomegaly. toxicosis can result.

Diagnosis

Source of toxicant

The history and circumstantial evidence are extremely important. It is vital to understand the chronic nature Cattle are often reluctant to eat bracken and will of the intoxication; in most cases animals will often have usually only do so when grassland grazing is sparse. eaten the plants for several weeks or months prior to Cattle need to graze bracken as a significant constituent the development of clinical signs. Bearing this in mind, of the diet for several weeks or months before clinical examination of hay, silage and pasture for the plants is disease may become apparent. The rhizomes, exposed vital, but their absence does not necessarily eliminate after ploughing of bracken-infested pasture, are more pyrrolizidine alkaloid toxicosis unless a history of an attractive and dangerous as are newly sprouted young

unchanged, uncontaminated diet can be positively fronds. Some of the toxicity remains when bracken established. Histological examination of the liver is the becomes dried in hay. Bracken contains a thiaminase method of choice in establishing a diagnosis and, in the but, unlike the situation in herbivorous non-ruminants, live animal, liver biopsy is the most useful diagnostic cattle are largely unaffected by it. Bracken also contains a variety of other toxic chemicals including a elevation of g-glutamyl transferase (GGT), and in some cyanogenetic glycoside (which is only usually present in cases an elevation in aspartate amino-transferase harmless amounts), toxins that depress bone marrow (AST). Chemical analysis for the alkaloids is not often and a carcinogen (ptaquiloside). These last two can of value.

cause disease in cattle.

Principal differential diagnoses

Signs

Particularly where hepatic encephalopathy is marked,

Acute poisoning: Acute or subacute toxicosis is the ragwort poisoning can resemble rabies, BSE (p. 909), result of bone marrow depression producing leucopenia and thrombocytopenia and occurs following consumption of comparatively large amounts of bracken. jaundice and diarrhoea are the dominant clinical signs, Clinical signs may develop for up to several weeks intestinal parasitism (p. 267), fascioliosis (p. 276), hep- after the exposure to bracken has ended. Signs may atitis and biliary obstruction should be considered. be sudden in onset and include anorexia, depression and dysentery, and there may be pyrexia; various signs of capillary fragility and haemorrhage become

Treatment and prevention

obvious including petechiae on mucosae, bleeding from Once the clinical picture has developed there is no nose, vagina and conjunctiva. Trauma may produce worthwhile treatment for pyrrolizidine alkaloid toxicohaematomata. Heart and respiratory rates are in-

*sis, but clinically normal cattle in the same group should
creased. Progressive weakness ensues and death may*

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*occur in one to five days. In calves there is often pyrexia,
bladder wall and/or carcinomas may be found in the
dysentery, frank haemorrhage and petechiation of
upper alimentary tract.*

*visible mucosae. Death is often the result of heart
failure. There may be laryngeal oedema and marked*

Principal differential diagnoses

*dyspnoea. Due to the leucopenia, a bacteraemia or
other secondary infections are often complications.*

*Acute disease can resemble leptospirosis, kale anaemia,
or babesiosis. Enzootic haematuria should be differen-*

*Enzootic haematuria: Where cattle have consumed
tiated from babesiosis (p. 748) and postparturient
comparatively small quantities of bracken over pro-
haemoglobinuria (p. 792). The syndromes caused by
longed periods, neoplastic changes can develop in the
upper alimentary squamous cell carcinoma can resem-
transitional cell epithelium of the bladder. Various*

ble Johne's disease (p. 857), copper deficiency (p. 298)

tumour types may occur including haemangiomas,

and mucosal disease (p. 853).

transitional cell carcinomas, adenocarcinomas and haemangiosarcomas. The resulting clinical picture is

Treatment and prevention

termed enzootic haematuria. The condition varies from a mild, persistent haematuria as the only clinical sign to

In acute cases the clinical outcome is probably more

severe cases in which there is pallor of the mucosae,

influenced by the degree of bone marrow damage than

dysuria and tenesmus. The urine in severe cases has

by treatment. Broad-spectrum antibiotics should be

visible blood clots in it (see p. 154).

administered to counteract bacteraemia and secondary

infections. Blood transfusions can be considered. The

Upper alimentary squamous cell carcinoma (see also

use of bone marrow stimulants including dl-batyl

p. 828): A third less common clinical syndrome result-

alcohol has been described. There is no successful ther-

ing from prolonged exposure to bracken is upper

apeutic management of urinary bladder or alimentary
alimentary squamous cell carcinoma, which may
neoplasia. Despite its widespread abundance in certain
accompany changes or tumour formation in the bladder
areas the bracken fern should always be viewed as a
wall. The disease has strong regional incidence, e.g. in
potentially toxic plant to cattle. Access to bracken-
western Scotland and Wales, often in suckler cows. Four
infested pasture should be always limited, especially if
clinical syndromes are recognized depending on the site
grass growth is poor. In particular, cattle should never be
of tumour formation. Oropharyngeal tumours produce
allowed access to recently ploughed land with bracken
loss of condition, drooling of saliva, coughing or
present and where the rhizomes are exposed, especially
snoring, halitosis. The nasal discharge may contain
if they have started to reshoot. Limited areas of bracken
ingesta, submandibular lymphadenopathy and diar-
infestation can be fenced off, and burning, ploughing and
rhoea. Oesophageal tumours produce diarrhoea, hali-
reseeded or herbicide control are all measures that can

*toxis, coughing, drooling and palpable masses in the
reduce the level of bracken contamination.*

*oesophagus. Ruminal tympany can result from tumours
in the lower oesophagus; there is initially intermittent
bloat with loss of condition, then diarrhoea and resist-*

Yew poisoning

*ance to the passage of a stomach tube. Tumours in the
dorsal rumen produce loss of condition, diarrhoea, dis-*

*Cattle that gain access to yew trees (genus Taxus) are tended abdomen and
bloat. Oropharyngeal papillomas*

frequently fatally poisoned.

may often accompany tumours at any site.

Sources of toxicant

Diagnosis

*The various species of yew (Taxus baccata, English yew; This is based on the
clinical signs and an association*

*T. lineata, Irish yew; T. cuspidata, Japanese yew) are with bracken feeding or of
prolonged exposure to*

common ornamental trees or hedging plants particu-

bracken. In acute or subacute cases, bone marrow

larly of old established gardens. The leaves and woody

depression results in thrombocytopenia and leucop-

twigs of the yew contain various toxic alkaloids collectively termed taxines. The taxines are also present in the seeds but not the fleshy red outer part of the fruit.

Necropsy

Circumstances of poisoning

In acute or subacute cases there are multiple internal haemorrhages of varying size and petechiation. The Yew is well known as a toxic plant among the agricultural and rural community, and poisoning is thus usually

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the result of neglect, ignorance or (rarely) malice. Cattle will often consume the fresh plant if they are allowed access and will also eat fresh hedge clippings or trimmed branches dumped into their field. The taxines are severe cardiac depressants.

copper is retained and accumulates within the liver. It has also been seen with improper use of copper supplements. Some problems have arisen from industrial

Signs

contamination, and from copper addition to the

If moderate amounts of yew have been ingested, pasture. There are some areas in the world with copper-sudden death is often the presenting sign. If seen alive, rich soil. While the problem of copper poisoning is cattle may show dyspnoea, abdominal pain, muscle common in sheep, it is much less seen in cattle. Prob-tremor, weakness and collapse. The pulse later becomes lems have increased in North America, Britain and slower before finally disappearing with the heart stop-other countries in recent years.

ping in diastolic arrest. Recovery is rare and is dependent on ingesting only relatively small amounts of the

Circumstances of poisoning

plant.

Cattle are less susceptible to copper toxicity than sheep and are able to ingest higher levels than the latter

Necropsy

without any deleterious effect. Animals with intercurrent liver disease may be more susceptible. Frequently, there are no significant gross findings except the presence of yew leaves and twigs among the ruminal contents or sometimes still in the mouth. The characteristic leaves of the yew when separated by careful washing from the general ruminal contents are thus released into the blood and causes an acute intravascularly suggestive of poisoning.

lar haemolytic crisis. Problems of copper intoxication were reported to be increasing in the first years of the twenty-first century (Bidewell et al. , 2000). In their findings grossly increased copper intakes were not always History or circumstantial evidence of access to yew, and found. However, points to consider include the duration of feeding a copper supplement and the type of

usually establish a diagnosis.

supplement (it is claimed that the copper in many amino acid chelated minerals is many more times available than that in inorganic compounds). Jerseys have

Principal differential diagnoses

been said to be more susceptible, possibly because of Yew poisoning should be distinguished from other genetic susceptibility or perhaps related to a lower causes of sudden death, for example lightning strike bodyweight.

(p. 930), anthrax (p. 717), blackleg (p. 723), etc.

Signs

Treatment and prevention

The problem can be either acute, which is rare, or There is no specific therapy. Symptomatic treatment chronic. The latter is more common but often also can be offered to clinically affected animals but the like-appears as a sudden presentation.

likelihood of recovery or death is principally influenced by the amount of the plant already ingested. Rumenotomy

Acute: The animal shows pronounced depression with

is possible immediately after observing cattle consum-
colic, rapid collapse and death. In some cases there is a
ing yew. Generally, yew is well known as a toxic plant
green diarrhoea when the animal has had access to the
but the necessity of preventing cattle from gaining
excess copper within the diet.

access to the plant should be constantly stressed.

Chronic: The average length of illness before death is **Copper poisoning**
one to three days, although individual animals may
(see p. 300)

show signs for up to three weeks. There is an acute
haemolytic crisis following a prolonged access to excess

Sources of toxicant

copper. The signs again often rapidly develop with
The normal problem is an extensive intake of copper,
anorexia, depression, rumen stasis, cold extremities,
usually from the diet and particularly where there is an
haemolytic jaundice, anaemia with pale mucous

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membranes, cyanosis of the teats, haemoglobinuria,
levels before any supplementation is given. The normal

methaemoglobinaemia and death. Some animals have minimum dietary copper level for adult dairy cattle is shown abortion, ataxia, photosensitization and other 12 mg/kg dietary DM. This level often has to be raised, signs. Recumbency is common before death.

especially where there are inhibitory factors present such as molybdenum (see p. 298). However, in many countries there are restrictions on the amount of copper

Necropsy

supplementation. Thus in Britain the Medicated Feed-

In acute cases there is a severe gastroenteritis with ingesta. Regulations allow dairy cattle to receive up to ulceration and erosion. The abomasum is particularly 35 mg/kg dietary DM (about 800 mg copper/head per affected. The chronic signs are those of a haemolytic day) without restriction. However, higher supplementation levels can be licensed up to a maximum of there is hepatomegaly. In sheep there is usually dark 2000 mg/head per day, but with certain restrictions. discoloration of the kidneys, but this is not so common

in cattle. Histologically there is a necrotizing hepatopathy and nephropathy.

Fluoride poisoning (fluorosis)

At normal levels the ingestion of fluorides causes

Diagnosis

no harm or is even beneficial to cattle. However, at

The signs and post-mortem picture are helpful. Usually higher levels a chronic (or rarely acute) intoxication there will be a known source of copper which has been results.

ingested, although this may not always be the case. The liver will contain more than 8000 mmol/kg dry matter

Sources of toxicant

(DM) (although often levels are between 10 000 and 20 000 mmol/kg DM). Plasma copper levels are usually

Cattle normally gain access to excess fluoride from very high (normally over 50 mmol/l). Blood biochemistry usually shows elevated liver enzymes such as aspartate aminotransferase, glutamate dehydrogenase

brick manufacture, aluminium smelters, steel works or and gamma glutamyl transferase, and this assists in phosphate production. More recently, other sources of diagnosis.

excess fluoride have been recognized including geothermal spring water (when used either as a source of drinking water or for pasture irrigation) and high fluo-

Principal differential diagnoses

ride-bearing soils. Inorganic rock phosphate is often

Nitrate poisoning (see p. 950) may need to be elimi-

highly toxic and requires defluorination prior to use as

nated. There are also many causes of haemolytic

a phosphorus supplement.

anaemia including babesiosis (p. 948), kale poisoning

(p. 941) and post-parturient haemoglobinuria (p. 792).

Circumstances of poisoning

In most cases the history should be helpful. However,

the use of blood copper examination should provide

Rarely will cattle consume sufficient fluoride to result

good evidence of the cause if due to copper toxicity.

in acute intoxication, but this may result following

heavy pasture contamination. More commonly, the grazing of fluoride-contaminated pasture over months

Treatment and prevention

or years results in an insidious chronic disease.

First remove the animals from the copper source, if known. While treatment is often of limited value,

Signs

calcium versanate iv at a dose of 70 mg/kg bodyweight (see p. 945 for details) has been used in calves. Oral

In the acute disease there is anorexia, depression, restlessness, dyspnoea, excessive salivation and muscle tremor leading to convulsions, collapse and death.

100 to 400 mg daily can also be of use. In sheep, iv injection of ammonium tetrathiomolybdate at a dose of

Chronic fluorosis: The developing teeth of young cattle are sensitive to excessive fluoride and dental lesions

of two to three days has been successfully used.

are common in animals exposed before the full adult

While not licensed, it might also be of benefit to cattle.

dentition has developed. The dental abnormalities

If cattle are suspected of having a copper deficiency, observed are: increased attrition and liability to fracture, blood samples should be taken to estimate the copper level, mottling of enamel with white areas, abnormal

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pigmentation, and yellow or brown discoloration. Teeth ingestion of high or suspect levels of fluoride is unavoidable may be poorly developed and mineralized.

able, aluminium sulphate or calcium carbonate added

Bones are also affected by excessive fluoride intake.

to the diet may be beneficial in reducing fluoride

Although the bones of young cattle are more susceptible, deposition in teeth and bone.

ble, the bones of cattle of any age can be affected by fluoride-induced changes. These are principally periaricular calcification and the formation of exostoses

Nitrate/nitrite poisoning

resulting in an intermittent lameness and stiffness of gait, particularly of the hind legs. Palpable exostoses

Under certain circumstances various types of grazing

appear on the metacarpals and metatarsals, ribs and
and forage crops may accumulate toxic levels of nitrate
mandible and these are painful on pressure. In severe
within their foliage. In addition, the widespread use of
cases fractures of the third phalanx are not uncommon.
inorganic nitrate fertilizers poses a potential threat of
Affected cattle are unthrifty and have a dull staring
nitrate poisoning in grazing cattle.

coat.

Source of toxicant

Necropsy

The Brassica plants, green cereals (wheat, barley, rye Dental lesions are found,
as described above. Bones

and maize) as well as the Sorghum and Sudan grasses,
have a roughened surface and are chalky white, and
docks, nightshades and sweet clover may accumulate
new bone formation is evident.

excess levels of nitrate in their tissues. Nitrate accumu-
lation may occur after excessive use of inorganic nitrate
fertilizers or following heavy rain after a period of

Diagnosis

drought, which leaches accumulated nitrate into the water table. Water run-off from heavily fertilized fields together with circumstantial evidence of access to or excess application of nitrogenous fertilizers are the suspect sources of fluoride, are usually suggestive of commonest dangers in intensively managed pastures fluorosis. Radiographic examination of lower limb and can lead to problems on grassland pasture or even bones may demonstrate the presence of exostoses.

hay made shortly after exposure of the sward to high Chemical analysis of urine, blood and bone for fluorine nitrate levels. Cattle thus become poisoned by consumption may be useful. The levels in bone rise with age ing normal amounts of the fresh pasture containing the and elevated levels are indicative of previous exposure abnormal nitrate load.

to fluoride. Fluorides are chiefly excreted via the urine Nitrate ions in the rumen are reduced by the ruminal and do not accumulate in soft tissues.

microflora to nitrite ions, which are subsequently ab-

Attempts should be made to identify the source of sorbed. The nitrite ion oxidizes the iron in haemoglobin (to its ferric state) producing methaemoglobin, which is unable to bind oxygen. A single intake of less than 50 ppm fluoride on a dry matter basis, and drinking water less than 8 mg/l.

As a guide the diet should contain less than 50 ppm fluoride on a dry matter basis, and drinking water less than 8 mg/l. Accumulating the same amount over several days or weeks. A degree of tolerance may also build up in stock receiving sublethal amounts over a prolonged period. High

Principal differential diagnoses

grain diets tend to be somewhat protective against the dental and bone lesions are fairly characteristic of effects of excess nitrate.

fluorosis but in their absence other causes of ill thrift including mineral deficiencies, parasitism and the

Signs

common forms of lameness must be differentiated.

The condition is usually acute, less commonly subacute.

In the acute condition, signs are seen within a few hours

Treatment and prevention

of consuming high levels of nitrate. The clinical picture

There is no specific antidote and dental lesions are

is due to methaemoglobinaemia and the associated

permanent. After removal from the source of excess

anoxia. The visible mucosae become blue and cyanotic,

fluoride the bones of affected cattle will gradually be

the pulse is weak and rapid, there is drooling of saliva

remodelled, with normal bone deposition ensuing, but

and tachypnoea, exercise is resented and, if forced, may

severely affected cattle will never completely recover.

result in severe dyspnoea with mouth breathing. If

Mildly affected cattle should be removed from the

untreated the condition deteriorates, and there is

source and given a good quality balanced diet. Where

weakness, ataxia, prostration, coma and death. The

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rapidity of onset of the clinical signs is dependent on

Oak poisoning

the dose consumed; death can occur in one hour or up

to a day following acute intoxication.

Poisoning by the various oaks of the genus Quercus can be a serious seasonal problem in grazing cattle.

Necropsy

Sources of toxicant

High methaemoglobin levels impart a chocolate-brown discoloration to the blood, which is seen particularly in Cattle may be poisoned at any time when they gain the fresh cadaver. This brown coloration is evident access to quantities of oak leaves or acorns, which throughout the mucosae, viscera and possibly in the contain the toxic tannins. Cattle can consume small urine.

quantities of acorns or oak leaves without ill effects.

Circumstances of poisoning

Diagnosis

In temperate countries the autumn fall of acorns is the The history may be suggestive if a link of known principal source. The acorn crop varies considerably exposure to nitrates can be established.

The

from year to year and hence there is wide variation in clinical picture and particularly the post-mortem prevalence. Storms and windy conditions that cause a findings are also highly suggestive. Methaemoglobin heavy sudden drop of acorns are also predisposing can be detected in blood but is not stable and a factors. Cattle may also be poisoned from browsing on fresh sample needs to be analysed within 4 hours. new leaves and buds or oak seedlings.

Alternatively, a sample of heparanized blood can be preserved, consulting the diagnostic laboratory for the preferred method. Nitrate levels in all sources of

Signs

feed and water should be analysed but tolerance to nitrate may be apparent in some circumstances as Although sudden death may be observed, poisoning described above. Levels in feed in excess of 1 per cent is usually seen as a subacute condition. Affected nitrate on a dry matter basis are highly suggestive of cattle become depressed and anorexic. Early on in the intoxication.

disease constipation occurs, often with marked straining perhaps accompanied by groaning and abdominal pain; small, dry faecal pellets may be observed at this

Principal differential diagnoses

stage. Later on this gives way to a dark coloured or tarry diarrhoea often with blood; straining may be persistent

Circulatory collapse or severe respiratory conditions

and severe. As the condition progresses polyuria and

can result in tissue anoxia and acute cyanosis, but in

subcutaneous oedema may be observed. Cattle then

few other circumstances does methaemoglobinaemia

become progressively weaker and collapse with death

with its characteristic chocolate-brown blood result.

supervening in four to seven days from the onset of

Intoxication with chlorates will also result in

clinical signs.

methaemoglobinaemia.

Pathogenesis

Treatment and prevention

The tannins are nephrotoxic and damage the renal

The animal should be immediately removed from the

tubules but the precise mechanism of their action is suspected source of nitrate. The specific antidote is unclear. There is a necrosis of the renal tubular cells methylene blue, which restores the iron in haemoglobin and the consequent renal dysfunction leads initially to to its ferrous state thus again allowing oxygenation of oliguria or anuria, later progressing in the subacute to the blood and so reversing the tissue anoxia. Methy- chronic stages of intoxication to polyuria with the pro- lene blue should be given by intravenous injection at up duction of a very dilute urine.

to 4 mg/kg body weight in a 2 per cent solution. It can be repeated if necessary. Defining the nitrate source

Necropsy

and reducing exposure are the key elements in control.

Ensiling will usually reduce the nitrate content of

Gross signs include those of oedema, particularly forage as will allowing the pasture to age and set seed, hydrothorax, ascites and ventral subcutaneous oedema, since nitrate is found mostly in stems rather than in oedema fluid may contain blood, and there is often

grain.

perirenal oedema together with swollen pale kidneys.

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The contents of the rumen are frequently doughy in

Treatment and prevention

consistency and often contain large numbers of acorns

There is no specific antidote. Cattle may survive poi-

and/or oak leaves, although this is not invariable in the

soning by oak but are frequently left with chronic renal

more chronic cases. There may often be colitis, with

damage, such that they will often perform poorly. The

tarry, bloodstained faecal contents. Histologically, the

only treatment that is likely substantially to affect the

acute renal lesions include hyaline cast formation

outcome of clinical cases is expensive and specialized

and a coagulative necrosis of the tubular epithelium;

involving full-scale renal supportive therapy including

later mononuclear cell infiltration and fibrosis are

measurements and adjustment of plasma electrolytes,

observed.

oral and parenteral rehydration and the re-establish-

ment of diuresis. Rumenotomy may be indicated.

Diagnosis

Anticipation of potential outbreaks by the recognition

The history and circumstantial evidence of poisoning

of the seasonal nature of the disease in inspection of

together with the clinical signs usually strongly suggest

pastures (particularly if the meteorological conditions

a diagnosis of oak poisoning. This is usually confirmed

would produce a sudden heavy acorn drop) followed by

by finding large amounts of acorns and/or oak leaves in

removing cattle from sources of contact is the key to

the rumen at post-mortem examination together with

successful prevention.

the characteristic gross and histological changes. In live

animals there will be an elevation of serum urea and

creatinine, and glucose and protein will appear in the

Reference and further reading

urine.

*Andrews, A.H. & Humphreys, D.J. (1992) Poisoning in Veteri-Principal
differential diagnoses*

nary Practice, pp. 1–114. National Office of Animal Health, Enfield.

The main differential diagnoses include clostridial

Bidewell, C.A., David, G.P. & Livesey, C.T. (2000) Copper

infections (Chapter 44) and other toxicoses.

toxicity in cattle. Veterinary Record, 147, 399–400.

Adult Cattle

Welfare

Chapter 55

Welfare

D.M. Broom

Public perceptions of the dairy and beef industries

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which appear well founded can be very damaging to

The concept of animal welfare

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producers, processors and retailers.

Welfare problem areas

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Public concern about animal welfare manifests itself

Ill treatment and neglect

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in actual product purchasing and in pressure applied to

Housing and management

957

retailers and to legislators. Major supermarket and

General points

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cooked food chains can be influenced rapidly by cus-

Calf welfare

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tomers pressure and can cause changes to be brought

Beef cattle welfare

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about in the methods used by suppliers. Retailers may

The welfare of dairy cows

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Farm operations

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impose codes of practice on suppliers and the execution

Handling, transport and slaughter of cattle

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of these codes is checked because the retailers cannot

afford public criticism of what they sell (Broom, 1999a).

In several European countries, certain housing systems

Public perceptions of the dairy and

and farm practices have been changed by many farmers

beef industries

because of the standards required by the purchasing

companies. For example, the use of crates for calves,

Most members of the public who are asked about the

stalls and tethers for sows, and castration of pigs slaugh-

dairy and beef industries think of cattle grazing in fields

tered at 100 kg or less has ceased on many farms.

and cows living for some time whilst a series of calves

The effect of public pressure on legislation is usually

are born and milk is produced. Milk products and beef

slower, but legislation makes for more equal constraints

are considered by the public in relation to their effects

on producers. Legislation is becoming more and more

on human nutrition and health, their effects on the envi-

international although it is clearly important that

ronment and their effects on animal welfare. If pro-

where there is legislation on wholly moral grounds, for

duction is perceived to be bad in relation to any of these

example in order to prevent poor welfare in animals, aspects, sales of the products could be severely affected. there should be restrictions on imports from countries Bovine spongiform encephalopathy (BSE) resulted in whose moral standards are lower and that such a substantial but sometimes brief decline in beef consumption should be authorized by the World Trade Organization. Some people may limit their intakes of

milk products because of a desire to reduce cholesterol intake whilst certain aspects of the dairy industry, such as methane production, may be criticized in relation to

The concept of animal welfare

pollution; however, it is animal welfare rather than these topics which is the subject of this paper. Until Animal welfare has to be defined in such a way that it recently, the welfare of the dairy cow was not often perceived to be poor and it has been only in calf rearing in legislation and in discussion amongst animal users that dairy production systems have been regularly criti-

and the public. Welfare is clearly a characteristic of an
cized. However, the dairy industry has been changing.
individual animal and is concerned with the effects of
Evidence of poor welfare in cows is accumulating and
all aspects of its environment on the individual. The
has had influence on public opinion in several countries.
welfare of an animal is its state as regards its attempts
It is important to the dairy industry that welfare
to cope with its environment (Broom, 1986). This state
problems should be addressed before there is any
includes the feelings of the individual, various physio-
widespread public condemnation of breeding and man-
logical and behavioural responses and its health. The
agement practices. Similarly, beef cattle production is
extent of the difficulty which the individual has in trying
not often criticized on welfare grounds. However, a few
to cope with its environment, the extent of any failure
critical newspaper articles or television programmes
to cope and the degree of happiness are all components

Box 55.1

Indicators of animal welfare (after Broom, 2000).

Box 55.2

Possible causes of cattle welfare problems.

Physiological indicators of pleasure

Ill treatment

Behavioural indicators of pleasure

Neglect: calculated, accidental or due to lack of knowledge

Extent to which strongly preferred behaviours can be shown

Inadequacies in design of housing/furniture

Variety of normal behaviours shown or suppressed

Inadequate management system or poor husbandry on the

Extent to which normal physiological processes and

farm

anatomical development are possible

Unnecessary or poorly executed mutilations of the animals

Extent of behavioural aversion shown

Poor conditions and procedures:

Physiological attempts to cope

during transport

Immunosuppression

at market

Disease prevalence

at slaughterhouse

Behavioural attempts to cope

Behaviour pathology

Self-narcotization

food access and conditions indoors or outdoors may

Body damage prevalence

lead to risk of injury (Schlichting & Smidt, 1987).

Reduced ability to grow or breed

Reduced life expectancy

Management methods and husbandry include all aspects of feeding, moving of animals, grouping, milking, serving, etc. Mutilations for the benefit of farming practice or of the animal require some special of welfare. Hence welfare varies from very poor to mention, both as regards the methods used and the very good and can be scientifically assessed (Broom & consequences for the animals. Methods of handling Johnson, 1993; Broom, 1998a, 1999b).

and moving animals are of importance before and after

Indicators of animal welfare are listed in Box 55.1.

transport, at markets and at lairage as well as on the farm. These include disease prevalence and reduced ability to grow and breed. As explained by Broom and Johnson (1993), the welfare of a diseased individual is poorer than that of an individual which is not diseased, and reduced ability to produce offspring given appropriate vehicle construction and usage, market accommodation and procedures, and slaughter methods. Welfare problems that should be taken into account in cattle practice have been reviewed by Broom (1988).

opportunities also indicates poor welfare. Individuals which are finding it difficult to cope with their environment, or which are failing to cope, may be more likely

Ill treatment and neglect

to become diseased, less likely to produce embryos, less likely to carry young to term and more likely to die early. Human actions that can lead to poor cattle welfare

include ill treatment, neglect and inadequate produc-

tion systems. Ill treatment occurs most frequently when animals are being moved around the farm, when they

Welfare problem areas

are being loaded into or out of vehicles, or when they are at market or lairage. Those who do ill treat animals

Whilst cattle management has been changing, our can be advised of likely economic effects of their knowledge of cattle physiology and behaviour has been actions as well as being told about the laws on the improving. It is clear that cattle have complex regulatory subject. Neglect includes failure to provide an adequate dietary processes, elaborate social structure and sophisticated diet, failure to treat disease and the lack of normal cated learning ability (Kilgour & Dalton, 1984; Stricklin husbandry procedures. The diet may be inadequate in & Kautz-Scanavy, 1984; Fraser & Broom, 1990). These nutrient composition or in quantity. Cattle are some- results have made many animal scientists reconsider the times undernourished for a period, whilst food is scarce effects of conditions and procedures on farms, both in or expensive, with the expectation that compensatory

terms of their efficiency as regards production and with growth will occur when more food is provided. If respect to the welfare of the animals.

the undernourishment amounts to starvation and this

The general range of welfare problem areas is the

is clear from the condition of the animal, then this is same for cattle as for other farm animals (Box 55.2).

serious neglect. Lack of knowledge on the part of the

Ill treatment refers principally to physical abuse of

farmer may result in the provision of a poor diet or in

animals. Neglect includes failing to give food and water,

failure to treat disease. This is poor husbandry, a very

or to clean out, or to treat disease or to assist as neces-

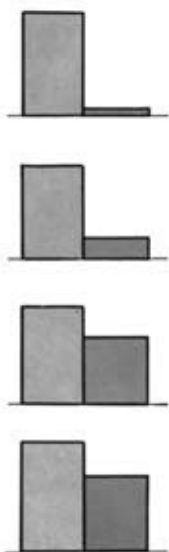
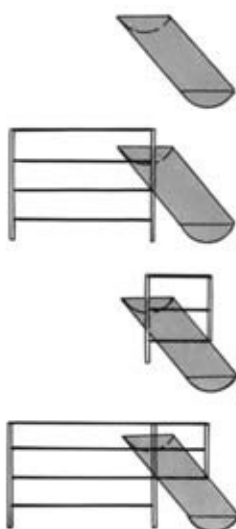
important cause of welfare problems. Advice on good

sary at calving. Accommodation for animals may, for

husbandry methods can be an important veterinary

example, give insufficient space, poor flooring or poor

service. If animals are diseased and require treatment



Feeding trough barriers		Feeding times in 3 min. competition trial	
None	High rank	100%	
		100%	
Body barrier	High rank	100%	
		100%	
Head barrier	High rank	100%	
		100%	
Complete barrier	High rank	100%	
		100%	
		High rank	Low rank

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there is a moral obligation upon the veterinarian to remain to be determined. Competitive feeding situations where there are no individual feeding places pose

sworn on admission to Membership of the Royal
extra problems for cattle. The subordinate individual
College of Veterinary Surgeons, includes the promise
has to attempt to obtain food despite the attacks or
that 'my constant endeavour will be to ensure the
threats of other individuals. Bouissou (1970) found that
welfare of animals committed to my care'. In certain
the greater the extent of the barrier between feeding
circumstances, treatment without any prospect of
places for cows, the fewer the attacks that occurred (Fig.
payment may be necessary. In other circumstances it
55.1). A trough that requires subordinate cows to come
may be best to call in the State Veterinary Service, the
close to dominant individuals results in those subordi-
police or the RSPCA, even if to do so might mean
nates walking greater distances and taking longer to
losing a client.

obtain a meal (Albright, 1969; Fig. 55.2). Calves of low
social rank obtain less of the favoured food if trough
space is restricted (Broom, 1982). In order to minimize

Housing and management (see also pp.

such welfare problems, which are often associated with poor weight gain, farmers should provide feeding spaces for all individuals, preferably with barriers between the individual places. Adaptation to food

General points

access from a single source is possible for cattle

Feeding of housed cattle may lead to difficulties for the

however, for a transponder-operated feeding stall can

animals because the acquisition of food in housing

be quite successful (Albright, 1981), but some indi-

conditions is very different from that when grazing.

viduals in a herd may have difficulties in learning how

Physical difficulties may occur, as described by Cermak

to reach the feed in such systems.

(1987), but social factors are also very important. Cattle

Another general problem for housed cattle is having

synchronize their feeding to a large extent (Benham,

to stand on floors that are wet, slippery, uneven or

1982; Potter & Broom, 1987), so where group feeding is

hazardous because of sharp edges. Slippery slats can

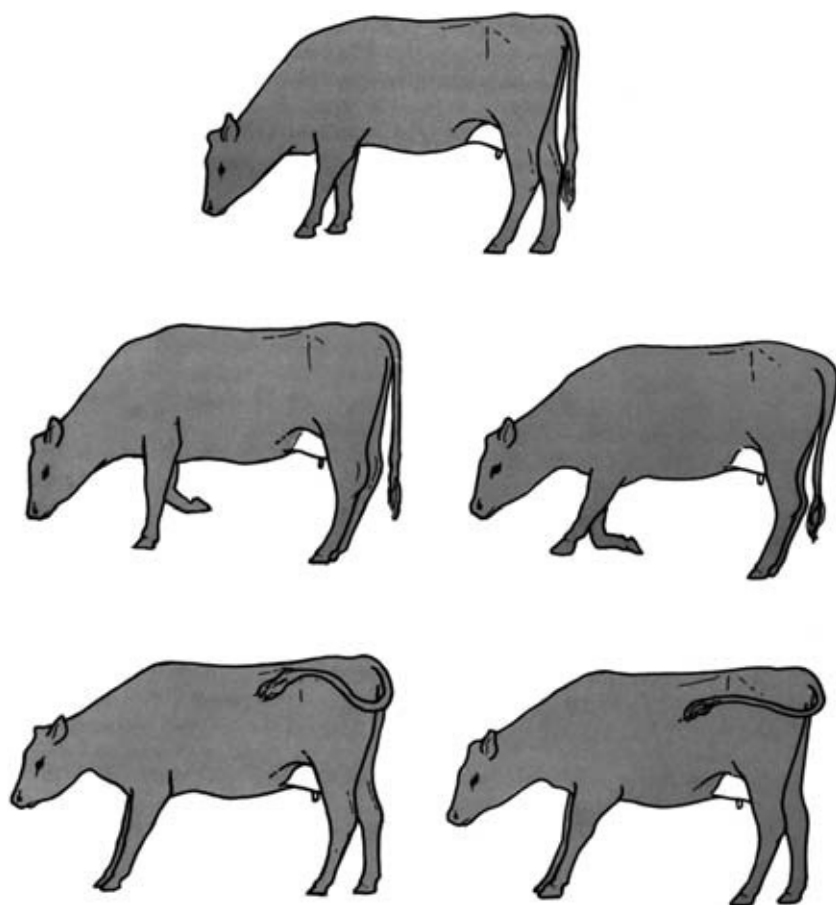
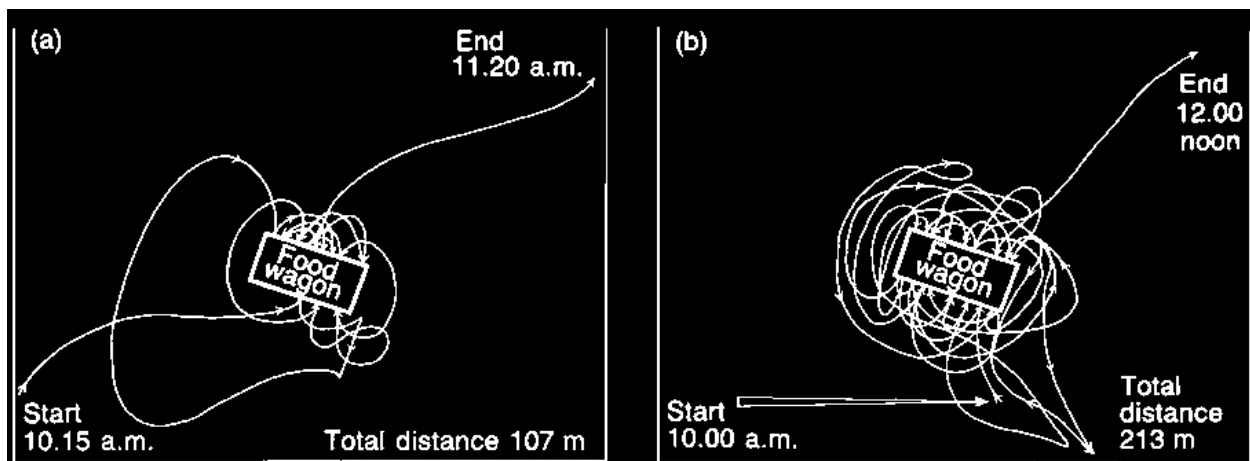
possible enough feeding places for each animal are lead to difficulties in standing or lying (Andreae & required (Metz, 1983; Wierenga, 1983). Those animals Smidt, 1982; Fig. 55.3). These and other inadequacies of that cannot find a feeding place may not get sufficient flooring can result in limb injuries, foot lameness, food and it is likely that there are adverse effects on tail-tip necrosis and various diseases. Lameness is the their welfare. The precise effects of the frustration that greatest welfare problem of housed dairy cows and occurs when food is inaccessible because of competition factors influencing its occurrence include floor quality

Fig. 55.1

Physical barriers affected feeding times by cows

ranking high and low in a competitive order. With no barrier (a) the low ranking cows were scarcely able to feed. A body barrier (b) improved the situation slightly for the low ranking cows but a head barrier (c) and a complete barrier (d) had a much

greater effect (redrawn after Craig, 1981; data from Bouissou, 1970).





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Fig. 55.2

The paths of two cows in a

herd after food is provided in a food wagon are shown. Animal (a) was found to be high in a competitive order whereas animal (b), which was low in that order, walked further because of displacement at the food wagon and took longer to feed (after Broom, 1981; modified after Albright, 1969).

Fig. 55.3

Behavioural alterations in young cattle on a slatted floor (after Andreae & Smidt, 1982).

and poor drainage, which results in cows standing in colostrum for a variety of reasons (Edwards, 1982; slurry (Wierenga & Peterse, 1987).

Edwards & Broom, 1982; Broom, 1983). Management practices that maximize the chance that colostrum will be obtained and minimize contact with pathogens have

Calf welfare

important beneficial effects on calf welfare. If calves of In the first few days after birth the major calf welfare

dairy cows are normally left with their mother for the problems are enteric and respiratory diseases. The first 24 or 48 hours, the risk that the calf will not suckle calves of dairy cows may fail to obtain sufficient early enough to obtain and absorb the immunoglobulin



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from colostrum can be minimized by the stockworker Grooming behaviour is important as a means of placing one of the mother's teats in the mouth of the minimizing disease and parasitism and calves make calf as early as possible after the calf stands. Group-considerable efforts to groom themselves thoroughly calving situations where several cows calve during a

(Fraser & Broom, 1990). Calves need to be able to short period can lead to a cow's colostrum being drunk groom their whole bodies effectively.

by a calf other than her own or to calves being rejected

A variety of nutrients are needed by calves. Sufficient

by their own mothers. Such occurrences can be pre-

iron is needed to allow normal activity and to minimize

vented by providing separate calving boxes, which

disease.

should ideally allow the cows some visual contact with

The needs of young calves are met most effectively

other cows. The provision of soft bedding for the calf is

by the presence and actions of their mothers. In the

also desirable and is easier where special calving

absence of their mothers, calves associate with other

accommodation is available.

calves if possible and they show much social behaviour.

Dairy calves are deprived of their mother from an

The need to show full social interaction with other

early age and many are individually housed so that they

calves is evident from calf preferences and from the

are confined in a small space and deprived of all or most adverse effects on calves of social isolation (Broom & social contacts.

Leaver, 1978; Dantzer et al. , 1983; Friend et al. , 1985; Lidfors, 1994).

The needs of calves

Comparisons of veal calf housing and

A need is a requirement, which is a consequence of the management systems

biology of the animal, to obtain a particular resource or respond to a particular environmental or bodily stim-

The major housing systems which have been compared

ulus. The needs of calves are described in detail by

in studies of calf welfare are individual crates (Fig.

Broom (1991, 1996) and examples are given here.

55.4), group housing on slats and group housing on

The calf needs to ingest colostrum very soon after

straw. Where calves are housed individually, the size of

birth and milk thereafter. It also needs to show sucking

the crate and whether or not the sides are solid have

behaviour and if a calf is not obtaining milk from a real

been varied. All aspects of diet are important in rela-

or artificial teat, it sucks other objects (Broom, 1982, tion to welfare. For example, if inappropriate proteins 1991; Metz, 1984; Hammell et al. , 1988).

or carbohydrates are fed, the calf may be unable to Calves need to rest and sleep in order to recuperate utilize them and if milk is acidified too much, calves may and avoid danger. They need to use several postures which include one in which they rest the head on the legs and another in which the legs are fully stretched out (de Wilt, 1985; Ketelaar-de Lauwere & Smits, 1989, 1991).

Exploration is important as a means of preparing for the avoidance of danger and is a behaviour shown by all calves (Kiley-Worthington & de la Plain, 1983; Fraser & Broom, 1990). Calves need to explore and it may be that higher levels of stereotypies (Dannemann et al. , 1985) and fearfulness (Webster & Saville, 1981) in poorly lit buildings are a consequence of inability to explore.

Exercise is needed for normal bone and muscle development and calves choose to walk at intervals if

they can, show considerable activity when released from a small pen and have locomotor problems if confined in a small pen for a long period (Warnick et al. , 1977; Dellmeier et al. , 1985; Trunkfield et al. , 1991).

Normal calf anatomical, physiological and behavioural development occurs only if the calves have some fibre-containing material to eat (van Putten & Elshof, 1978; Webster, 1984; Webster et al. , 1985a) so it is clear that they need fibre in their diet after the first few weeks

Fig. 55.4

Veal calf in crate with slatted floor. The front of the crate has been removed.

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find it very unpalatable. However, the two aspects of Vries et al. , 1986). Individual housing of beef animals is diet which have been of greatest concern in relation to more frequent when they are bulls than when they are calf welfare have been the amount of fibre and the steers. In Germany almost all beef animals are bulls, but amount of iron.

in the UK most were steers before the ban on growth

The incidence of disease in young calves is too high, promoters.

for example, 25 per cent of veal calves had to be treated

Fighting and mounting can lead to welfare problems

for respiratory disease in a study by van der Mei (1987).

when beef animals, especially bulls, are kept in groups.

The use of antibiotics to prevent disease is also a

The most important way of minimizing such problems

problem. It is important for calf welfare and for farm

is to keep the animals in stable groups, since social

economics that disease levels be lowered.

mixing leads to much fighting with consequent injuries,

One aspect of management which causes problems is

bruising and extreme physiological responses (Kenny &

the practice of mixing calves from different sources.

Tarrant, 1982). In stable groups, mounting may lead to

Webster (1984) found that calves purchased and

more injury than does fighting (Appleby & Wood-

brought into a unit were five times more likely to

Gush, 1986). Animals that are frequently mounted

require treatment for disease. A second aspect is

become bruised and may suffer severe leg injuries.

hygienic practice by farm staff and a third is early detection of disease. These variables seem to be more important than the housing system in exacerbating disease.

Mounting can be greatly reduced by the use of overhead bars, which physically prevent it, or an electrified grid, which deters animals that wish to mount. The brief initial experience of an electric shock has a relatively small adverse effect on welfare as compared with the 1997 which required group housing of calves after 8 weeks of age, individual pens at least as wide as the height of the calf at the withers, no tethering of calves provided also have considerable effects on welfare.

As a consequence of the evidence of poor welfare in veal calves, the European Union passed a directive in 1997 which required group housing of calves after 8 weeks of age, individual pens at least as wide as the height of the calf at the withers, no tethering of calves provided also have considerable effects on welfare.

except for <1 h at feeding time, sufficient iron to ensure High stocking densities lead to more aggression, injury and bruising. Beef animals increase rapidly in body-

in the diet increasing from 50 g/day at 8 weeks to weight, but they have little exercise if they are housed 250 g/day at 20 weeks. Many EU calf producers have in small pens and their leg growth may not be able to found group housing of calves to be more successful keep pace with that of the rest of the body. The final economically than the old crate system and white veal weights reached are much higher now than they used can still be produced from systems which comply with to be so the legs are scarcely adequate to support the the new law.

body. The consequence is cartilage damage, clear indications of limb pain and obvious difficulties in standing and lying (Dämmrich, 1987). Graf (1984) found that

Beef cattle welfare

these problems were absent if fattening bulls were reared on deep straw and that such conditions also The housing conditions for calves destined for beef led to fewer behavioural problems. Beef cattle have a production are sometimes similar to those kept for strong preference for straw or other bedding.

veal production so they have similar welfare problems.

All of these issues are reviewed in the EU Scientific

Older beef animals are kept in small individual pens or

Committee on Animal Health and Animal Welfare

are tethered in some countries and they then show

Report on the Welfare of Cattle Kept for Beef Produc-

much stereotyped behaviour. Riese et al. (1977)

tion, 2001.

reported that stereotyped behaviour included tongue

rolling, weaving movements and self-licking. Wierenga

(1987) reported that one-third of young, individually

The welfare of dairy cows

housed bulls spent several minutes in every hour

showing tongue rolling. Physiological responses to

The major welfare problems of dairy cows are lameness,

confinement also occur. Ladewig (1984) reported that

mastitis and any conditions which lead to impaired

tethered bulls showed more frequent episodes of high

reproduction, inability to show normal behaviour, emer-

blood cortisol levels than did bulls able to interact

gency physiological responses or injury (Broom, 1999c).

socially in groups. Such abnormal behaviour and physiology are probably exacerbated by both social deprivation and inability to perform behaviours because of spatial restriction. Tethered animals lack exercise and

Leg and foot problems (see Chapters 31, 32)

For a review of lameness, including the extent to which have different patterns of muscle fibres from those free it is a welfare problem, see Greenough and Weaver

to walk (Jury et al. , 1998) and more osteochondrosis (de (1996). Almost all animals which walk with a limp, or

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reduce walking to a low level or avoid walking when- have some lameness problems which are exacerbated ever possible suffer from some leg or foot pain. Their

by social factors (Bickert et al. , 1997). Since the best ability to carry out various preferred behaviours is gen-straw yards, with an abrasive area on which normal hoof

erally impaired and there may be adverse consequences wear occurs, have little lameness, these may be the best for various other aspects of their normal biological solution for housed cows. Mastitis incidence is affected functioning. Lameness always means some degree of

by hygiene at milking and various other conditions of poor welfare and sometimes means that welfare is very management. Poorly designed housing systems can poor indeed.

result in a variety of welfare problems and these can be Measurements of the extent to which some degree of exacerbated by high stocking density. Most of these lameness occurs in dairy cows include 35–56 cases per problems, such as those resulting from cubicles being 100 cows per annum in the USA, 59.5 cases per 100 too short for the length of the cows now occupying cows per annum in the UK and more than 83 per cent them or of poor design of cubicles which do not allow of examined cows in The Netherlands. The actual adequate movements in the cow, are well known so are figures depend upon the method of assessment and mentioned briefly here. In general it seems that many most of these cases were not treated by veterinary dairy cow housing systems, and cubicles in particular, surgeons, but there is no doubt that lameness is often do not provide an environment to which cows can adapt

a severe welfare problem (Broom, 1999c).

easily. The best straw yards seem to be the most successful as they give the cows more opportunity to Mastitis (see Chapter 23)

control their interactions with their environment.

Mastitis in mammals is a very painful condition. The sensitivity to touch of affected tissues is clearly evident

Milk yield and welfare in dairy cows

and there is obvious damaging of normal function.

The dairy cow of 1998 could produce 18 000 l or more

Mastitis prevalence should have declined greatly with of milk per annum with a peak milk yield of 75 l per day.

improved methods of prevention and treatment, but it

This compares with UK figures of 6000 l and 30 l per day

has not declined as much as it should have done.

10 years ago (Webster, 1993) and a beef cattle average

Webster (1993) reports 40 cases of mastitis per 100 cows

of 1000–2000 l and 10 l per day. The dairy animal is pro- per year as an average for the UK.

ducing considerably more than its ancestor would have.

This raises the questions of whether it is at or beyond

Reproductive problems (see Chapters 34, 35 and 36)

its maximum production level and the extent of any

Reproductive problems in dairy cows have become welfare problems.

very common in recent years with large numbers of

The peak daily energy output of the dairy cow per

cows being culled because of failure to get in calf. In a

unit bodyweight is not very high in comparison with

study of 50 dairy herds in England, Esslemont and

some other species such as seals or dogs, but the product

Kossaibati (1997) found that farmers reported failure to

of daily energy output and duration of lactation is very

conceive as the predominant reason for culling, with 44

high indeed. Hence long term problems are the most

per cent of first lactation, 42 per cent of second lacta-

likely to occur (Nielsen, 1998). This is what we see

tion and 36.5 per cent of cows in total being culled for

because, although some cows seem to be able to

this reason. However, mastitis, feet and leg problems,

produce at high levels without welfare problems, the

ketosis and other disease conditions can lead to repro-

risk of poor welfare indicated by lameness, mastitis or
ductive problems and it is difficult to discover their
fertility problems is greater as milk yield increases.
initial cause from farmers' records. A report by Plaizier
The steady increase in reproductive problems as milk
et al. (1998) concerning Canadian herds indicated that yields have increased is
well known. As Studer (1998)
reproductive culling risk varied between 0 and 30 per
states, 'despite programmes developed by veterinarians
cent, with a mean of 7.5 per cent.
to improve reproductive herd health, conception rates
have in general declined from 55–66% 20 years ago to
45–50% recently (Spalding et al. , 1975; Foote, 1978;
Housing systems and welfare (see pp. 40–44,
Ferguson, 1988; Butler & Smith, 1989). During the
Chapter 56)
same periods, milk production has greatly increased.'
The incidence of lameness is much worse in housed
Studies showing that milk yield is positively corre-
cows than in cows at pasture. Cows at pasture may have
lated with the extent of fertility problems have come

stone damage to hooves if they do not have a suitable
 from a range of different countries (van Arendonk et
 place to walk, but wet cubicle houses or poorly main-
 al. , 1989; Oltenacu et al. , 1991; Nebel & McGilliard, tained straw yards can
 result in very high levels of lame-1993; Hoekstra et al. , 1994; Pösö &
 Mäntysaari, 1996; ness. Even the best cubicle housing systems seem to
 Pryce et al. , 1997, 1998). Studer (1998) explains that high

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Table 55.1

Positive correlations between milk production level
 welfare of cows. Current trends towards ever greater
 and indicators of poor welfare (from Pryce et al., 1997 with per-milk production
 and feed conversion efficiency should
 mission of the American Society of Animal Science).
 not be continued unless it can be ensured that welfare
 is good (Broom, 1994; Phillips, 1997). Bovine soma-
 Milk yield from 33 732 lactation records:
 totropin (BST) (see p. 1073) results in high milk yields
 calving interval
 0.50 ± 0.06
 and higher levels of mastitis, lameness, reproductive
 days to first service

0.43 ± 0.08

disorders and other problems such as those at the injection site (Broom, 1993; Willeberg, 1993; Kronfeld, 1997; mastitis

0.21 ± 0.06

foot problems

0.29 ± 0.11

milk fever

0.19 ± 0.06

Willeberg, 1997; Broom, 1998a). Whether or not much of the effect of the genetically engineered hormone is a consequence of the milk yield, the poorer welfare

Table 55.2

Positive correlations between milk production level caused by the BST is unacceptable.

and indicators of poor welfare (from Pryce et al., 1998 with perThe Report of the EU Scientific Committee on

mission of the American Society of Animal Science).

Animal Health and Animal Welfare (1999) on animal welfare aspects of the use of bovine somatotrophin

Milk yield from 10 569 lactation records:

concluded as follows:

calving interval

0.28 ± 0.06

days to first service

0.41 ± 0.06

'BST is used to increase milk yield, often in already high-mastitis

0.29 ± 0.05

producing cows. BST administration causes substantially somatic cell count

0.16 ± 0.04

and very significantly poorer welfare because of foot problems

0.13 ± 0.06

increased foot disorders, mastitis, reproductive disorders and other production related diseases. These are problems which would not occur if BST were not used and producing cows which are thin and whose body condition often results in unnecessary pain, suffering and distress. tion score declines by 0.5–1.0 during lactation often If milk yields were achieved by other means which

experience anoestrus. A loss of condition score of about 1.0 during lactation was normal in the review presented by Broster and Broster (1998). Data on the relationship between milk yield and reproduction measures also causes localized swellings which are likely to result in discomfort and hence some poor welfare.’ and 55.2.

In some studies, effects of health problems on repro-

The Committee also made the following

duction are evident, for example, Peeler et al. (1994) recommendation:

showed how cows which were lame in the period before

‘BST use causes a substantial increase in levels of foot

service were less likely to be observed as being in

problems and mastitis and leads to injection site reac-

oestrus. The lameness could be more likely in high

tions in dairy cows. These conditions, especially the first

producing cows. Direct links between level of milk

two, are painful and debilitating, leading to significantly poorer welfare in the treated animals. Therefore from evident from a range of studies, positive correlations the point of view of animal welfare, including health, being reported by Lyons et al. (1991), Uribe et al. (1995) the Scientific Committee on Animal Health and Animal and Pryce et al. (1997, 1998; see Tables 55.1, 55.2). In Welfare is of the opinion that BST should not be used addition to mastitis and leg and foot problems, which in dairy cows.'

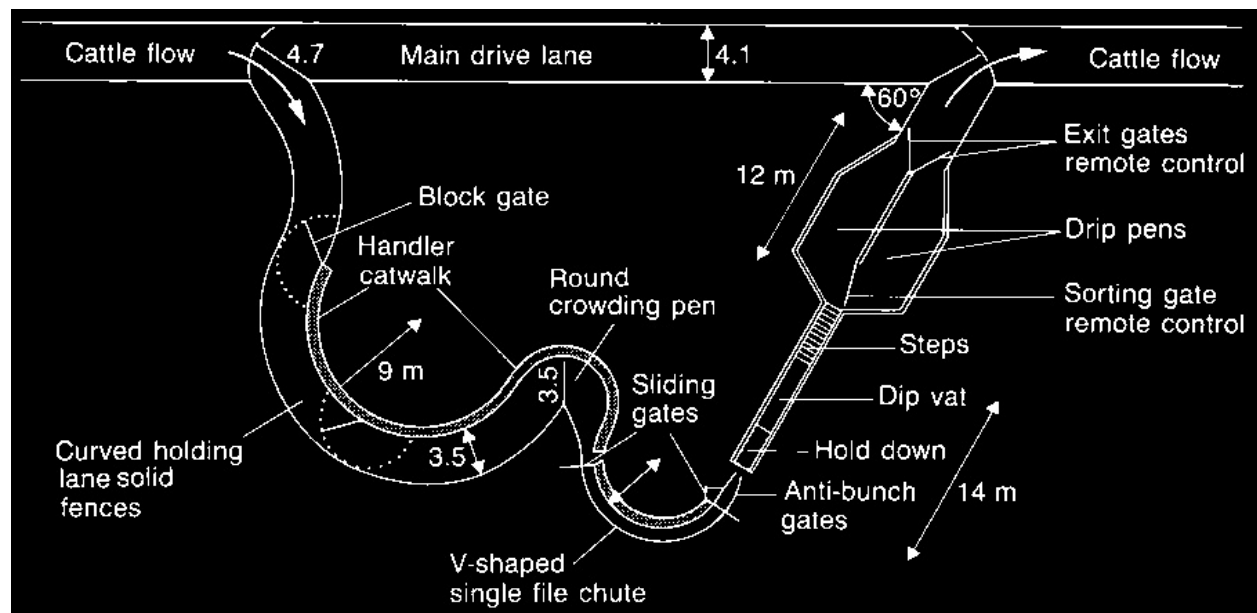
are often measured in such studies, the occurrence of other clinical conditions can also be affected by production level. Modern, high producing cows with good body condition have a high incidence of milk fever,

Farm operations

retained placenta, metritis, fatty liver and ketosis (Studer, 1998).

Certain mutilations of cattle are performed on farms by The high yields of modern dairy cows are a consequence of genetic selection and feeding. Cows are staff with no veterinary qualification. In most cases, no

anaesthetic is used. The most widespread of these adapted to high fibre, low density diets. The ways in operations are disbudding or dehorning, castration and which they have been modified genetically do not various sorts of individual marking. Some of the procedures used must also cause pain to the animals, but adapt easily to high grain diets or to manufactured diets there is little precise information about this. The use of with high protein and low fibre. Genetic selection has caustic materials that remain in contact with living not taken adequate account of the adaptability and tissue for any length of time is likely to cause severe



pain and should be avoided. Any use of hot irons on adverse effect on some cows so that their milk let-down living tissue must also cause pain and hot iron branding may be prevented and they may become extremely is a painful and unnecessary form of marking. Castration is often carried out by applying a rubber ring unwilling to move towards the parlour.

tion is often carried out by applying a rubber ring The problems associated with the design of races for around the testicles until the tissue in them dies and it moving cows to the milking parlour are very similar to seems likely that this is also very painful for a long those of designing races used for other purposes, such period. Even the ubiquitous ear notching and punching as movement towards vehicles prior to transport. The must be painful for a few days and it would be better if most extensive study of how to design good races is that alternative marking methods such as freeze branding or of Grandin (1983, 2000). She reported that cattle often tattooing could be used.

balk if they encounter dark areas or areas of extreme

More extensive mutilations should only be carried out under anaesthetic by qualified veterinarians and they may also pose problems for cattle that are being driven and long straight races may result in animals practices that necessitate the use of operations should be avoided. An example of a problem area is the breed-consequence of these observations, Grandin recommending of cows such that their calf cannot be born normally. No animal should be made pregnant if there is a likelihood that caesarean section or a difficult birth will occur.

corners or long straights (Fig. 55.5). Other studies also suggest that if animals are being loaded into vehicles, the ramp should be long and sloping not more than 1

Handling, transport and slaughter

*in 7, should allow a good grip for the feet of the cattle
of cattle*

*and should have solid sides, and the interior of the
vehicle should be well lit.*

Every dairy farmer has to be able to move dairy cows

*Vehicles are often not well designed as regards floor-
in milk to and from the milking parlour. If the races and
ing, ventilation and ease of subdivision. Just as impor-
collecting yards that are used or the methods of moving
tant as vehicle design, as regards the welfare of animals
the animals are inadequate and disturbing to some or
during transport, is the behaviour of the transport staff.
all of the cows, there will be welfare problems. Such
Problems arise because of rough treatment during
welfare problems will often be associated with reduced
loading, over- or understocking of compartments on the
milk yield. Cows may be reluctant to enter a milking
vehicle, inconsiderate driving or leaving the animals in
parlour because of the behaviour of the stockworker or
conditions that are too hot or too cold and windy for
because of design faults in the parlour that result in*

them. The other major transport problems are the effect uncomfortable milking stalls or stray voltages. Such of very long journeys, especially where there are no problems can lead to the use of excessive force by stops for food and water, and contact between transported animals in the collecting yard or to forcing animals towards the parlour entrance using gates or disease transmission and poor animal welfare. This area an 'electric dog'. The 'electric dog', which is a row of electrically live wires hanging downwards and moved towards the cow in the rear of the yard, has a marked (CEC) Report (1984) (also see Broom et al., 2002).

Fig. 55.5

Races for cattle should have no long straights or sharp corners so Grandin (1980) designed this race for movement to vehicles. (Measurements are in metres.)

When cattle arrive at an abattoir they are often

*production in dairy cows. *Livestock Production Science*, **21**, injured or bruised during unloading because of*

1–12.

too much haste on the part of animal handlers or

Benham, P.F.J. (1982) Synchronisation of behaviour in grazing

inadequate ramps. Grandin (1979, 1980) reported that

*cattle. *Applied Animal Ethology*, **8**, 403–404.*

66 per cent of bruises of the loin area occurred during

Bickert, W.G., Shaver, R.D., Galindo, F.A., Broom, D.M. &

Cermak, J. (1997) Laminitis and factors predisposing to

loading or unloading of trucks. At lairage, animals are

*lameness: nutrition, behavior, and housing. In *Lameness in often mixed with individuals that are strange to them.**

Cattle, 3rd edn (ed. by P.R. Greenough & A.D. Weaver), pp.

This causes much fighting amongst bulls and consider-

293–307. W.B. Saunders, Philadelphia.

*able emotional disturbance in other cattle. *Studies of**

Bouissou, M.F. (1970) Role du contact physique dans la

bulls by Kenny and Tarrant (1982) show that mixing at

manifestation des relations hierarchiques chez les bovins:

lairage causes much fighting, high levels of bruising and consequences pratiques. Annales de Zootechnie, 19, 279–85.

other injury and a great increase in the incidence of

Broom, D.M. (1981) Biology of Behaviour. Cambridge University Press, Cambridge.

meat are of economic importance as well as indicating

Broom, D.M. (1982) Husbandry methods leading to inadequate social and maternal behaviour in cattle. In Disturbed Behaviour in Farm Animals (ed. by W. Bessei).

In an efficient slaughterhouse the period during Hohenheimer Arbeiten, Vol. 12b. Eugen Illmer, Stuttgart.

which animals are moved from pens to the point of Broom, D.M. (1983) Cow–calf and sow–piglet behaviour in slaughter can be very brief and the stunning and slaughter relation to colostrum ingestion. Annales de Recherches

ter procedure itself can result in no pain for the animal.

Vétérinaires, 14, 342–8.

Welfare is worse if the animals are kept in a confined

Broom, D.M. (1986) Indicators of poor welfare. British

race for a period of more than one or two minutes

Veterinary Journal, **142**, 524–6.

before stunning, if stunning is carried out inadequately

Broom, D.M. (1988) Welfare considerations in cattle practice.

or if there is inversion before slaughter or no stunning.

In Proceedings of the British Cattle Veterinary Association Poor equipment or lack of care by slaughter staff can

1986–87 (ed. by M. Vaughan), pp. 153–64. British Cattle result in extreme pain and discomfort for the animals.

Veterinary Association, London.

Extreme pain and discomfort are also inevitable if

Broom, D.M. (1991) Needs and welfare of housed calves. In

New Trends in Veal Calf Production. Proceedings of the

animals are not stunned, for example in the Jewish sche-

International Symposium on Veal Calf Production (ed. by chita or the Muslim halal ritual slaughter procedures.

J.H.M. Metz & C.M. Groenestein), EAAP Publication No.

There is a period during which evoked potentials in the

52. Pudoc, Wageningen.

brain can still be produced after the throat is cut that

Broom, D.M. (1993) Assessing the welfare of modified or

may last for from a few seconds to two minutes, during

treated animals. *Livestock Production Science*, **36**, 39–54.

which the animal must be in great pain and distress. As

Broom, D.M. (1994) *The effects of production efficiency on*

the heart still beats after stunning and blood drains from

animal welfare. In Biological Basis of Sustainable Animal

the animal just as effectively whether or not the animal

Production. Proceedings of the 4th Zodiac Symposium (ed.

is stunned there is no logical reason why stunning should

by E.A. Huisman, J.W.M. Osse, D. van der Heide, S. Tam-

not be carried out before the throat is cut.

minga, B.L. Tolkamp, W.G.P. Schouten, C.E. Hollingsworth

& G.L. van Winkel), EAAP Publication 67, pp. 201–10.

Wageningen Pers, Wageningen.

Broom, D.M. (1996) *Scientific research on veal calf welfare.*

In Veal Perspectives to the Year 2000, Proceedings of the

References

International Symposium, Le Mans, pp. 147–53. Fédération de la Vitellerie Francaise, Paris.

Albright, J.L. (1969) *Social environment and growth. In*

Broom, D.M. (1998a) The effects of biotechnology on animal

Animal Growth and Nutrition (ed. by E.S.E. Hafez & I.A.

welfare. In *Animal Biotechnology and Ethics* (ed. by A.

Dyers). Lea & Febiger, Philadelphia.

Holland & A. Johnson), pp. 69–82. Chapman & Hall,

Albright, J.L. (1981) Training dairy cattle. In *Dairy Sciences* London.

Handbook, Vol. 14, pp. 303–70. Agriservices Foundation, Broom, D.M. (1998b) Welfare, stress and the evolution of feel-Clovis, CA.

ings. *Advances in the Study of Behaviour*, **27**, 371–403.

Andreae, U. & Smidt, D. (1982) Behavioural alterations in

Broom, D.M. (1999a) Welfare and how it is affected by regu-

young cattle on slatted floors. In *Disturbed Behaviour in*

lation. In *Regulation of Animal Production in Europe* (ed.

Farm Animals (ed. by W. Bessei), *Hohenheimer Arbeiten*, by M. Kunisch & H. Ekkel), pp. 51–7. KTBL, Darmstadt.

Vol. 121, pp. 51–60. Eugen Ulmer, Stuttgart.

Broom, D.M. (1999b) Animal welfare: the concept and the

Appleby, M.C. & Wood-Gush, D.G.M. (1986) Development of

issues. In *Attitudes to Animals: Views in Animal Welfare* (ed.

behaviour in beef bulls: sexual behaviour causes more prob-

by F.L. Dolins), pp. 129–42. Cambridge University Press,

lems than aggression. *Animal Production*, **42**, 464.

Cambridge.

Arendonk, J.A.M. van, Hovenier, R. & Boer, W. de (1989)

Broom, D.M. (1999c) The welfare of dairy cattle. In Proceed-Phenotypic and genetic association between fertility and

ings of the 25th International Dairy Congress, Future Milk

Welfare • 965

Farming (ed. K. Aagaard), pp. 32–9. Danish National Com-EU (1999) Report of the EU Scientific Committee on Animal

mittee of the International Dairy Federation, Aarhus.

Health and Animal Welfare. Aspects of the use of bovine

Broom, D.M. (2000) Welfare assessment and problem areas

somatotrophin. CEC, D.G. Sanco, Brussels.

during handling and transport. In Livestock Handling and

EU (2001) Report of the Scientific Committee on Animal

Transport, 2nd edn (ed. T. Grandin), pp. 43–61. CABI,

Health and Animal Welfare on the welfare of cattle kept for

Wallingford.

beef production. CEC, D.G. Sanco, Brussels.

Broom, D.M. & Johnson, K.G. (1993) Stress and Animal

Ferguson, J.D. (1988) Feeding for reproduction. In Proceed-Welfare. Kluwer, Dordrecht.

ings of the Dairy Production Medicine, Continuing Educa-

Broom, D.M. & Leaver, J.D. (1978) The effects of group-

tion Group Annual Meeting, pp. 48–56. Vet. Learning

rearing or partial isolation on later social behaviour of

System Co, Trenton, NJ.

calves. Animal Behaviour, 26, 1255–63.

Foote, R.H. (1978) Reproductive performance and problems

Broom, D.M., Barton-Grade, P., Ferlazzo, A., Hartung, J.,

in New York dairy herds. Search Agriculture (Geneva NY), Mantela, X. & Warriss, P. (2002) The Welfare of Animals, 8, 1.

during Transport. Report of the Scientific Committee for Fraser, A.F. & Broom, D.M. (1990) Farm Animal Behaviour Animal Health and Welfare, European Commission. D.G.

and Welfare. CABI, Wallingford.

Sanco, Brussels.

Friend, T.H., Dellmeier, G.R. & Gbur, E.E. (1985) Compari-

Broster, W.H. & Broster, V.J. (1998) Body score of dairy cows.

son of four methods of calf confinement. I. Physiology.

Journal of Dairy Research, 65, 155–73.

Journal of Animal Science, 60, 1095–101.

Butler, W.R. & Smith, R.D. (1989) Interrelationships between Graf, B.P. (1984) Der Einfluss unterschiedlicher Laufstall energy balance and post partum reproductive function in

systeme auf Verhaltensmerkmale von Mastochsen. Doktor dairy cattle. Journal of Dairy Science, 72, 767–83.

Dissertation der Eidgenossischen Technischen Hochschule,

Ceppi, A. & Blum, J.W. (1994) Effects of growth hormone on Zürich.

growth performance, haematology, metabolites and hor-

Grandin, T. (1979) *The effect of stress on livestock and meat*

mones in iron-deficient veal calves. *Journal of Veterinary quality prior to and during slaughter. International Journal Medicine A*, **41**, 443–55.

for the Study of Animal Productions, **1**, 313–37.

Cermak, J. (1987) *The design of cubicles for British Friesian*

Grandin, T. (1980) *Observations of cattle behaviour applied*

dairy cows with reference to body weight and dimensions,

to the design of cattle-handling facilities. Applied Animal spatial behaviour and upper leg lameness. In Cattle Housing Ethology, **6**, 19–31.

Systems, Lameness and Behaviour (ed. by H.K. Wierenga & Grandin, T. (1983) Welfare requirements of handling facilities.

D.J. Peterse), Current Topics in Veterinary Medicine and

In Farm Animal Housing and Welfare (ed. by S.H. Baxter.

Animal Science, Vol. 40, pp. 119–28. Martinus Nijhoff, M.R. Baxter & J.A.C. MacCormack), Current Topics in

Dordrecht.

Veterinary Medicine and Animal Science, Vol. 21, pp. 137–49.

Commission of the European Communities (1984) Interna-

Martinus Nijhoff, The Hague.

tional transport of farm animals intended for slaughter.

Grandin, T. (ed.) (2000) Livestock Handling and Transport, EUR 9556 EN. CEC, Luxembourg.

2nd edn. CABI, Wallingford.

Craig, J.V. (1981) *Domestic Animal Behaviour*. Prentice Hall, Greenough, P.R. & Weaver, A.D. (1996) *Lameness in Cattle*, Englewood Cliffs.

3rd edn. Saunders, Philadelphia.

Dämmrich, K. (1987) *The reactions of the legs (bone; joints)*

Gygax, M., Hirni, H., Zwahlen, R., Lazary, S. & Blum, J.W.

to loading and its consequences for lameness. In Cattle

(1993) *Immune functions of veal calves fed low amounts of*

Housing Systems, Lameness and Behaviour (ed. by H.K.

iron. *Journal of Veterinary Medicine A*, **40**, 345–58.

Wierenga & D.J. Peterse). *Current Topics in Veterinary Hammell, K.L., Metz, J.H.M. & Mekking, P. (1988) Sucking*

Medicine and Animal Science, Vol. 40, pp. 50–5. *Martinus behaviour of dairy calves fed milk ad libitum by bucket or Nijhoff, Dordrecht.*

teat. Applied Animal Behaviour Science, **20**, 275–85.

Dannemann, K., Buchenauer, D. & Fliegner, H. (1985) *The*

Hoekstra, J., Lugt, A.W. van der, Werf, J.H.J. van der &

behaviour of calves under four levels of lighting. Applied Ouweltjes, W. (1994) Genetic and phenotypic parameters

Animal Behaviour Science, **13**, 243–58.

for milk production and fertility traits in up-graded dairy

Dantzer, R., Mormede, P., Bluth, R.M. & Soissons, J. (1983) *cattle. Livestock Production Science*, **40**, 225–32.

The effect of different housing conditions on behavioural

Jury, C., Picard, B. & Geay, Y. (1998) Influences of the method and adrenocortical reactions in veal calves. *Reproduction, of housing bulls on their body composition and muscle fiber*

Nutrition and Development, **23**, 61–74.

types. *Meat Science*, **4**, 457–69.

Dellmeier, G.R., Friend, T.H. & Gbur, E.E. (1985) Compari-

Kenny, F.J. & Tarrant, P.V. (1982) Behaviour of cattle during son of four methods of calf confinement. II. Behaviour.

transport and penning before slaughter. In *Transport of*

Journal of Animal Science, **60**, 1102–109.

Animals Intended for Breeding, Production and Slaughter

Edwards, S.A. (1982) Factors affecting the time to first suck-

(ed. by R. Moss), *Current Topics in Veterinary Medicine and ling in dairy calves*. *Animal Production*, **34**, 339–46.

Animal Science, Vol. 18, pp. 87–102. Martinus Nijhoff, The Edwards, S.A. & Broom, D.M. (1982) Behavioural interac-Hague.

tions of dairy cows with their newborn calves and the effects

Ketelaar-de Lauwere, C.C. & Smits, A.C. (1989) Onderzoek of parity. *Animal Behaviour*, **30**, 525–35.

naar de uit ethologisch oogpunt minimaal gewenste box-

Esslemont, R.J. & Kossaibati, M.A. (1997) Culling in 50 dairy maten voor vleeskalveren met een gewicht van 175 tot 300 kg.

herds in England. *Veterinary Record*, **140**, 36–9.

IMAG Rapport 110. IMAG, Wageningen.

966 • Chapter 55

Ketelaar-de Lauwere, C.C. & Smits, A.C. (1991) *Spatial*

Olsson, S.O., Viring, S., Emanuelsson, U. & Jacobsson, S.O.

requirements of individually housed veal calves of 175 to

(1993) Calf disease and mortality in Swedish dairy herds.

300 kg. In New Trends in Veal Calf Production. Proceedings Acta Veterinaria Scandinavica, 34, 263–9.

of the International Symposium on Veal Calf Production (ed.

Oltenacu, P.A., Frick, A. & Lindhe, B. (1991) *Relationship of* by J.H.M. Metz & C.M. Groenestein), EAAP Publications

fertility to milk yield in Swedish cattle. Journal of Dairy No. 52. Pudoc, Wageningen.

Science, 71, 264–8.

Kiley-Worthington, M. & Plain, S. de la (1983) *The Behaviour* Peeler, E.J., Otte, M.J. & Esslemont, R.J. (1994) *Interrelation-of Beef Suckler Cattle. Birkhäuser Verlag, Basel.*

ships of periparturient diseases in dairy cows. Veterinary Kilgour, R. & Dalton, C. (1984) Livestock Behaviour: A Prac-Record, 134, 129–32.

tical Guide. Granada, London.

Perez, E., Noordhuizen, J.P.T.M., Van Wuijkhuis, L.A. &

Kronfeld, D.S. (1997) *Recombinant bovine somatotropin:*

Stassen, E.N. (1990) *Management factors related to calf*

ethics of communication and animal welfare. Swedish

mortality and mortality rates. *Livestock Production Science, Veterinary Journal*, **49**, 157–65.

25, 79–93.

Ladewig, J. (1984) *The effect of behavioural stress on the*

Peters, A.R. (1986) *Studies of mortality, morbidity and per-*

episodic release and circadian variation of cortisol in bulls.

formance in intensive calf rearing. World Review of Animal In Proceedings of the International Congress on Applied

Production, **22**, 83–8.

Ethology of Farm Animals (ed. by J. Unshelm, G. van Putten Phillips, C.J.C. (1997) *Review article: animal welfare consid-*

& K. Zeeb), pp. 339–42. KJBL, Darmstadt.

erations in future breeding programmes for farm livestock.

Lidfors, L.

(1994) *Mother–young behaviour in cattle.*

Animal Breeding Abstracts, **65**, 645–54.

PhD thesis, Swedish University of Agricultural Science,

Piquet, M., Bruckmaier, R.M. & Blum, J.W. (1993) *Treadmill Skara.*

exercise of calves with different iron supply, husbandry

Lindt, F. & Blum, J.W. (1993) *Physical performance of veal and work load. Journal of Veterinary Medicine A*, **40**, 456–65.

calves during chronic iron deficiency anaemia and after

Plaizier, J.C.B., Lissemore, K.D., Kelton, D. & King, G.J. (1998) *acute iron*

overload. *Journal of Veterinary Medicine A*, **40**, Evaluation of overall reproductive performance of dairy

444–55.

herds. *Journal of Dairy Science*, **81**, 1848–54.

Lyons, D.T., Freeman, A.E. & Kuck, A.L. (1991) Genetics of Pösö, J. & Mäntysaari, E.A. (1996) Genetic relationships

health traits. *Journal of Dairy Science*, **74**, 1092–100.

between reproductive disorders, operational days open and

Mainsant, P. (1996) Les consommateurs de veau en France et

milk yield. *Livestock Production Science*, **46**, 41–8.

en Europe. In *Le veau a l'horizon de l'an 2000*. Le Mans.

Potter, M.J. & Broom, D.M. (1987) The behaviour and welfare Mason, G.J. (1991) Stereotypies: a critical review. *Animal of cows in relation to cubicle house design*. In *Cattle*

Behaviour, **41**, 1015–37.

Housing Systems, Lameness and Behaviour (ed. by H.K.

Mei, J. van der (1987) Health aspects of welfare research in

Wierenga & D.J. Peterse), Current Topics in Veterinary veal calves. In *Welfare Aspects of Housing Systems for Veal Medicine and Animal Science*, Vol. 40, pp. 129–47. *Martinus Calves and Fattening Bulls* (ed. by M.C. Schlichting & D.

Nijhoff, Dordrecht.

Smidt), p. 99. CEC, Luxembourg.

Pryce, J.E., Veerkamp, R.F., Thompson, R., Hill, R.G. & Simm, Metz, J.H.M. (1983) Food competition in cattle. In *Farm*

G. (1997) *Genetic aspects of common health disorders and*

Animal Housing and Welfare (ed. by S.H. Baxter, M.R.

measures of fertility in Holstein Friesian dairy cattle.

Baxter & J.A.C. MacCormack), *Current Topics in Veterinary Animal Science*,
65, 353–60.

Medicine and Animal Science, Vol. 24, pp. 164–70. Martinus Pryce, J.E.,
Esslemont, R.J., Thompson, R., Veerkamp, R.F.,

Nijhoff, The Hague.

Kossaibati, M.A. & Simm, G. (1998) *Estimation of genetic*

Metz, J.H.M. (1984) Regulation of sucking behaviour of
parameters using health, fertility and production data from

calves. In Proceedings of the International Congress on
a management recording system for dairy cattle. Animal

Applied Ethology of Farm Animals, Kiel (ed. by J. Unsholm, Science, **66**, 577–
84.

G. van Putten & K. Zeeb), pp. 70–3. KTBL, Darmstadt.

Putten, G. van & Elshof, W.J. (1978) *Zusatzfütterung von Stroh Morisse, J.P.,*

Cotte, J.P. & Huonnic, D. (1995) L'anémie chez an mastkalber. Aktuelle
Arbeiten zur artgemäßen Tierhal-le veau de boucherie. Evolution maîtrisée on
probleme

tung, **233**, 210–19. KTBL, Darmstadt.

pathologique. Seminaire International sur le Veau de

Riese, G., Klee, G. & Sambraus, H.H. (1977) Das Verhalten

boucherie. A. paraître.

von Kälbern in verschiedenen Haltungsformen. Deutsche

Moser, M., Bruckmaier, R.M. & Blum, J.W. (1994) Iron status, Tierärztliche Wochenschrift, 84, 388–94.

erythropoiesis, meat colour, health status and growth per-

Schlichting, M.C. & Smidt, D. (eds) (1987) Welfare Aspects of formance of veal calves held on and fed straw. Journal of

Housing Systems for Veal Calves and Fattening Bulls. EUR

Veterinary Medicine A, 41, 343–58.

1977 EN. Commission of the European Communities,

Nebel, R.L. & McGilliard, M.L. (1993) Interactions of high Luxembourg.

milk yield and reproductive performance in dairy cows.

Spalding, R.W., Everett, R.W. & Foote, R.H. (1975) Fertility Journal of Dairy Science, 76, 3257–68.

in New York artificially inseminated Holstein herds in dairy

Nielsen, B. (1998) Interspecific comparison of lactational

improvement. Journal of Dairy Science, 58, 718–23.

stress: is the welfare of dairy cows compromised? Proceed-

Stricklin, W.R. & Kautz-Scanavy, C.C. (1984) The role of

ings of the 32nd Congress of the International Society of

behaviour in cattle production: a review of research.

Applied Ethology (ed. by I. Veissier & A. Boissy), p. 80.

Applied Animal Ethology, 11, 359–90.

INRA, Clermont Ferrand.

Studer, E. (1998) A veterinary perspective of on farm evalua-

Welfare • 967

tion of nutrition and reproduction. *Journal of Dairy Science*, Webster, A.J.F., Saville, C., Church, B.M., Gnanasakthy, A. & **81**, 872–6.

Moss, R. (1985a) The effect of different rearing systems on

Trunkfield, H.R., Broom, D.M., Maatje, K., Wierenga, H.K.,

the development of calf behaviour. *British Veterinary*

Lambooy, E. & Kooijman, J. (1991) Effects of housing on

Journal, **141**, 249–64.

responses of veal calves to handling and transport. In New Webster, A.J.F., Saville, C., Church, B.M., Gnanasakthy, A. & Trends in Veal Calf Production. *Proceedings of the Interna-* Moss, R. (1985b) Some effects of different rearing systems

tional Symposium on Veal Calf Production (ed. by J.H.M.

on health, cleanliness and injury in calves. *British Veterinary Mnetz & C.M. Groenestein*), EAAP Publications No. 52.

Journal, **141**, 472–83.

Pudoc, Wageningen.

Webster, J. (1993) *Understanding the Dairy Cow*, 2nd edn.

Uribe, H.A., Kennedy, B.W., Martin, S.W. & Kelton, D.F.

Blackwell, Oxford.

(1995) Genetic parameters for common health disorders of

Wierenga, H.K. (1983) *The influence of space for walking and*

Holsteins. Journal of Dairy Science, **78**, 421–30.

lying in a cubicle system on the behaviour of dairy cattle. In Vries, F.W.K. de, Wierenga, H.K. & Goedegebouwe, S.A.

Farm Animal Housing and Welfare (ed. by S.H. Baxter, M.R.

(1986) Een orienterend onderzoek naar het voorkomen van

Baxter & S.H. MacCormack), Current Topics in Veterinary afwijkingen aan het carpaalgen wrichtbijkleestieren en naar Medicine and Animal Science, Vol. 24, pp. 171–80. Martinus het vereend met de wijze van opstaan en gaan liggen.

Nijhoff, The Hague.

Instituut voor Veeteelkundig Onderzoek 'Schoonoord'.

Wierenga, H.K. (1987) *Behavioural problems in fattening*

IVO Report B-278.

bulls. In Welfare Aspects of Housing Systems for Veal Calves Waltner-Toews, D., Martin, S.W. & Meck, A.H. (1986) Dairy

and Fattening Bulls (ed. by M.C. Schlichting & D. Smidt), calf management, morbidity and mortality in Ontario Hol-pp. 105–22. CEC, Luxembourg.

stein herds. III. Association of management with morbidity.

Wierenga, H.K. & Peterse, D.J. (eds) (1987) *Cattle Housing Preventive Veterinary Medicine*, **4**, 137–58.

Systems. Lameness and Behaviour. Current Topics in Veteri-

Warnick, V.D., Arave, C.V. & Mickelsen, C.H. (1977) Effects nary Medicine and Animal Science, Vol. 40. Martinus

of group, individual and isolated rearing of calves on

Nijhoff, Dordrecht.

weight gain and behaviour. *Journal of Dairy Science*, **60**, Willeberg, P. (1993)
Bovine somatotrophin and clinical

947–53.

mastitis: epidemiological assessment of the welfare risk.

Webster, A.J.F. (1984) *Calf Husbandry, Health and Welfare.*

Livestock Production Science, **36**, 55–66.

Collins, London.

Willeberg, P. (1997) *Epidemiology and animal welfare.*

Webster, A.J.F. & Saville, C. (1981) *Rearing of veal calves. In Epidemiol. Santé
anim. , 31, 3–7.*

Alternatives to Intensive Husbandry Systems, pp. 86–94.

Wilt, J.G. de (1985) *Behaviour and welfare of veal calves in Universities
Federation for Animal Welfare (UFAW),*

relation to husbandry systems. Thesis, Agricultural Univer-London.

sity, Wageningen.

Adult Cattle

Therapy and Disease Prevention

Chapter 56

Health, Housing and Hygiene

D.W.B. Sainsbury

Introduction

971

This chapter is concerned with those underlying princi-

The essentials for good health

971

ples of the management, hygiene and housing of cattle

The size of livestock farms

971

that are essential to assist in the prevention and control

Depopulation

971

of disease.

Group size

972

Isolation facilities

972

Housing types and systems

972

The essentials of good health

Climatic housing

973

Controlled environment house

973

The size of livestock farms

‘Kennel’ accommodation

973

The environmental requirements of cattle

974

From the point of view of animal health, the smaller the

Environmental factors affecting susceptibility to disease

975

livestock unit the better; nevertheless, the current trend,

Intensification and immunity

977

which appears likely to continue, has been towards

Disinfection

977

increasing the size of units to achieve economies of scale

Natural disinfectant agents

977

– in manpower, buildings and services. However, in

Chemical disinfectants

977

dealing with biological material, in this case cattle, there

Disinfectants

979

Recommended procedure in disinfection and

is clear evidence to show that if the farm becomes too

disinfestation of animal buildings and equipment

980

large, efficiency generally decreases and health and pro-

Ventilation

982

ductivity are adversely affected. Economic surveys have

Underlying principles and calculations

982

shown that very small farms as a group are generally less

Natural ventilation

983

efficient but once the three-man unit has been reached,

Mechanical ventilation

984

further improvements in performance are small or non-

Open-fronted yards and kennels

985

existent (Britton & Berkeley Hill, 1975). This knowl-

Dangers from gases in farm buildings

985

edge often goes unheeded and overlarge livestock

enterprises are a cause of great health and management

problems and enormous economic loss. It is, however,

Introduction

pertinent to emphasize that it is the young immature

animal which finds itself at greater risk from disease and

Good health is the birthright of every animal. Con-

mismanagement. Therefore, whilst there should be a

siderable progress has been made towards achieving

limit on unit size at a reasonably low level for the young

improved health in cattle but the disease pattern has

animal, it may be much higher for the adult. The main

changed radically in recent years. The most acute, spe-

reason for this, apart from the extra vulnerability of the

cific, diagnosable and preventable or treatable diseases,

young animal to stress caused by bad management, is

using vaccines, sera and medicines for their control,

the varying immunity to infectious disease among the have been largely controlled. In their place have stock that is unavoidable on a large unit with a mixture emerged more chronic, insidious and complex diseases of animals of different ages and possibly of various not infrequently caused by organisms that are the sources of origin. The adult presents a more stable normal inhabitants of the animal body but which circulate in both respects, being rather more resistant to circumstances allow to become pathogenic. These disease management changes or errors and very much more conditions may be difficult to diagnose, have a large and uniform in its disease resistance or susceptibility. confusing number of causal agents, create a massive morbidity and economic loss and require for their

Depopulation

control great expertise in the exercise of husbandry, hygiene, housing and management skills rather than So far as possible the principle of periodic depopulation recourse to the use of medicines or biological products. tion of a building or, if possible, even a site should be

*followed. The benefits of eliminating the animal hosts
ingly no end to it unless some measures are taken to
to disease-causing agents are well understood and the
ensure a better distribution of the stock.*

*virtue of being able to clean, disinfect and fumigate a
When animals are kept together in large numbers the
building is also accepted. Nevertheless, in practice the
effects of a fright caused by an unusual disturbance can
whole concept of the 'all-in, all-out' policy is more
be extremely serious. It is almost impossible to guard
complex than the preceding sentences would indicate.
against all the extraneous sounds and sights that may
For example, it is in the young animal that it is particu-
adversely affect the stock. The best safeguard, there-
larly important to consider this and far less so in the
fore, is once again to have the animals in small groups
older beast, which has achieved an immunity to many
so that the effect of a panic movement will be more
of the local diseases. Much also depends on whether the*

limited and will never build up into highly dangerous herd or flock is a 'closed' one with few or no incoming proportions.

animals, or an 'open' one with a constant renewal of the animal population. If the latter is the case, then the

Isolation facilities

depopulation principle is of much greater importance as there is little or no opportunity for natural immunity There is a critical need for farmers to give better con- to develop, and there is a constantly running risk, and sideration to the isolation of sick and incoming animals.

usually a near certainty, of introducing animals that are

There are numerous reasons why this should be done.

either clinically infected or carriers of disease. Basic

First and foremost, isolation removes some of the

design specifications for 'open' herds should be quite dangers of contagion to the normal animals; it also different from those of the closed herd.

makes it much more likely that the sick animal will recover without the unwelcome attentions of the other animals who will always tend to act as bullies. In nature,

the sick animal usually separates itself from the rest of

Group size

the herd, but it is often impossible for the housed

One of the principal ways of putting the housing on a

animal to do this. Also, under the more intensive

firm footing is to keep the animals in groups of minimal

systems of management, more animals are likely to

size. At first sight this sounds an old-fashioned and even

suffer from the aggression of their pen-mates and it is

reactionary concept that may eliminate all those advan-

essential that such animals are removed to prevent

tages from automation that large units can give us, but

them from suffering or being killed. It is also easier,

this certainly need not be the case. If groups are small

with an isolated animal, to give it such therapy as it

it is easier to match the animals in them for size, weight

needs and to give it any special environmental condi-

and age. Under these circumstances it is well estab-

tions, such as extra warmth or adjustments to comfort

lished that fighting and bullying are kept to a minimum,

requirements. For new entrants to the farm it will also

if not completely prevented.

enable such tests to be carried out to ensure the animals

There is yet another very important advantage in

are free from infection before they can be safely intro-

keeping animals in small groups. It is obviously good

duced into the herd.

practice for a farmer to keep his livestock somewhere

Many modern livestock units completely ignore the

near the level that has been shown to be the densest

provision of facilities to isolate animals and much

possible for optimal productivity yet still satisfying the

greater attention should be given to this need. It is also

welfare essentials. If livestock are kept in a house at a

an essential part of helping the attendant to do a satis-

high density it is essential that they spread across the

fying job.

house uniformly, so they do in actual practice occupy

and use the whole area. Regrettably, this is very rarely

the case in practical experience and especially so when

Housing types and systems

large numbers are housed together without any subdi-

(see pp. 40–44)

vision at all. If the animals crowd in certain parts of the building there may be grossly overstocked floor areas.

There are three essentially contrasting types of housing:

This is bad enough in itself but it has further unfortu-

‘climatic’, giving only a cover and protection to the

nate side-effects. If they crowd excessively in a part of

animals; ‘controlled environment’, which regulates the

the house, this part may become polluted with dung and

microclimate as completely as is required for the partic-

exhalations to an abnormal and harmful degree;

ular stock being housed; and the ‘kennel’, which is in a

the humidity becomes high, proper air movement is

sense a half-way house between the other two forms and

impeded and the animals may soon become ill. Sick

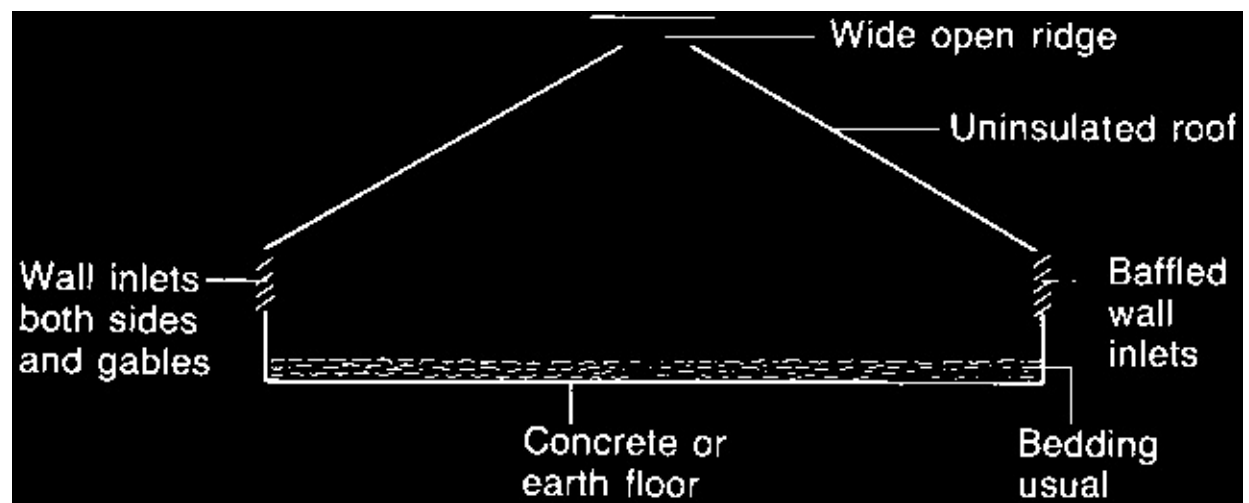
gives two environments in the one building allowing

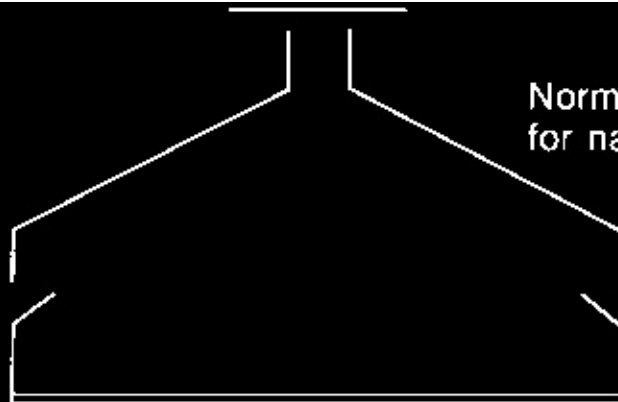
animals feeling cold tend to huddle together even more,

some free and appropriate choice for the animals. The

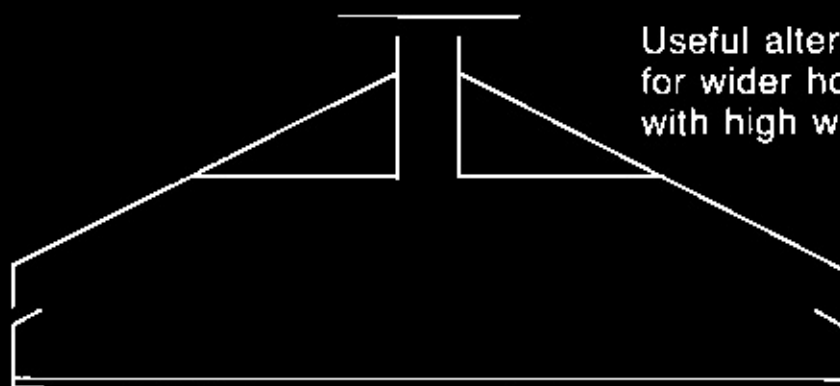
so the vicious circle is perpetuated and there is seem-

methods of use of each of the types and their suitability

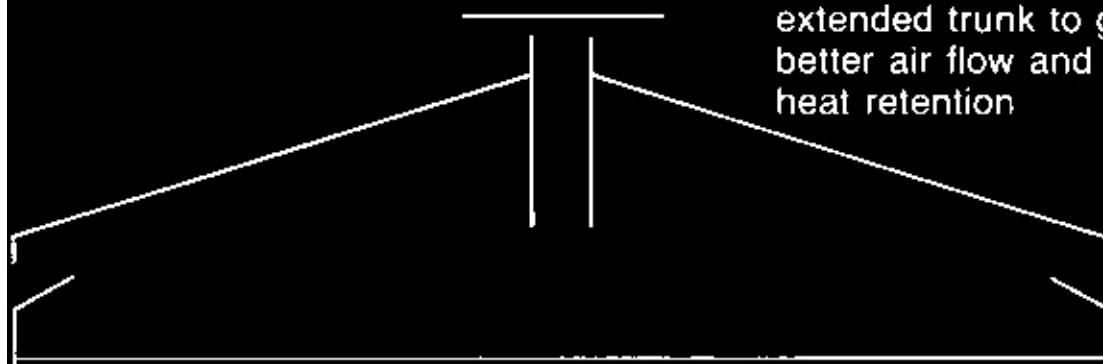




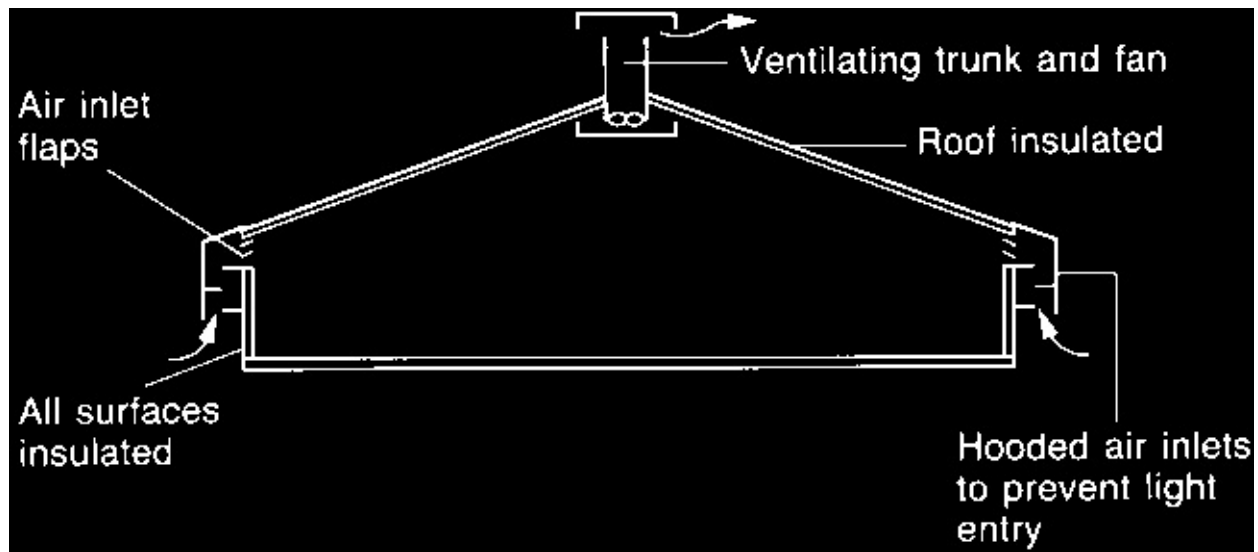
Normal arrangements
for narrow buildings



Useful alternative
for wider houses
with high walls



For wide buildings,
extended trunk to give
better air flow and
heat retention



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Fig. 56.1

Cross-section of 'climatic' livestock house with open ridge and baffled wall ventilators.

Fig. 56.3

Cross-section of a typical conventionally ventilated controlled environment house with roof fan extraction and hooded wall inlets.

In general, stocking densities tend to be half or less those in the controlled environment house. A major problem is created by agriculturists when they attempt to apply the high stocking rates that are suitable for the controlled environment house to the climatic house, as the building is unable to cope with the demands of the

stock and poor productivity and high disease incidence can result. Climatic housing is usually the correct choice for cattle over about six months of age. Such housing usually requires deep bedding for its success, particularly in cooler climatic areas.

Controlled environment house (Fig. 56.3)

The controlled environment house is in great contrast. It has a limited use for cattle, being sometimes used for young calves and to a lesser extent for cattle kept on any bare surfaces, solid or slotted, without bedding. It is most economically viable with livestock that are fed largely concentrate food rather than substantial quantities of roughage, since the former is too expensive to be utilized as a form of energy. The housing is expensive and because of the cost it is usually necessary to

Fig. 56.2

Alternative arrangements suitable for natural ventilation stock the buildings as densely as possible to make them
tion of various widths of buildings and using outlet ('chimney') viable economically, and this can place a great strain on
trunks.

health control. To cope with the special requirements, management needs to be of the highest standards and unless these criteria can be observed it is the wrong type for different countries, climatic regions and forms of live-of housing to have. The rewards can be great but the stock vary enormously and must be carefully defined. dangers are greater.

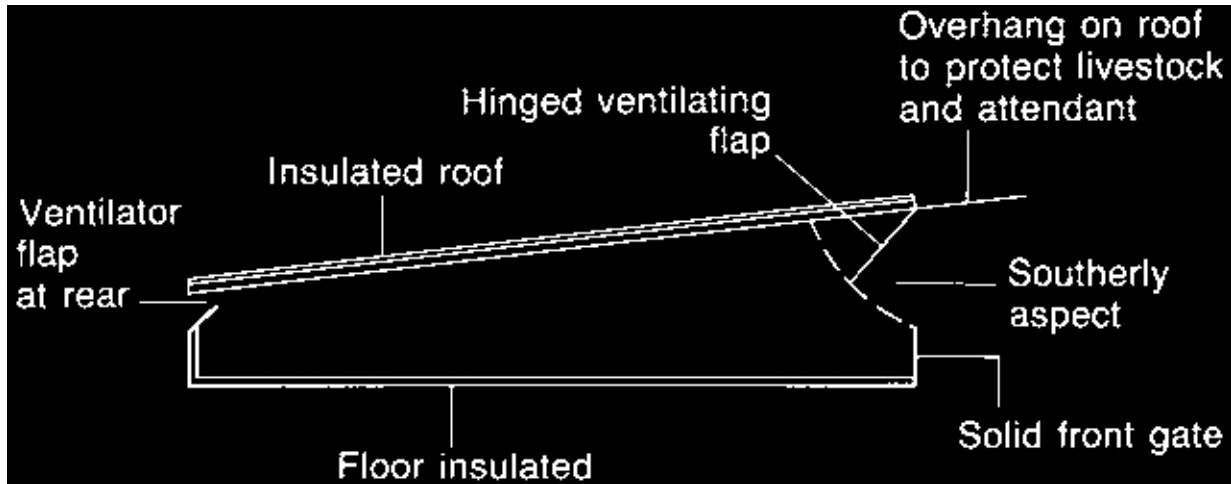
Climatic housing (Figs 56.1 and 56.2)

‘Kennel’ accommodation (Fig. 56.4)

The climatic house is ideal for the adult beast that has This is an increasingly popular system of accommoda-developed a large measure of adaptability to climatic tion that is in a sense half-way between the climatic stress. The house can be cheap because it is basically a and controlled environment housing. It attempts to cover alone but because of the lack of control of the combine the virtues of both, but at low cost, and often climate the space given to the animals must be much succeeds. The essence of the system is that the animals greater, especially since there usually is no powered are kept in pens or groups that are sufficiently small to

ventilation.

allow them to be closely confined, at least while resting,



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Table 56.1

*A guide to the minimal space allowance for cattle
at different stages of growth in strawed yards.*

Age

Area/animal (m²)

Up to 14 days

0.9–1.4

14 days to 3 months

1.4–1.8

3–6 months

1.8–2.4

6–9 months

2.4–2.7

Fig. 56.4

Cross-section of a mono-pitch ('kennel') naturally

9–18 months

2.7–3.8

ventilated stock building.

Adults

small

5

large

7

without too great a danger from respiratory or other disease. The close confinement makes it possible to keep the groups warm and draught-free by utilizing their own body heat and by good insulation of the embrace the effluent from the animals, which is not only kennel and a limited cubic air space. This part of the a considerable expense but is also likely to intensify the accommodation is a simple form of controlled environment disease risk.

ment house: the rest is the climatic house and can be

Whatever the system, the principal practical faults in justified as this is an area where the animals will be livestock housing systems are as follows:

moving about freely and will not normally be inactive

- *Having too close a confinement of the animals.*

or lying down. It is likely to contain the dung and often

- *Placing too many animals in one common air space.*

the feeding and watering arrangements and so must be

- *Reducing both cubic area and floor space.*

very adequately ventilated, usually by non-mechanical

- *Failing to remove muck from close proximity to the means. In harsh climatic areas it is covered; in milder animals.*

regions there is no need for this and the 'yard' can, with

- *Transferring muck through pens, aiding the transfer benefit to health and productivity, be left uncovered. of disease.*

It is instructive to examine the logic of the 'kennel'

- *Failing to separate age groups.*

system.

- *Neglecting clean feeding and water arrangements.*
- *The cost of the housing can be as low as any system,*
- *Having poor or sometimes no drainage.*

especially with uncovered yarding, and such build-

- *Neglecting the comfort of the stock.*

ings are also the easiest type of building for the

- *Inefficient ventilation systems.*

farmer himself to erect.

Table 56.1 shows the space allowances for cattle in

- *Good health is promoted by separation of the
strawed yards.*

animals into small groups. The separation of the

kennels one from another should be as absolute as

possible as this will limit the build-up and spread of

The environmental requirements

respiratory disease. It will also be a great assistance

of cattle

to the health of the animals if the muck is not

allowed to accumulate in the warmth or the closely

In most cool, temperate environments, ruminants main-

confined resting area. It is often possible as well, by

tain a stable body temperature by regulating evapora-
good design, to keep the dunging areas separate so
tive loss, at little metabolic cost. Ruminants have a very
that the muck from different pens does not come
marked ability to alter their zone of thermal neutrality
together until after it is out of reach of the animals.

in response to previous thermal history. Thus there are

- The cheapness of the housing is largely due to the
no absolute criteria for the thermal requirements of any
fact that the environment is controlled only where
class of ruminant livestock, except perhaps the new-
it is absolutely necessary.

born animal; they depend to a large extent on what the

It is certainly a distinct virtue if the dung can be in
animal has grown accustomed to.

an area where the air is moving more freely and the

Some examples of the lower critical temperatures for
temperature and the humidity possibly much lower,
cattle under conditions of varying air movements are
at least in temperate climates. Feeding in an outside
shown in Table 56.2. This table illustrates a situation

yard achieves this objective. Controlled environment where the beasts are standing up, in a dry enclosure, housing is becoming more and more expensive and under two variations in air movement (Webster, 1981).

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Table 56.2

Lower critical temperature of ruminants housed in depends as much on the thermal properties of the floor a building with very low air movement (0.2 m/s) and in a draught as on the temperature.

(2 m/s). Source: Webster (1981).

It is worth stressing that European cattle tend to be tolerant to cold but intolerant to heat. Their ‘comfort

Type of animal

Liveweight

Lower critical

zone’ is between about 0 and 20°C. The critical tem-

(kg)

temperature (°C)

perature leading to a decline in milk yield at the higher

level is approximately 21–25°C for most European

Low air speed

Draught

cattle, including Friesians and Jerseys, but as high as

Calf

30–32°C with Brown Swiss (see Chapter 8).

Newborn

35

+9

+17

The effect of high temperatures may be ameliorated

1 month old

50

0

+9

in practice in a number of ways. The provision of shade

Veal

100

-14

-1

makes a great difference, also of wallows and artificial

Store cattle

250

-32

-20

*showers. For example, great benefit has been found in
(maintenance)*

*tropical climates with cattle kept under a so-called
Beef cow*

450

-17

-9

*desert-cooler consisting of a three-sided shelter open to
(maintenance)*

the north, with an upper roof of aluminium, which

Dairy cow

500

-26

-13

*reflects the heat rays, a ceiling underneath of three
layers of old hay and an evaporative cooler under the
roof at the south end of the shelter. There are a number
In an unheated building at low air movement the only*

of highly effective methods of reducing heat stress by cattle likely to experience cold sufficient to elevate the use of water atomizers. In some cases atomizing heat production are newborn calves or young calves units are suspended under the roof of the buildings whose metabolic rate is low by virtue of starvation, sickness or emaciation. Such animals can undoubtedly be of mist propagators above the stock connected by a stressed by cold and may require special attention. pipe-line carrying the water. Provided the humidity of Increasing air movement to 2.0 m/s, which is not uncommon in draughty buildings, increases the critical temperature effects can be achieved at a modest running cost (see Chapter 8). True refrigeration plants are very occasionally used but are rarely economic. At high temperatures ventilation for calf houses is achieved without allowing farm stock also attempt to reduce their heat production

excess air movement (or 'draught') to impinge on the by reduced feed intake. This leads to less production calves.

and often also to metabolic and fertility problems.

By one month of age the healthy calf with a good

Under hot sunny conditions the temperate breeds, par-

appetite and access to an adequate feed source has a

ticularly, spend less time in grazing and, in fact, under

critical temperature close to 0°C and is not likely to be

these conditions most of the grazing will be done at

stressed by cold while indoors. Well-grown veal calves,

night. There are very marked breed differences, as

by virtue of their very high energy intake and heat pro-

might be expected, the tropical breeds being least

duction, are particularly tolerant to cold and by the

affected.

same criteria sensitive to heat. The traditional belief

In loose housing systems with open fronted barns,

that veal calves should be kept in a warm environment

protection from solar radiation and maximum provision

because they are not ruminants and are therefore

*of air movement represents the limit of environmental
somehow more sensitive to cold is unscientific and
improvement. With closed buildings, such as the cow-
untrue.*

*house, mechanical air cooling may be used and, in parts
No other class of cattle is likely to experience a sys-
of the world where climatic temperatures warrant it, has
temic stress of cold when standing up in a dry enclosure
been found worthwhile. For a comprehensive consider-
unless air movement is exceptionally high. However,
ation of the effects of the environment on cattle the
problems do arise in damp conditions. For the dairy
reader is referred to Clark (1981).*

*cow, however, cold stress should not be considered as a
systemic but as a local problem. Heat production in the
high-yielding cow is again very high and so the critical*

Environmental factors affecting

*temperature is low. Milk synthesis, however, depends
susceptibility to disease*

*on blood flow to the mammary gland, which is reduced
by local cooling. The production of dairy cows has*

*Many of the organisms that cause ill-health in intensive
been shown to fall at temperatures below about 0°C,
livestock units are normally present in the animals and
although it is obvious that direct chilling of the udder
their surroundings and it is environmental stress that*

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*acts as a trigger, inducing their multiplication to such an
humidity. Rhinoviruses appear to survive better at high
extent that clinically obvious disease breaks out. There
humidities but infectious bovine rhinotracheitis (IBR)
are numerous examples, such as respiratory diseases,
and parainfluenza 3 (PI3) viruses at low humidities. In
diarrhoea and mastitis. Whilst these diseases may
these last examples the differences in survival time
always become clinically obvious in the long term, there
reflect the association between seasonal patterns of
may be a lengthy period beforehand when their effects
relative humidity and disease incidence. There is the
are limited to poor growth or productivity and an inef-
further possibility that at high relative humidity there
ficient conversion of feed by the animal. It is an essen-*

is accelerated sedimentation of airborne pathogens in
tial feature of a farmer's recording system that records
large aerosols. Mycoplasma, on the other hand, tends to of productivity are kept
which should allow the detec-be stable at very low and at high relative
humidities but
tion of results that are beginning to deteriorate, and this
sensitive to mid range relative humidity.
can well be a warning that urgent action is needed to
So far as bacteria are concerned, a rise in tempera-
stop a slide in results leading eventually to disease.
ture produces an increasing destruction of bacteria but
Most of the conditions of this nature are insidious,
airborne spores are highly resistant. Escherichia coli
chronic and difficult to diagnose by any of the popular
survives and multiplies best at about 15°C, as also does
methods that are so effective with the well-described
Mycoplasma. Bacteria tend to be resistant to low and
diseases. Very limited use can be made of vaccinations
high relative humidity but sensitive to mid range rela-
or antisera, nor are antibiotics or drugs more than a
tive humidity; bacterial spores, however, are resistant
short-term answer. The only effective way is to attend

almost totally to relative humidity effects.

to the whole environment of the animal by such

It is important to be aware that in airborne infections

methods that are known to reduce the risk of disease.

the size of the infecting dose will determine whether or

It is extremely difficult to reproduce clinical disease

not disease will result. Also, its severity and the likeli-

by experimental infections even when huge doses of

hood of infection making headway will depend on the

mixed organisms are used. Whether or not an animal

animal's overall resistance; for example, chilling can

becomes clinically affected depends on non-specific

markedly lower an animal's resistance to inhaled

factors of climate, housing and husbandry, which

pathogens by depressing lung clearance mechanisms

together form the total environment that is considered

(Whittlestone, 1976).

in this chapter. Above all it should be made clear that

Systems in which the air is recirculated are being

the state of health of the animals can transcend all other

favoured as a means of reducing heating costs, but in

factors in determining the economic viability of a live-stock enterprise. Disease at its worst kills the animal, but even in sublethal infections seriously affects productivity. It is known from the work of Honey and McQuitty (1976) that animal buildings are often very dusty, but the factors influencing the dispersal, survival and deposition of airborne pathogens in farm animals have been reviewed comprehensively by Donaldson (1978). Small changes in relative humidity have been found to have significant effects on settled dust; lower humidities result in more settled dust. There is, however, mounting concern about the deleterious effect of dust on live-stock health, and good systems of ventilation should be the animals' breath and also from secretions and excre-

but even in sublethal infections seriously affects

It is known from the work of Honey and McQuitty productivity.

(1976) that animal buildings are often very dusty, but

The factors influencing the dispersal, survival and

the factors affecting dust production are ill defined.

deposition of airborne pathogens in farm animals have

Small changes in relative humidity have been found to

been reviewed comprehensively by Donaldson (1978).

have significant effects on settled dust; lower humidities

Droplet nuclei are the primary mode of spread of a

result in more settled dust. There is, however, mounting

variety of contagious diseases and many pathogens

concern about the deleterious effect of dust on live-

have been shown to have been spread in this way via

stock health, and good systems of ventilation should be

the animals' breath and also from secretions and excre-

able to remove it from the building in the same way and
tions of the infected animals (see p. 242).

as efficiently as other environmental pollutants.

Referring specifically to virus particles, the ambient

The disposal of large accumulations of animal excre-

temperature has little effect on their survival in an

ment, bedding and litter is a further problem and can,

animal house but a general trend is that high tempera-

in some instances, be the limiting factor in determining

tures are more harmful to their survival than low ones

the size of unit. It is common practice to dilute the efflu-

(Sanger, 1969). In fact it has been suggested that the

ent from farm buildings into a slurry and then spray it

inactivation of viruses immediately after exhalation and

over farmland as a fertilizer. The hazard from the dis-

aerosol formation is dependent on the relative humid-

semination of slurry-associated pathogens over a wide

ity and any temperature effect occurs secondarily

area in this way can be considerable, particularly in

(Donaldson & Ferris, 1976). The effect of relative

windy weather (Wray, 1975). A dairy herd of 50 cows

humidity on viruses in general is that viruses with a causes a potential pollution load as large as that of a lipoprotein envelope survive best at a low relative village of over 500 people, yet frequently the manure is humidity, whereas non-enveloped viruses are unstable carelessly disposed of without thought of its potential in dry conditions but survive best at a high relative dangers to man or animal.

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Intensification and immunity

Natural disinfectant agents

There are some underlying truths in connection with Most pathogenic micro-organisms do not survive long disease and immunity that need to be explained as they outside the animal body, but unfortunately sufficient have a close bearing on the incidence of disease and its may always remain to cause renewed infection. Some relationship to the environment.

vegetative bacteria and viruses can live several months

An animal at birth has a degree of passive immunity if protected with organic matter and the spores of bac-

to local disease that is passed from the dam to the offspring. Bacteria can live almost indefinitely in the soil or protected spring, partly in utero and partly from the colostrum or in cracks and crevices of the building. For example, first milk. Such immunity is, however, only practically Clostridium tetani and Bacillus anthracis can live for effective to local infections to which the dam has been many years. Even coccidial oocysts may survive for challenged. If the birth takes place in a 'foreign' environment years in infected quarters. environment, then the young may have little or no passive immunity. The factors contributing towards natural destruction of immunity to the infective organisms in their new local environment of microbes are nevertheless important as they do environment. In many cases the young are housed reduce the numbers and this in itself is a worthwhile aid during the early stages of life in such a 'foreign' environment to the artificial processes. Sunlight, heat, cold, desiccation and it is therefore most important that the ventilation and agitation all contribute. Sunlight is the most challenge of disease-causing organism is reduced to a potent and its powers of destruction are enormous; its

minimum.

*efficiency is entirely due to the ultraviolet range of
Perhaps the first and foremost methods for reducing
wavelengths. Unfortunately, these ultraviolet rays have
this challenge are by the processes of depopulation,
little penetrating power and cannot pass through glass
cleansing and disinfection. This can obviously be under-
or translucent roofing sheets or through clouds of
stood to be most important in the case of the younger
industrial haze; the value of sunlight in animal buildings
animal, which has a poor or uncertain resistance to
is therefore wholly unreliable. Desiccation, from fresh
disease. Whilst it can never be said to be other than
air and wind, will also contribute to the destruction, par-
advantageous, it is most important on sites that have a
ticularly when the micro-organisms are exposed to this
large population of animals in an area where there are
by prior cleansing of the building.*

*substantial numbers of animals since many diseases,
Another process is antibiosis; many bacteria and
especially viral ones, can pass a great distance by the*

fungi produce substances that are antagonistic to other airborne route. Whilst small infective particles can be organisms. Penicillin and streptomycin are agents of carried directly by dust particles moved by the wind, this nature whose antibacterial action is well known. In other pathogens are carried indirectly by vectors, especially the soil, in floors and buildings generally, pathogenic cially birds.

organisms will be acted upon by antibiotics produced by non-pathogenic organisms that are normal inhabitants of the soil. Warm moist conditions will assist the action of such saprophytic agents.

Disinfection

The action of heat

The disinfection of a building implies the elimination from the house of all micro-organisms that are capable For many years heat has been used for disinfection, dry of causing disease, thus converting the place from a heat being used with the 'flame-gun' and moist heat in potentially infective state into one that is largely free the form of the 'steam-jenny', but both tend to be

from infection. A disinfectant is the agent that is inexact and uncontrolled methods of applying the capable of achieving this and in livestock farming it is agents.

usually a chemical agent.

Many bacterial spores can easily survive the transi-

The processes of disinfection of a building can take tory attention of the heat source and pathogenic organ- place by the action of nature (natural disinfection) and isms may readily be protected from the heat in cracks by artificial means (artificial disinfection). Before dis- and crevices of the building.

cussing these processes in some detail, it should be emphasized at the outset that 'cleansing' is an essential

Chemical disinfectants

preliminary to disinfection. Organic matter has the ability considerably to reduce the effectiveness of

Disinfection on the farm is generally carried out by disinfectants, so without good cleansing the action of using chemical agents. The lethal action of disinfectants disinfectants is often readily diluted.

is due in the main to their ability to react with the

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protein and, in particular, the essential enzymes of greater than 120°C over long periods are needed to micro-organisms. Therefore, any agents that will coagulate, precipitate or otherwise denature proteins will to common sterilants and other chemical agents is act as general disinfectants. Among these agents are very high, as is resistance to extremes of pH and to phenols, alcohols, acids, alkalis, aldehydes, halogens, ultraviolet or ionizing irradiation. The only chemical chloramines and quaternary ammonium compounds, treatment so far found to be effective against all as well as heat and certain radiations.

transmissible spongiform encephalopathies is sodium hypochlorite at 20 000 parts per million of available chlorine, for about one hour (see p. 909).

Selective action

Many disinfectants have a selective action on different

Dynamics of disinfection

types of microbes. For example, Gram-positive and Gram-negative bacteria differ in the structure of their membranes, the latter being of a more complex nature. Disinfection is not an instantaneous process; it takes place gradually. However, many more microbes are killed at the beginning of disinfection than at the end, as every protein has its own characteristic isoelectric point, each responds individually and will be influenced by the acidity or alkalinity of the disinfectant; for example, fungi are extremely acid-resistant, whereas viruses tend to be more susceptible to acid disinfectants, high humidity or excessive organic matter. An examination of the number of organisms surviving at different stages during disinfection shows that the number of

so quick that it goes undetected. The action of most two groups, lipophilic and hydrophilic. Lipophilic disinfectants is increased, often quite markedly as the viruses have lipid envelopes, which make them sensitive temperature rises.

to the majority of disinfectants. Most of the animal pathogenic viruses are in this group. Hydrophilic

The effect of organic matter

viruses are enveloped and are less sensitive to disinfect-

Almost invariably when disinfection is carried out on

tants in general. Some very important viruses are in this

the farm, organic matter will be present. It can be said

group and these have a tendency to become danger-

that organic matter always interferes with the action of

ously persistent on farms and in animal buildings; exam-

the disinfectant and may do so in the following ways:

ples are the enteroviruses and reoviruses. Against this

latter group quarternary ammonium compounds have

(1)

The organic matter may protect the cell by

a poor effect, but are very good if associated with

*forming a coating on it and preventing the ready
formaldehyde, gluteraldehyde, chloramines and certain
access of the disinfectant.*

organic acids.

(2)

*The disinfectant may form an insoluble compound
with the organic matter to remove it from poten-
tial activity.*

Disinfection of prions

(3)

*The disinfectant may react chemically with the
The various infective prions, such as those causing
organic matter giving rise to a non-germicidal
bovine spongiform encephalopathy or scrapie, have a
reaction product.*

number of common characteristics. They are extremely

(4)

*Particulate and colloidal matter in suspension may
resistant to heat and, whilst some loss of infectivity
absorb the antibacterial agent, so that it is sub-
occurs at temperatures above 100°C, temperatures*

stantially removed from solution.

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(5)

Fats, etc. in serum and milk may deactivate the

Formaldehyde and glutaraldehyde

disinfectant.

Formaldehyde is a widely used disinfectant in both the

gaseous and the aqueous forms, being bactericidal,

Approval of disinfectants

virucidal and fungicidal. In aqueous solutions, formalin,

which is a solution of formaldehyde gas containing 40 g

There is no completely satisfactory method of assessing

of formaldehyde in 100 ml of the solution, is widely used

or standardizing disinfectants and an enormous number

at 5 per cent strength as a general disinfectant, but it

of different techniques have been used in the past.

does need to be in contact with the surface for some

There is also no universally effective disinfectant, so

time to be effective. Its action is greatly affected by tem-

that different disinfectants are required for different

perature (i.e. it has a high temperature coefficient) and

purposes.

the warmer it is the better, blood heat being most sat-

*In the UK, approvals for disinfectants are under five
isfactory. It also acts more efficiently when the surface
headings, as follows:*

*is wet. In agricultural use formalin is largely used as a
Group 1A are active against foot-and-mouth disease;
gas or an aerosol spray.*

Group 1B are active against swine vesicular disease;

A very active complex disinfectant, which is useful

Group II are active against fowl pest;

against the most persistent viruses, is one containing

Group III are active against tuberculosis;

formaldehyde, glutaraldehyde and a quarternary

Group IV are for general farm use, the test organism

*ammonium compound. This is active at much lower
being *Salmonella cholerae suis*.*

temperatures than formaldehyde.

*The official test lays down a rigid procedure for spe-
cified reduction of the organisms in the presence of*

Quarternary ammonium compounds

organic matter. The most recent lists of approved disinfectants list over 300 of them for livestock use.

These are cationic neutral detergents available as aqueous solutions, powders or pastes. They are effective against a wide range of bacteria and moulds and have

Disinfectants

a high surface activity. Generally, when dissolved in water, they have a high wetting power and the ions

Phenols, cresols and related compounds

adhere to the surfaces, giving a long-lasting residual

The phenols, cresols and related compounds are an effect. They also have low toxicity and lack odour or important group of disinfectants that were once the taste. Combined with other disinfectants they can give most popular of all such agents.

a wide spectrum of activity.

All phenols can act bactericidally or fungicidally but generally are neither sporicidal nor particularly viruci-

Oxidizing disinfectants

dal. They have high dilution coefficients, i.e. small concentration changes give rise to large differences in their

Hydrogen peroxide and other oxidizing agents include killing rates. They are always more active as acid solutions. Peracetic acid and propionic acid are emerging as alternatives. Organic matters can severely interfere with their increasingly popular disinfectants. At quite low concentrations they are active against bacteria, their spores, reduce their effectiveness to 10 per cent. There are viruses and fungi. Several new proprietary forms are many proprietary 'coal tar disinfectants', phenolic in marketed for use in livestock buildings.

nature, and in view of their inevitable low solubility in water they are either solubilized or emulsified. The

The halogens

solubilized types, known as 'black fluids', are those in which the phenol fraction is dissolved in a soap base

In agriculture, chlorine is widely used for disinfecting

and the emulsified types, known as 'white fluids', are

water and in the cleansing of dairy and other farm

those in which the phenol is emulsified into a perma-

equipment. Chlorine-releasing compounds containing

nent suspension with the aid of gelatin or dextrin.

up to 90 per cent available chlorine are available and

In more recent years a number of synthetic phenols have an important use in the formulation of bactericidal detergent powders of the alkaline variety. Products o-phenylphenol or one of the other diphenyl derivatives of this type are suitable for use in the washing down of an animal house. The inorganic chloramines, being a coal tar disinfectants, are non-toxic, non-irritant and concentration of chlorine and ammonia, are used in have a more pleasant odour, arising chiefly from their water and sewage treatment; organic chloramines, such purer contents.

as halazone, are excellent water sanitizers.

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The normal use of chlorine in cleaning milking and dimethyl- O-2,3,5-trichlorophenyl phosphorothioate other utensils is to use a detergent wash with a solution (fenchlorphos 12 per cent) (see Chapter 60).

containing 250–300 ppm of available chlorine followed by a rinse with a weaker chlorine solution. Failure of Health and safety

the chlorine disinfectant is more likely due to the presence of organic matter, which does seriously interfere

There are certain dangers to people and mammals in with its action, so that it is not normally used where the use and disposal of disinfectants and ectoparasiticide much dirt is present. In favourable circumstances it is cides and the residues of their applications. For this best used warm and will act quickly. Chloramines are reason alone it is advised that only officially approved much more active in the presence of organic matter and products should be used and the health and safety have found a secure place as general disinfectants.

regulations attached to them are followed rigidly. An awareness of the possible harm to those using the substances and their effect on polluting the environment

Iodine and iodophors

has increased immensely lately so that the manufacture

Iodine is an effective germicide, although like chlorine

turers have been forced to update the health and safety
its action is much depressed by organic matter. It is
data regularly. There are particular dangers with some
effective against vegetative organisms and spores. The
ectoparasitocides as well as certain disinfectants and it
most widely used iodine preparations are the
is vital that protective clothing is worn.

iodophores in which iodine is mixed with surface-active
agents, which act as carriers or solubilizers for the

Rotation of hygiene products

iodine. They lack any real odour and are not irritant.

Iodophors have been used at dilutions of around

There are no products that are ideal. All of the dis-

25 ppm available iodine for utensils. They have built-in

infectants and ectoparasitocides have their strengths

indicators that reveal quickly the approximate concen-

and weaknesses. To minimize the adverse effects of this

tration of the use dilution; a yellow tinge or pale amber

fact, it is recommended that a rotational programme is

colour is imparted by an iodophor even when only a few

instituted so that regular programmed changes are

parts per million of free iodine are present in the solution made in the type of product used, thereby reducing the chance of certain pathogens escaping destruction.

or brown to the solution. The use dilution of 25 ppm is equivalent to available chlorine concentrations up to

Recommended procedure in disinfection

200 ppm. Iodophors are an ideal germicide to use in

and disinfection of animal buildings

water utensils.

and equipment

Basically, two procedures should be adopted. The first

Ammonia

is used between batches of livestock within a building

A 10 per cent aqueous solution of ammonia is an effective

in the absence of overt disease, and the second after an

tive agent for the destruction of coccidial oocysts. This

outbreak of a contagious and infectious disease.

is the only use for ammonia as a disinfectant.

Procedure of disinfection with no disease present

Ectoparasites

(1)

All equipment and fittings that are removable

It is of great importance to control ectoparasites in and should be demounted and taken out of the build-

around the farm buildings. They are a nuisance both to ing. It is advisable for them to be soaked in a bath

the attendants and the animals, may be the cause of of disinfectant where the materials are able to

direct irritation and disease and may indirectly spread stand up to this treatment. Alternatively, they

disease; indeed any infectious agent can be carried by may be power-sprayed or steam sterilized. They

ectoparasitic vectors. These ectoparasites include flies should be left outside exposed to the elements and

(the ordinary housefly, the lesser housefly and the away from animals and their products for as long

stable fly), mites, ticks, lice, fleas, bugs, beetles and as possible.

cockroaches.

(2)

All litter should be removed (Fig. 56.5).

*For the control of external parasites of animals a
(3)*

*The roof and structural elements of the house
variety of compounds are available, such as gamma-
should be dusted and cleaned, preferably with a
benzene hexachloride (lindane), piperoxyl-butoxide
vacuum cleaner.*

pyrethrum (pybuthrin) or O, O-dimethyldithiophosphate (4)

*The lower part of the walls and all the floors
of diethylmercaptosuccinate (malathion) and O, O-
should be thoroughly cleaned, preferably using a*





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Fig. 56.5

*Cleaning out the litter after
removal of the animals.*

Fig. 56.6

*Washing down with a pressure
hose using a detergent disinfectant.*

*pressure washer, with a heavy duty, wide-spectrum
Procedure after contagious and infectious disease
detergent/disinfectant mixture (Fig. 56.6).*

(5)

The surfaces must then have a disinfectant applied

(1)

The building should be closed and isolated from

(Fig. 56.7) with a wide spectrum of activity,

all visitors.

capable of killing all the pathogens that are likely

(2)

The bedding, litter and all areas in intimate

to be present.

contact with the stock should be sprayed with a

(6)

In some cases, if there is a heavy insect infestation,

strong disinfectant.

it will be necessary to apply a special insecticide

(3)

The litter should subsequently be removed from

spray.

the building and may be burnt or buried so there

(7)

As a final measure a disinfectant is often sprayed

is no possible contact with livestock.

over all the surfaces to give a residual effect.

(4)

Portable equipment and fittings should be given

Thermal ‘fogging’ machines are widely used.

the same treatment as previously suggested and



Fig. 56.7

Spraying with a disinfectant.

preferably in the house, later to be taken out and aerated.

(5)

The floors and lower part of the walls should be cleaned with detergent disinfectant.

(6)

The house should then be treated in the same way as suggested in the previous section.

(7)

It may sometimes be advisable to skim off the top few inches of the soil around a heavily infected area.

(8)

The approaches to the building should be treated with disinfectant and foot-dips provided (Fig. 56.8).

Ventilation

There are few topics in animal husbandry where there have been more totally erroneous statements made

than on the reasons why housed livestock require ventilation or 'air-change'. For example, for many years it was accepted that the main deleterious effect of having too little ventilation was due to the build-up of harmful concentrations of the gaseous products of respiration, and in particular carbon dioxide, combined with a depletion in the oxygen content of the air. It is now known that this is far too simple and usually quite an incorrect hypothesis.

Underlying principles and calculations

In any badly underventilated building the stagnant air gradually becomes both warmer and more humid and

Fig. 56.8

Using protective clothing and dipping boots in disinfectant there is a rising concentration of dust, other particulate infectant to reduce the risk of introducing infection.

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matter, ammonia and other gases and any pathogenic microorganisms the livestock may be carrying. The end climate accommodations far more control is needed

result is that the animals suffer from 'heat stagnation', and down-draughts, which can be common with the involving a susceptibility to chilling. Along with this will fixed open ridge, must be avoided. Experience has go poor productivity and a likelihood of disease, par-shown that for the latter, good results are obtained by ticularly respiratory, which modern experience shows to the use of a limited area of controlled outlet ventilation be of the greatest danger in intensive forms of housing. and for this purpose a simple chimney type or insulated In addition, in a badly ventilated building, condensa-'flue' is satisfactory. It is good practice to have one or a tion on the surface is often likely and bedding and floors few outlets, but a much larger number of smaller air may become wet and the animals uncomfortable. This inlets, as the fresh air should come in slowly all round will tend to exacerbate the effect of disease conditions, the building, diffusely and carefully baffled. The basis respiratory or enteric in nature, or infections such as of a useful inlet system consists of hopper-type mastitis.

windows, fitted with gussets to prevent direct draughts, In recent years a new hazard to livestock and their serving as principal inlets, and small baffled openings attendants has been the risk of gaseous intoxication of between the windows which alone are left open during livestock due to the gases arising from slurry pits or cold or windy weather. Since cold air coming into the channels under the animals. The risk is very great and building falls naturally towards the floor, there is no has caused the death of many animals and several necessity to site inlets at floor level, where the danger stockworkers.

of chilling draught is considerable.

The object of ventilation, therefore, is to remove the stale air in a building and replace it with fresh air and

The totally covered yard

remove any danger of toxic gases. But while too little ventilation is obviously serious, so also is too much. In

In practice the following major faults are commonly cold weather much valuable animal heat will be wasted.

found in the construction of covered yards:

Further, overventilation may be synonymous with

- *Either there is no ridge outlet for stale air or it is draught. Draughts are themselves responsible for inadequate.*

causing many deaths by direct chilling and they may

- *Eaves openings are either absent or, where present, also act indirectly by lowering the animals' resistance uncontrollable and at the mercy of the weather, to disease-producing organisms.*

causing chilling draughts in cold weather.

In planning a ventilation system allowance has to be

- *The extreme width of many yards (20m upwards) made for all the factors that can influence the ventilation prevents natural ventilation functioning properly.*

tion required, i.e. type, age and number of livestock,

This problem is often combined with multispan

their management, the construction and locality of the

construction and low eaves and roofs, so that the

house and finally the weather conditions.

flow is generally impaired.

- *The gable ends are closed and sealed.*

Natural ventilation (see p. 40)

- *There is no insulation of the roof. While insulation is not usually necessary it may be a desirable 'extra'*

If natural ventilation is to be effective, it must function under a few circumstances, particularly where well in all weathers. To try to avoid too great a dependence on external wind and weather, it is best to make and natural air flow is difficult.

use of the so-called 'stack effect' as its foundation. The

- *Poor drainage under or around the yard leads to warm stale air around the body of the animal, being excessive straw usage and contributes to the humidity lighter than cold fresh air, will rise to the top of the of the atmosphere.*

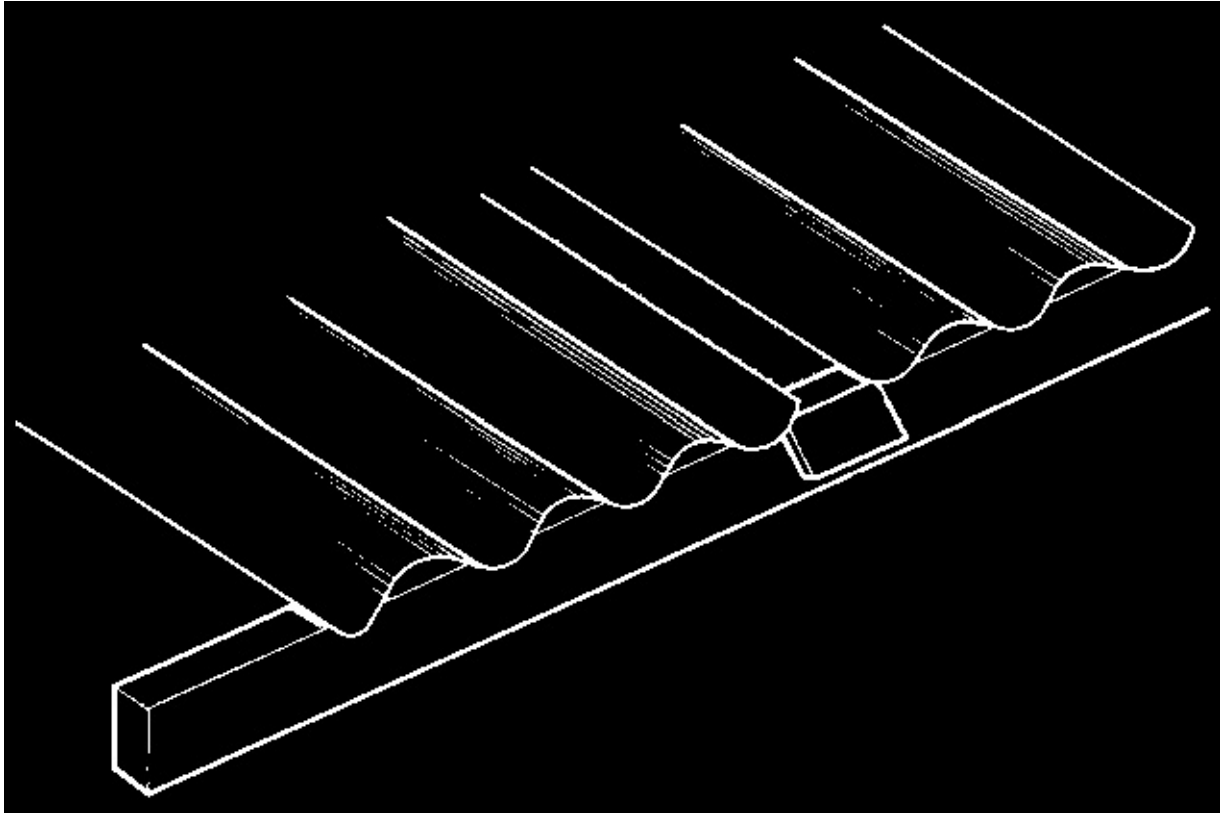
building. Outlet ventilation is therefore provided at a

- *Bad construction of the walls leads to excessive suitable point or points in the ceiling or roof. The moisture penetration at all points.*

greater the difference between inside and outside temperatures, and the greater the distance between inlets

Natural ventilation works most satisfactorily on open and outlets, the greater the stack effect. Therefore, if an sites; it is always useful to have it controllable, but this area of outlet ventilation is provided to cope with diffi- is not usually done. An open ridge 300 mm wide, with a cult conditions (i.e. a small difference between inside flat continuous top at least 150 mm above, is a simple and outside temperatures) it can easily be restricted answer to extraction in narrow yards, and a 600 mm when temperature differences are greater or appre- wide ridge in yards above 14 m width.

ciable winds help in the ventilation. As mentioned, in An even more satisfactory arrangement is to have a houses where no great control is necessary, such as cli- series of chimney-type ventilators along the ridge, matic housing for cattle, a fixed open ridge with a pro- allowing 0.09 m²/animal. A useful size of chimney is



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of variable fan speed on a motorized thermostat or electronic control will give full automation on all fans.

Fan ventilation halves the number of roof outlets that need to be installed. The total capital cost of a fan-operated system will hardly be more than £30 per animal-place, an economical cost for automatic environmental control. Running costs may be a rather more daunting charge.

The main use of fan ventilation in cattle yards is in those cases where extremely high stocking rates are

used, as with slatted floor units, or where the topography of the site or restriction of wind around the building makes natural flow all but impossible.

Fig. 56.9

The 'breathing' roof: ventilation achieved by leaving

The inlet of fresh air is no less important, and it has a gap between the roof sheets.

been found satisfactory to provide inward-opening hopper flaps along both side walls, bottom-hung and 700–900 mm deep, with gussets and variable control 1 m²; this is sufficient for 11 animals. The throat can be through casement stays or remote control from the controlled by a butterfly valve or hinged flap.

feeding passage. Alternatively, the hoppers may be controlled from the ends with horticultural glasshouse type are as follows:

fittings. It is preferable if the inlets are not closer than 600 mm to the eaves, to prevent incoming air 'bouncing'

(1)

Raised sheets, consisting of conventional steel

off exposed purlins and causing an irritating draught on the animals. The flaps should be made so overlapping, allowing air to pass between the sheets fitted with a batten or washers raising the draught on the animals. The flaps should be made so that they can be removed or hinged down flat in the sheets. A spacer batten of treated timber measuring 50 mm \times 25 mm is suitable. preferably two-thirds of the wall length.

(2)

Uprturned corrugated sheeting. Conventional galvanized corrugated steel sheeting is fixed upside down with a gap between each sheet, forming a Venturi flow between each sheet. Typically, a 25 mm gap may be used, which would give 3.75 per cent opening of the roof. (Fig 56.9).

in narrow-span yards. In addition, there are a number Systems (1) and (2) are often described as ‘breathing of other excellent techniques for achieving good inlet roofs’.

ventilation for cattle and other yards.

‘Spaced boarding’ is excellent all around a yard – at the gable ends and along the walls above ‘animal height’

Mechanical ventilation

(Fig. 56.10). It gives an opening area of about 20 per cent of the total but is uncontrollable; an alternative is forms of intensively housed stock. With a system of to have an adjustable system with fixed outer slats and chimney trunks, fans can be added as a complementary inner sliding slats. There are also various other arrangement, since an extractor fan may be fitted at the ments, such as the Ventair steel sheet with patented base. If a 600 mm fan running at a maximum of louvres (13 per cent open); slatted hardboard with slats 900 rev/min is fitted, 10 194 m³ hour will be extracted. A 5 mm ¥ 25 mm giving 30 per cent opening; Netlon

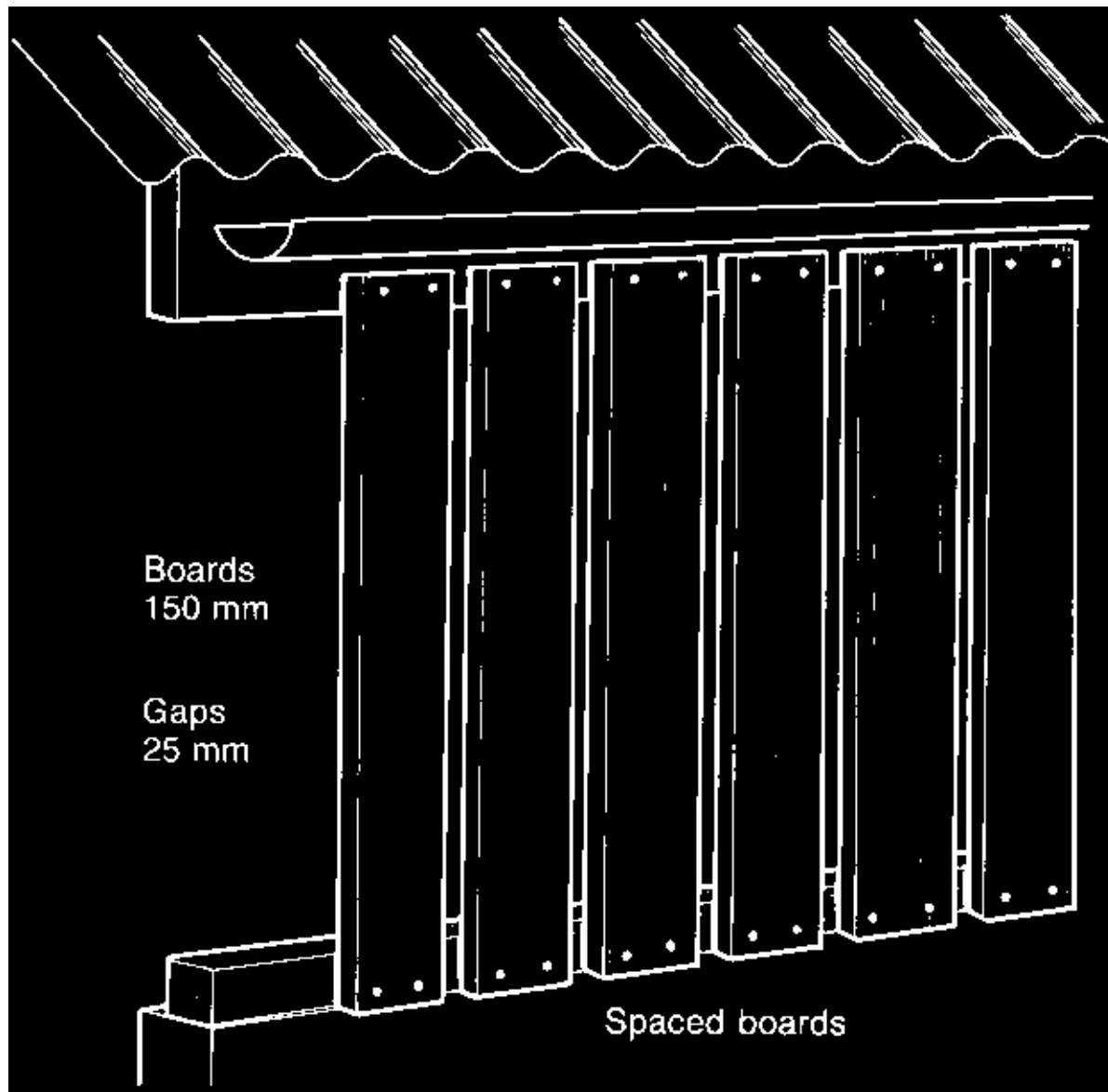
maximum allowance of 510 m³/hour per beast (or polypropylene mesh (45 per cent open); Ventrex steel approximately 0.42 m³/hour per kg bodyweight) is sheet with louvres (0.7 per cent open) and a common found satisfactory, so that one fan will serve approximately 30 beef animals to slaughter.

wall, giving a baffled protected inlet.

Automatic or semi-automatic control can be

It is in no way suggested that the systems outlined are achieved if a proportion (one-half to two-thirds) of the the only satisfactory methods of ventilating intensive fans are put on a thermostatic control, with a thermocattle yards. For example, other mechanical means have stat that is easily seen and adjusted. Fans thus operated been used, often with equal success. These do, however, should have automatic antiback-draught flaps fitted to often need more careful designing and management to prevent down-draughts when they are switched off. All prevent draughts and to give adequate control. The fans should be speed controlled. Alternatively, a system

systems suggested are logical and generally understood



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air comes in than to open up the ventilation space in a sheltered site. Sometimes the proximity of other buildings makes it impossible to increase the airflow at all. Livestock yards are now often made very wide, of the

order of 40–50 m. These are invariably the most difficult units to ventilate. From the point of view of ventilation it is desirable to limit the span to some 30 m. Indeed, if the trend towards the use of thermal insulation continues, views on what constitutes an adequate cubic area may change. In an uninsulated yard a large cubic area helps to buffer the outside weather extremes. But in an insulated construction, the roofing area must be reduced to a minimum to cut costs and more economical dimensions would be a height of 3.6 m to eaves with a maximum yard span of 21 m. These recommendations widely used and required for cattle buildings apply, with only minor modifications, to climatic yard accommodation generally, including that for pigs, sheep and turkeys.

Fig. 56.10

Wall ventilation with spaced boarding.

Open-fronted yards and kennels

A fundamentally different approach to climatic housing and easily controlled by the stockworker. Mechanical is the 'lean-to' or open-fronted yard, often called the assistance with the designs shown is complementary to

mono-pitch building. This is attractive in several natural flow and indeed may be used as an additional respects and continues to gain in popularity as an alter-stage in the development and improvement of a design. native to the span roof yard. For a start it is usually Draughts are least likely where there is the fullest cheaper. Also, by its very nature, it is impossible to trol of the system. Ventilation continues to function at make it very wide – a depth of 10 m being about the a reduced rate in the event of power failure with the limit – so the enormous problems of ventilation created system advocated, since natural stack-effect ventilation by wide spans are avoided and stock concentrations in takes over from the fans to tide the stock over this one air space are limited. Best of all, such a system gives period.

a cheap isolation of groups by carrying vertical parti- If the trend towards high stocking rates continues it tions to the roof and if necessary supporting it every is inevitable that the farmer will turn increasingly to 3–6 m. With an open-fronted design animals have a

thermal insulation of the surfaces, especially the roof.

choice of environment, as they can go towards the lower

Farmers who have used insulation of the roof have

back part in colder conditions to keep warm, or come

recorded considerable benefits in terms of liveweight

to the front for sunlight and more fresh air in warmer

gain and feed conversion efficiency, but above all

weather. Control of airflow can be by simple hinged

because it helps also to solve environmental problems.

flaps on the front and kennels can be incorporated if

A popular way of arranging the insulation is by using

desired. It is most desirable that open-fronted yards in

two skins of metal or fibre-cement sheeting for the roof

the northern hemisphere face in a southerly direction

separated by a vapour-sealed layer of mineral wool or

only; it is preferable not to face two rows together, since

glass wool. For the latter, a 50 mm thickness is minimal

there is a risk of the passage-way between becoming a

but 100 mm is very much better. A cheap treatment for

wind tunnel and in this situation one side at least will

an existing uninsulated building is to build a false

face north or east and so may get little sun and indeed ceiling of wire mesh supporting vapour-sealed insulation may suffer from distinctly cooler conditions.

It is an economical way of insulating the building, though somewhat temporary. It must be emphasized

Dangers from gases in farm buildings

that individual attention does need to be given to each yard according to the site and locality. It is one of the Problems associated with gases in and around the live-stock farm have been highlighted recently, particularly advantages of fan ventilation that with mechanical ventilation this statement is much less true. The use of fans in association with gas effusion from slurry channels partially rules out the dependence on site and weather. under perforated floors, but it would be wrong to regard

It is generally better to have yards in exposed positions these as the only dangers. While there have been because it is easier to restrict the open area if too much numerous fatalities of livestock recorded, and indeed of

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man, due to gas intoxication, there is also mounting

References

evidence that there may be concentrations of gases in

many livestock buildings that may affect production

Britton, D.K. & Berkeley Hill (1975) Size and Efficiency in adversely by reducing feed consumption, lowering

Farming, pp. 132–47. Saxon House Studies, Farnborough.

growth rates and increasing the animals' susceptibility

Clark, J.A. (ed.) (1981) Environmental Aspects of Housing for to invasion by pathogenic micro-organisms.

Animal Production. Butterworth, London.

The most serious incidence of gas intoxication arises

Donaldson, A.I. (1978) Factors influencing the dispersal, sur-

from areas of manure storage in slurry pits or channels

vival and deposition of airborne pathogens of farm animals.

under the stock, usually but not always associated with

Veterinary Bulletin, 48, 83–94.

Donaldson, A.I. & Ferris, N.P. (1976) The survival of some forms of perforated floors. The greatest risk arises when

airborne animal viruses in relation to relative humidity.

the manure is agitated for any reason, usually when it

Veterinary Microbiology, 413–20.

is removed. There is also an ever-present danger if a

Honey, H.F. & McQuitty, J.B. (1976) *Dust in the Animal mechanical system of ventilation fails and this is the*

Environment. Departement of Agricultural Engineering,

only method of moving air in the house. Several cases

University of Alberta, Canada.

of poisoning have been reported when sluice-gates are

Sanger, J.R. (1969) *Influence of relative humidity on the sur-*

opened at the end of slurry channels and the movement

vival of some airborne viruses. Applied Microbiology, 15, of the liquid manure has forced gas up at one end of

35–42.

the building.

Webster, A.J.F. (1981) *Optimal housing criteria for ruminants.*

High concentrations of gases, chiefly ammonia, may

In Environmental Aspects of Housing for Animal Produc-

also arise from built-up litter in animal housing. The

tion (ed. by J.A. Clark), pp. 217–32. Butterworth, London.

Whittlestone, P. (1976) *Effect of climatic conditions on*

danger has undoubtedly been exacerbated within more

enzootic pneumonia in pigs. International Journal of Bio-

recent years owing to the mistaken insistence of main-

meterology, 20, 42–8.

taining relatively high house temperatures in order to

Wray, C. (1975) Survival and spread of pathogenic bacteria

reduce food costs. The farmer has often attempted to

of veterinary importance within the environment. Veteri-

achieve such temperatures by restricting ventilation

nary Bulletin, 45, 543–57.

and in the absence of good thermal insulation of the

house surface, so that the result may often be generally

harmful if not actually dangerous.

Chapter 57

Biosecurity

D.C. Barrett and A.J. Taylor

Introduction

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While the public health aspect of biosecurity is

Benefits of improved biosecurity

988

obviously extremely important, the prime role of the

Routes of infection

988

veterinary surgeon is to safeguard the health of

Infectious diseases that can be introduced into cattle herds

989

food-producing animals, because disease will reduce

General principles of biosecurity

989

productivity and/or be detrimental to animal welfare

The 'closed' herd

990

(Barrett, 2001a). It is this animal health aspect of biose-

Introduction of purchased stock

990

curity which will be dealt with in most detail in this

Risk of disease in the herd affecting an incoming animal

991

Principles of biosecurity planning

991

chapter.

Producing a biosecurity plan

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From the point of view of cattle health, biosecurity

Herd biosecurity: where is the final frontier?

can be defined as ‘the efforts put forth to prevent the

Conclusions

introduction of pathogens or toxins that have the potential to damage the health of a herd of cattle or the safety and quality of a food product’ (Sanderson

Introduction

et al. , 2000). An interrelated concept is that of biocontainment, which is the ‘effort to contain the Biosecurity means a multitude of things to different spread of disease or intoxication within a herd’ people. In its broadest interpretation in relation to (Sanderson et al. , 2000). For the purposes of this human health it is defined as ‘those cumulative steps chapter, we will concentrate on the infectious disease taken to prevent transmission of pathogens and other components of both biosecurity and biocontainment, harmful agents to individuals or populations of human and use the term biosecurity in a generic sense to cover beings’ (Gillespie, 2000). Gillespie (2000) further states

both concepts.

that the harmful effects of these agents may be direct

In the past, efforts to use biosecurity principles to

(e.g. smallpox virus causing disease) or indirect, by

control diseases of cattle in the UK have been largely

disrupting the food supply (e.g. animal or plant

confined to the statutory control and eradication, or

pathogens), and that any substance that disrupts bio-

attempted eradication, of notifiable diseases such as

logical systems necessary for human health and well-

foot-and-mouth disease (FMD), bovine tuberculosis

being is a threat to biosecurity. In addition to

(TB) and brucellosis, or specific diseases such as bovine

pathogens, it is also suggested that naturally produced

herpes virus-1 (BHV-1), the cause of infectious bovine

poisons, such as aflatoxin, toxic chemicals and pesti-

rhinotracheitis (IBR) (p. 286), and leptospirosis (L.

cides, all have the potential to disrupt beneficial and

*borgpetersenii serovar hardjo and L. interrogans necessary biological systems
and can therefore also be*

serovar hardjo) (p. 735) within non-statutory cattle

considered to threaten biosecurity.

health schemes. This has resulted in an attitude amongst

From the point of view of human health, the ultimate

many cattle producers that infectious disease control is

aim of biosecurity is therefore to safeguard the human

either something that government should take charge

food chain. While major epidemic diseases of cattle

of, and pay for, on their behalf, or is something that is

such as foot-and-mouth disease and rinderpest may

only relevant to a small number of herds who may

threaten food supplies, they do not have direct public

benefit from having an 'elite' health status. The major-

health implications. However, most foodborne illnesses

ity of commercial herd owners have considered that

are the result of inadvertent contamination of foods

endemic infectious diseases are always going to be

by pathogens, including viruses, bacteria, protozoa and

present within their herds. This has resulted in many of

perhaps also prions. These organisms may be zoonotic,

them tolerating the infections and acting in a reactive

causing disease in cattle and humans e.g. Salmonella

way to treat disease when it occurs while living with the spp., or foodborne pathogens that are non-pathogenic associated depression in productivity, rather than taking in cattle, e.g. Escherichia coli O157.

a proactive disease control and/or eradication stance.

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This is in contrast to the attitude in the UK pig and

Box 57.1

The benefits of improved biosecurity to an poultry industries where there are pyramidal structures, individual farmer.

designed amongst other things to help control infec-

*Increases in production, productivity and profit
tious disease. Pig and poultry producers, whether they*

Improved animal welfare

are breeding companies or commercial units, would not

More efficient utilization of resources

entertain the idea of introducing new stock without

*Reduced use of medication, such as antibiotics and
taking specific precautions to protect both the new-*

anthelmintics, with an associated reduction in the risk of comers and the indigenous herd or flock from infectious resistant pathogens emerging diseases. Although the cattle industry is structured very Enhanced value of the individual herd, with livestock, and differently from the pig industry, cattle producers could livestock products, of known health status commanding learn a lot from the biosecurity principles that have premium prices

been developed for pigs over many years (Moore, 1992). Even so, the outbreak of classical swine fever in Eastern England in 2000 (Sharp et al. , 2001) and the

Box 57.2

The benefits of improved biosecurity to the wider spread of new diseases such as post weaning multisys-farming community.

temic wasting syndrome with the movement of pigs Maximizing export markets by the prevention of disease-between farms (Done et al. , 2001) has shown that orientated barriers being put in place by importing countries disease transmission between pig units remains a

Possible limitation of imports by creating disease-orientated problem.

barriers to the importation of livestock, and livestock

Even before the devastating outbreak of FMD in the products, from countries of lower health status

UK in 2001, the challenge to enhance biosecurity on cattle units was already very real. Biosecurity had become an increasingly topical issue in relation to cattle selected infectious diseases, there will be potential production, prompted by suggestions that TB control benefits related to international trade (Box 57.2).

measures would be enhanced if biosecurity were

Farm biosecurity is also an important first step in improved (Anon, 1999a,b, 2001a; Collins, 2000; Phillips safeguarding the human food chain with benefits to the

et al. , 2000; Barrett et al. , 2001), reducing both cattle-to-community at large. It is an important part of any

cattle (Menzies & Neill, 2000) and badger-to-cattle

hazard analysis and critical control point (HACCP)

transmission of Mycobacterium bovis (see p. 857). More plan to safeguard human health. Food retailers are

generally, much needed to be done at a herd level to

taking much more interest in this area as a means
improve herd health and productivity by implementing
of combating potential risks to human health
well-designed 'biosecurity plans' that were proactive in
(Noordhuizen & Wentink, 2001), for example the pos-
preventing the spread of numerous infectious diseases
sible link between *Mycobacterium avium* subsp. *paratuberculosis* (Johne's disease) (p. 862) in cattle and
The 2001 outbreak of FMD, which affected France,
Crohn's disease in humans (Hermon-Taylor, 2000). The
The Netherlands and the Republic of Ireland as well as
general population is now much more sensitive to
the UK, emphasized the need to safeguard the national
disease epidemics and food scares, e.g. salmonella in
herd from exotic diseases, particularly in these times of
eggs and bovine spongiform encephalopathy (BSE) (p.
a single European Market. The last frontier for disease
909), and farmers may in future need to show that they
control must be the farm boundary; cattle farmers have
have adequate biosecurity measures in place to mini-
to take responsibility for disease control in their own

mize such risks in order to sell to certain retailers.

herds, and, with advice from their veterinary surgeons, decide what precautions they need to take to reduce the risk of disease transmission.

Routes of infection

There are many ways in which an infectious agent can enter a herd of cattle. These are summarized in Box 57.3.

Benefits of improved biosecurity

The greatest risk for most diseases is by contact with infected cattle, either by the introduction of infected

Improving biosecurity on cattle units, both to control cattle into a herd or by contact with neighbouring herds infectious diseases already present and to prevent the via cograzing or across boundaries. Attendance at shows introduction of new infections, has a number of potential benefits at the farm level (Box 57.1). If improved introduction of disease. These infected animals may be biosecurity measures are widely adopted such that either asymptomatic ‘carriers’ of a specific disease such increased numbers of cattle herds become free of

as bovine herpes virus-1 (BHV-1), bovine virus diar-

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Box 57.3

Common routes by which infectious agents can

Box 57.4

Diseases endemic in the UK and elsewhere,

enter a herd of cattle.

which may be controlled in part by enhanced biosecurity measures.

Introduction of infected cattle or direct cattle-to-cattle contacts Feed or water, contaminated with pathogens such as Sal-Bovine virus diarrhoea (BVD)

monella spp. or the prion responsible for BSE

Infectious bovine rhinotracheitis (IBR)

Visitors, including those regularly working with cattle on other Leptospirosis premises, such as relief milkers, foot trimmers, AI techni-

Salmonellosis

cians and veterinary surgeons

Johne's disease

Mechanical vectors such as vehicles and farm implements

Mastitis, e.g. Streptococcus agalactia

Wildlife, including badgers, rabbits, rodents, deer and birds

Digital dermatitis

Other species of livestock such as sheep

Parasitic diseases

Airborne transmission, notably FMD

Ringworm

Udder skin infections

*Venereal *Campylobacter* spp. infection*

Neospora infection (causing abortion)

*rhoea virus (BVDV) (p. 853), *Neospora caninum* (p.*

*584), *Streptococcus agalactiae* (p. 333) or cattle incubat-a Zoonotic diseases transmissible to humans.*

*ing a particular disease such as *Salmonella typhimurium**

b There is some evidence that links this disease with Crohn's disease DT 104 (p. 850). In the case of cattle incubating disease,

in humans and its importance may therefore increase further in the it may be possible to prevent the introduction of a

future.

pathogen by imposing quarantine measures of a dura-

tion that exceeds the expected incubation period.

Box 57.5

Diseases notifiable in the UK.

However, some pathogens may not cause infected

These are the diseases subject to national control policies

animals to exhibit overt clinical signs, e.g. Leptospira

and/or international trade rules and include:

hardjo (L. borgpetersenii serovar hardjo and L. interrogans serovar hardjo).

Others may have excessively long incubation periods, making simple quarantine impractical

without the use of accompanying diagnostic testing,

Bluetongue

Bovine spongiform encephalopathy (BSE)

e.g. Mycobacterium avium subsp. paratuberculosis

Brucellosis

(Johne's disease).

In the case of 'carrier' animals that do

Contagious bovine pleuropneumonia (CBPP)

Enzootic bovine leukosis (EBL)

not exhibit clinical signs, detection is reliant on diagnostic

Foot-and-mouth disease (FMD)

testing, either of the individual animals or of the herd

Lumpy skin disease

from which they have been obtained. The reliability of

Rabies

diagnostic tests and the design and implementation of

Rift Valley fever

testing regimens have been discussed in detail by

Rinderpest

Caldow et al. , (2001). With or without adequate isolation Tuberculosis (TB)^b

and quarantine measures, appropriate diagnostic tests

Vesicular stomatitis

will need to be undertaken to ensure, as far as is realis-

Warble fly^a

tically possible, freedom from disease in introduced

a

animals or at least to define their health status and prior

Those diseases considered by the authors to pose the greatest

threat of re-emergence or introduction into the UK.

exposure to certain pathogens.

b Diseases known to be present in the UK in early 2002 (cNorthern To prevent direct or indirect contact between herds

Ireland only).

of neighbouring cattle, there should be a 3 m gap

d Occurred in the UK in 2003.

between boundary fences and hedges. This should mini-

mize the risk of short-distance airborne disease trans-

gories: diseases endemic in the UK and elsewhere, mission such as is possible for BHV-1, the cause of IBR. examples of which are shown in Box 57.4, notifiable diseases Long-distance airborne transmission, as is classically seen with FMD, is almost impossible for an individual other diseases not considered present in the UK (Box 57.6).

Infectious diseases that can be

General principles of biosecurity

introduced into cattle herds

Biosecurity is not a new concept; the introduction of The numerous infectious diseases that can be introduced into cattle herds can be divided into three categories: 'cattle plague' (rinderpest) (p. 707) into the UK in 1865 resulted in the Cattle Disease Prevention Act of 1866.

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Box 57.6

Other important diseases not widely present in can present some risk (see Chapters 39 and 40). When

the UK that may be introduced.

using ET, embryos must be transferred into home-bred

recipients; pregnant recipients or animals for use as

If cattle are purchased from another country, there are dis-

eases that are currently not present or at minimum levels

recipients must not be purchased. No animals must be

in the UK but which are also currently not subject to

loaned or hired out. No animals must be returned to the

national control measures or importation rules. For

herd from shows or sales. There must be no contact with

example:

other cattle when grazing.

Trichomoniasis

Some species of Leptospira

*Type 2 BVD virus**

Introduction of purchased stock

**Recently found in Great Britain.*

It will not be appropriate for many herds to be closed,

due to management constraints such as cograzing or a

This Act enforced the use of what we would now con-

production system based on the rearing of purchased

sider biosecurity measures to eradicate rinderpest from stock. It is therefore essential to minimize the risk of the British Isles after an estimated 400 000 cattle had introducing infectious diseases into the herd when died of the disease; the last cases were seen in 1877 buying new stock. The introduced animals may bring (Dunlop & Williams, 1996). The basic principles for the with them an infectious organism not previously prevention and control of many infectious diseases in present on the unit, leading to disease in the indigenous cattle have therefore been well recognized for over 125 population, e.g. Johne's disease. They may also introduce a different strain of a pathogen, e.g. anthelmintic-

Duncan (1990) set out the principles of infectious resistant gastrointestinal nematodes, or virulent BHV-1, disease control in cattle in the early 1990s. At a meeting or may themselves succumb to a disease endemic in the of the British Cattle Veterinary Association (BCVA) in indigenous population. Even where there is no desire 1994, three papers related to this subject were pre-

to achieve or maintain a high health status herd, the risk sented (Duncan, 1994; Taylor, 1994a,b), followed by a of introducing more virulent strains of a pathogen, or further paper in 1996 (Pritchard, 1996). Duncan (1990) drug-resistant pathogens, is a very real threat whenever set out a seven-point plan, subsequently modified cattle move between herds.

slightly as follows:

The safest source for new stock is a herd certified free of specific diseases, usually by a cattle health scheme. If

(1)

Maintain a closed herd, with no introduction of such stock are not available, the next safest route is to animals and no contact with outside cattle.

buy from farms of known health status. In order of

(2)

If introduction of cattle is unavoidable, operate an increasing risk:

isolation and testing policy for introduced stock, including other species such as sheep.

(1)

A health scheme herd being monitored for speci-

(3)

Ensure feed and water are free from

fied diseases.

contamination.

(2)

A single source herd of known disease status.

(4)

Control visitors and vehicles, and operate a policy

(3)

Multiple source herds of known disease status.

to reduce the risk of transmission of pathogens

from visitors and vehicles.

The highest risk is to purchase from multiple sources of

(5)

Control direct and indirect contact between

unknown disease status or herds in which the disease

wildlife and cattle, particularly rodents and birds.

is known to have occurred. Animals from livestock

(6)

Define and monitor the health status of the herd.

markets or dealers where cattle from multiple sources

(7)

Establish and operate an agreed disease control

are mixed together should be considered a very high programme or biosecurity plan.

biosecurity risk.

Unless from certified disease-free herds, incoming

animals should be isolated and tested before they enter

The 'closed' herd

the herd, preferably on the farm of origin before pur-

chase. Use of the farmer's own transport minimizes the

Maintenance of a closed herd is the main plank of a

risk of contact with sources of infection in transit, par-

biosecurity programme. No animals must be purchased,

ticularly if the haulier cannot guarantee not to mix

borrowed or hired. New genetic material must only be

stock in the lorry. The incoming animals should ideally

introduced to the herd by artificial insemination (AI)

be isolated on arrival and tested according to a specific

or embryo transfer (ET), but even these introductions

pre-agreed protocol before entering the herd.

Some suggested control measures for diseases that with the use of certain management practices in relation to the target infections. The next step is to assess of cattle are outlined in Table 57.1.

the benefits of risk reduction within the specific biosecurity plan. In an ideal world, this would involve a quantitative risk analysis for each target infection. However,

Risk of disease in the herd affecting

this is usually not practical on an individual herd basis, as many of the required data are often unavailable and

an incoming animal

gathering these data and undertaking the required modelling would not be cost-effective. This is not the

While many farmers will realize that introduced cattle case at a national level and it is now the norm when may transmit disease to their herd, few consider the risk policy decisions are made on international live-to the incoming animals from pathogens within the stock trade to employ quantitative risk assessment

indigenous population. This can be a particular problem (Wooldridge, 1996; Murray, 2002). Nevertheless, a qualitative risk assessment, using what limited information is available for an individual herd, may be very helpful as well as by specific infections of the reproductive tract. when beginning to construct a biosecurity plan. The In addition, pregnant females may abort if infected with many pathogens during gestation. For this reason, the basis for risk assessment is not to eliminate all risk, but to divide risks into various levels to assist informed disease status of all incoming animals should be established with regard to pathogens endemic in the herd decision-making (Wells, 2000). Focusing on the major hazards and major areas of risk will allow the cost- and, whilst in isolation, they should be treated and/or benefits of a biosecurity plan to be maximized. vaccinated as necessary.

In the specific case of an introduced bull, it is worth remembering that there is a time lag of approximately

Producing a biosecurity plan

two months from the start of spermatogenesis to the ejaculation of the sperm. This means that an insult to A farmer needs help and advice from his veterinary the testes may not result in deterioration in semen surgeon if he is to make rational decisions to protect quality for many weeks and, conversely, it can take the health of his herd. This advice is best given in the months for semen quality to recover following a disease form of a written biosecurity plan for the farm. This episode. Many diseases may also reduce conception could be a stand-alone document or a component of a rates in cows and heifers if they become infected general Herd Health Plan such as the BCVA Herd around the time of conception. Therefore new bulls Health Plan (Anon, 2001b).

should be introduced to a herd only after an appropri- The safest policy will always be to ascertain the ate isolation and testing regimen has been undertaken. disease status of the indigenous herd and then produce However, this may prove extremely difficult to achieve

a considered biosecurity plan based on a qualitative (or when bulls are purchased through commercial sales, as if possible quantitative) risk assessment, looking at all the timing of bull sales is often very close to the time likely hazards for disease transmission. Particular care they are required for breeding, especially in seasonally-needs to be taken when animals are to be introduced calving suckler herds.

into a herd or where members of the herd are known to have had contact with outside animals, such as at a show. The action to be taken when new stock are to be

Principles of biosecurity planning

introduced to the herd must be decided in advance.

This should include details of where animals are to be

An effective biosecurity programme needs to be

obtained from, the provision and management of isola-

decision-focused and flexible enough to adapt to the

tion facilities, the testing, treatment and vaccination

unique situation of the individual enterprise. This

strategy and what is to happen if an animal fails to meet

requires an understanding of biosecurity principles and

the predetermined health criterion.

the goals of disease prevention, as well as specific infor-

*The written plan must have some flexibility built into
mation relating to the biology and epidemiology of the
it and it must be agreed by both the farmer and his
particular pathogens of interest.*

veterinary surgeon. It must be reviewed regularly and

*When considering the construction of a biosecurity
both parties must work together to maintain interest
plan, it is important to establish which infections are
and motivation.*

worth concentrating on, to ascertain the current disease

*A possible future aid to biosecurity planning in Great
status of the herd and then to assess the risks associated
Britain may come from the British Cattle Movement*

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Chapter 57

Table 57.1

*Examples of infectious conditions likely to be introduced to UK herds with added
animals, with brief suggestions of measures to prevent disease introduction.
(Modified and extended from Pritchard, 1996.)*

Disease/infection

Preventive measure

Vaccines

Comments

available in

Clinical

Prophylactic

Laboratory

UK

examination

treatment

tests

BVDV

(+)

~

+

Yes

It is essential to test for BVD virus (up to 1.8% of cattle may be virus positive); serology also useful. Calve pregnant females in isolation and test calves at birth for virus if dam seropositive; or isolate calves and test when over 4 months of age for virus and antibody. Bulk milk tests available.

IBR (BHV-1)

(+)

~

+

Yes

Significance of adding seropositive (potentially infectious) animals depends on virus strain, vaccination type if used, existing herd status and possible implications for sale of cattle, semen and embryos. Bulk milk tests available.

Leptospirosis

(+)

+

+

Yes

Test animals using ELISA, 25 mg/kg dihydrostreptomycin given twice within 14 days of entry reduces risk of excretion. Bulk milk tests available. Zoonotic.

Salmonellosis

(+)

(+)

+

Yes

Repeated culture of faecal samples should help to detect carriers.

Positive SAT titre for S. dublin/ S. typhimurium on serum may indicate recent exposure. Zoonotic.

Johne's disease

(+)

~

(+)

Yes

Individual animal screening to detect subclinical infection is hampered by a lack of suitable tests. Tests such as adsorbed ELISA may be useful on a group basis. Request certification of clinical freedom in the herd of origin for a substantial period of time, e.g. 5 years. If diarrhoea develops in isolation examine faeces repeatedly for acid fast organisms and undertake culture. Consider rejecting animals with intermittent/

persistent diarrhoea regardless of test result. (Zoonotic?)

Streptococcus agalactiae

(+)

+

+

No

Should be readily preventable by using CMT, culturing positive quarters and/or treating. Bulk milk screening may be of use.

Digital dermatitis

+

(+)

~

No

Good clinical examination. If in doubt treat with topical antibiotic spray/

footbath.

Ectoparasites

+

+

(+)

No

Use appropriate treatment.

Gastrointestinal parasites

~

+

(+)

No

Use carefully selected, appropriate anthelmintics.

Lungworm

~

+

(+)

Yes

Use carefully selected, appropriate anthelmintics. Vaccine will not totally prevent infection.

Liver fluke

~

+

(+)

No

Use carefully selected, appropriate treatment.

Ringworm

+

(+)

(+)

Yes

Can cause quite serious problems in naïve herds, particularly amongst adult animals. Zoonotic.

Miscellaneous udder/skin

+

(+)

(+)

No

Troublesome and best avoided. Some are zoonotic.

conditions

Venereal campylobacteriosis

~

+

(+)

No

Treatment probably more cost-effective than laboratory tests, although not 100% effective especially in older bulls.

Trichomoniasis

~

~

(+)

No

Probably absent from UK, but check imported bulls and remain vigilant.

Tuberculosis

(+)

~

(+) *Skin test*

No

Increasing incidence in UK making the introduction of infected cattle into a herd more likely than in the past. Consider isolation and private testing.

Brucellosis

~

~

+

No

Present in Northern Ireland but absent from Great Britain except for a small outbreak in 2003.

Neospora caninum

~

?

(+)

No

Problematic, epidemiology complex and not fully understood. Consider not retaining calves from seropositive cows and not introducing seropositive cows or youngstock into a herd. Antibody titres wax and wane, likely to be highest around time of calving. Control dogs on farms.

Other exotic diseases, e.g.

Foot-and-mouth disease

+

~

+

No

Major threat if national biosecurity is breached.

Contagious bovine

(+)

~

+

No

Threat if national biosecurity is breached.

pleuropneumonia (CBPP)

Enzootic bovine leukosis

(+)

~

+

No

Threat if national biosecurity is breached.

(EBL)

Warble fly

(+)

+

+

No

Threat if national biosecurity is breached.

Biosecurity

CMT, California mastitis test; ELISA, enzyme linked immunosorbent assay; SAT, serum agglutination test.

+, Indicated; (+), possible benefit; ~ not indicated.

Note: All animals entering a herd should have a period in isolation; this is assumed in the table.

-

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Service (BCMS). Introduced in 1998, it gave British animals, but even when animal movements were farmers, for the first time, information on the origin and banned in the early days of the outbreak, the disease movement of all cattle they may be considering pur- continued to spread due to movement of people and chasing. This allows some assessment of disease risk to vehicles (Gibbens et al. , 2001). Increasingly stringent be made based on the animal's movement history. This rules were introduced in the worst affected areas and it can be enhanced by declarations of health status by was only when these rules were rigidly enforced that the farmers, supported where applicable by veterinary cer- spread of the disease was stopped. tificates and copies of laboratory test results. In future, Elimination of the FMD virus from the UK and the there may be an opportunity to include health infor- EU does not, however, reduce the need for good biose-

mation on cattle passports, including vaccination
curity. On the contrary, it emphasizes the need for
history and the health status of all herds in which the
continued vigilance so that the impact of any future out-
animal has previously resided.

breaks of disease, whether at farm, local or national
level, is kept to a minimum.

***Herd biosecurity: where is
the final frontier?***

Conclusions

For many years, efforts to use biosecurity principles to
In an ideal world all herds would be maintained as
control diseases of cattle in the UK were largely con-
closed units of known and preferably high health status,
fined to control and eradication of statutory diseases.
with the introduction of genetic material only by the use
Cattle imported into the UK were required to be kept
of AI and ET following strict quality control standards
in official quarantine premises before being allowed
to minimize the disease risk. However, we do not live
to move to the destination farm. The general attitude

in that world. We live in a world of widespread, frequent, long distance transport of animals and animal products, often with little regard for biosecurity. When the single European Market came into force in January 1993 the quarantine requirement was dropped and it became much easier to import cattle, not only from other EU member states but also elsewhere. Nevertheless, there is a lot that can be done, even on commercial units, to manage and reduce the risk of infectious disease.

It was only then that farmers started to realize that ‘The principles of biosecurity are applicable at some farm gate is the frontier’ (Taylor, 1994b). That principle level to all cattle units and each and every one stands still holds today, ‘The primary frontier for disease to gain by the implementation of a specific tailor-made control must be the farm boundary’ (Gibson & Andrews, 2000).

surgeons to evaluate the risks of various hazards on

Responsibility for biosecurity at farm level is the each cattle enterprise and use this to design and implement an appropriate biosecurity risk management plan joint responsibility of the farmer and his veterinary surgeon. However, prior to the outbreak of FMD in specific to the individual farm.

the UK in 2001, only a small number of herds were members of voluntary non-statutory cattle health

References

schemes such as the Premium Cattle Health Scheme and Herdcare. Standards for such schemes are set by Anon (1999a) Farm biosecurity – protecting herd health, pp. 1–5. MAFF PB 4517. MAFF, London.

which is a self-regulatory body established in 1999 by Anon (1999b) TB in cattle – reducing the risk, pp. 1–5. MAFF the UK cattle industry. The CHeCS technical document PB 4516. MAFF, London.

(Anon, 2000) includes rules on biosecurity, which are Anon (2000) Cattle Health Certification Standards (CHeCS). mandatory for members in the accreditation and

*Technical document. CHeCS, Dairy House, 60 Kenilworth
screening and eradication programmes and are recom-*

Road, Leamington Spa, Warwickshire CV32 6JX.

mended as good practice for all cattle herds. Other

Anon (2001a) TB and cattle husbandry – government

high status herds include commercial bull studs, which

response to the report of the independent husbandry panel

(TBF 51). Available at <http://www.defra.gov.uk/>.

operate under EU Directives and UK Regulations

Anon (2001b) BCVA herd health plan for the purposes of farm (Barrett, 2001b).

*assurance (revised), pp. 1–21. BCVA Office, The Green, The 2001 FMD
outbreak in the UK brought home to*

Frampton-on-Severn, Glos GL2 7EP.

the whole cattle industry the absolute necessity of good

Barrett, D.C. (2001a) Biosecurity and herd health – a chal-

biosecurity to control the spread of the disease. In this

lenge for the 21st century. Cattle Practice, 9, 97–103.

outbreak, there was very limited airborne spread of

Barrett, D.C. (2001b) Biosecurity and its importance in non-

the virus. The initial spread was due to movement of

regulated venereal diseases of cattle. In Proceedings of the

5th Annual Conference of the European Society for Domes-

*plies and its impact on human health. Cattle Practice, 8, tic Animal
Reproduction (ESDAR), Vienna, Austria, 13–15*

355–60.

September 2001, pp. 28–29.

*Menzies, F.D. & Neill, S.D. (2000) Cattle-to-cattle transmission Barrett, D.C.,
Sibley, R.J. & Taylor, A.J. (2001) British Cattle of bovine tuberculosis. The
Veterinary Journal, 160, 92–*

Veterinary Association – TB isolation and test protocol.

106.

Cattle Practice, 9, 147–50.

*Moore, C. (1992) Biosecurity and minimal disease herds. The Caldow, G., Gunn,
G., Humphry, R., Crawshaw, M. &*

Veterinary Clinics of North America – Food Animal Prac-

*Rusbridge, S. (2001) Herd health security: the role of labo-
tice, 8, 461–74. W.B. Saunders, Philadelphia.*

ratory tests. Cattle Practice, 9, 105–10.

*Murray, N. (2002) Import risk analysis; animals and animal Collins, J.D. (2000)
Tuberculosis in cattle, reducing the risk of products, pp. 1–188. Ministry of
Agriculture and Forestry, herd exposure. UK Vet, 5, 35–9.*

Wellington, New Zealand.

Done, S., Gresham, A., Potter, R. & Chennells, D. (2001)

Noordhuizen, J.P.T.M. & Wentink, G.H. (2001) Developments

*PMWS and PDNS – two recently recognised diseases of
in veterinary herd health programmes on dairy farms: a
pigs in the UK. In Practice, 23, 14–21.*

review. The Veterinary Quarterly, 23, 162–9.

*Duncan, A.L. (1990) Health security in cattle herds. In Prac-Phillips, C.J.C.,
Foster, C., Morris, P. & Teverson, R. (2000) The tice, 12, 29–32.*

role of cattle husbandry in the development of a sustainable

*Duncan, A.L. (1994) Health security in cattle herds. Cattle policy to control M.
bovis infection in cattle. Report to Practice, 2, 415–23.*

MAFF, available at <http://www.defra.gov.uk/>.

Dunlop, R.H. & Williams, D.J. (1996) Some animal plagues

Pritchard, G.C. (1996) Added animals: the challenge to pre-

*unmasked. In Veterinary Medicine – An illustrated History, ventive medicine.
Cattle Practice, 4, 253–7.*

pp. 377–93. Mosby, London.

Sanderson, M.W., Dargatz, D.A. & Garry, F.B. (2000) Biose-

Gibbens, J.C., Sharpe, C.E., Wilesmith, J.W., Mansley, L.M.,

*curity practices of beef cow-calf producers. Journal of the Michalopoulou, E.,
Ryan, J.B.M. & Hudson, M. (2001)*

American Veterinary Medical Association, 217, 185–9.

Descriptive epidemiology of the 2001 foot-and-mouth

*Sharp, K., Gibbens, J., Morris, H. & Drew, T. (2001) Epidemi-disease epidemic
in Great Britain: the first five months.*

ology of the 2000 CSF outbreak in East Anglia: preliminary

Veterinary Record, **149**, 729–43.

findings. *Veterinary Record*, **148**, 91.

Gibson, L.A.S. & Andrews, A.H. (2000) Disease security. In Taylor, A.J. (1994a) *Bull hiring. Cattle Practice*, **2**, 425–32.

The Health of Dairy Cattle (ed. by A.H. Andrews), pp.

Taylor, A.J. (1994b) Herd health security at Genus bull studs.

328–50. Blackwell Science, Oxford.

Cattle Practice, **2**, 537–40.

Gillespie, J.R. (2000) The underlying interrelated issues of

Wells, S.J. (2000) Biosecurity on dairy operations: hazards and biosecurity. *Journal of the American Veterinary Medical*

risks. *Journal of Dairy Science*, **83**, 2380–6.

Association, **216**, 662–4.

Wooldridge, M. (1996) Risk analysis, risk assessment, animal

Hermon-Taylor, J. (2000) *Mycobacterium avium* subspecies health and the decision-making process. *State Veterinary*

paratuberculosis in animals, food products and in water sup-Journal, **6**, 4–6.

Chapter 58

Immunological Fundamentals

W.P.H. Duffus

Introduction

996

of molecules that make up the myriad of different types

Innate immunity

997

of 'non-self' material that individuals are exposed to

Humoral

997

constantly. These are termed antigens, each site on the Cellular

997

antigen that retains the ability to be recognized by the

Acquired immunity

998

immune system being termed an epitope. Note the use

Humoral

998

of the word 'non-self'; this is a useful term as 'self'

Cell-mediated immunity

1001

means the immune system has lost the ability to recog-

T lymphocytes

1001

nize that particular antigen within an individual and
B lymphocytes

1002

Maternal immunity

1002

therefore the latter remains free of potentially harmful

Homeostasis and stress

1003

effector mechanisms. Unfortunately this protection
can break down leading to an increasing plethora of
autoimmune disease. The third main concept is the divi-

Introduction

sion of both the acquired and innate immune response
into cellular (e.g. lymphocyte, etc.) and humoral (e.g.
What does the word 'immune' mean? The derivation of
antibody, etc.) components.

the word stems from 'exempt from burden'. In reality

A study of immunology as it relates to bovine medi-

the word was coined to explain what we now term

cine is artificially skewed towards two of the four main

immunological memory. Populations, both human and

areas that cause sickness/death: injury and infection.

domestic stock, suffered and continue to suffer from

The other two main areas are degenerative disease and

epidemic disease; the old observation was that individ-

cancer but these are problems of the older individual;

uals recovering from a clinical syndrome were 'exempt'

subsequently defence mechanisms against these latter

at the appearance of the next epidemic.

two are not as important within a population, such as

This ability of the vertebrate immune system to

cattle, with a fast turnover of generations.

'remember' a previous exposure to a foreign or non-self

The majority of the mechanisms, both cellular and

material is at the very heart of preventive medicine.

humoral, that make up the ruminant immune system

The response is normally termed an adaptive immune

are common to all mammals. The highly developed

*response. This is in contrast to the innate immune mammalian immune system is
the culmination of evo-response which covers the mechanisms pre-existing*

lutionary pressures with effector mechanisms that are

within an individual. The division between these two

traceable in the lower animal kingdom. However, there main areas of the immune system is not absolute; there are aspects of this mammalian immune system that are is much interaction and interdependence between specifically enhanced and specialized within ruminants effector mechanisms in both camps. What do we mean (such as the passive transfer of lactogenic antibody). by effector mechanisms? The term usefully describes These differences will be discussed within the relevant the sharp end of the immune response: the antibody sections. An important conceptual point to get across that actually causes the destruction of a bacteria, the early on is one of balance between two conflicting cell that actually destroys another, but potentially points of view. On one side is the animal trying to pre-malignant, cell.

serve its integrity against a variety of potential

There are three other broad concepts to consider at pathogens by utilizing the effector mechanisms them- an early stage. First, the immune response can itself be selves comprising the innate and adaptive immune

harmful. The millions of individuals suffering from response. On the other side are the organisms that are allergy will know this fact only too well. The second trying to survive in the face of these mechanisms, main concept is one of specificity/recognition. The relying on a fast turnover of generations and natural immune system is exquisitely specific and has the selection. This balance is at the heart of almost all host ability to recognize millions of different combinations –pathogen interrelationships. With cattle and intensive

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Immunological Fundamentals • 997

husbandry we are tilting this balance in favour of the pathway going, an important point in assessing com-organism and allowing them to replicate and cause displacement as a defence mechanism.

eases such as mastitis, enzootic pneumonia, etc. Intensive husbandry is here to stay for production reasons

Lysozyme and other serum proteins

and we will have to turn to artificial means to restore the balance. This scenario will involve research to ascer-

Lysozyme, a protein with a molecular weight of approximately 14 000, is highly active against bacteria and is to a particular pathogen, and then how can we artificially boost this immunity.

the 'natural antibiotic'. It is not often realized that Fleming discovered both penicillin and lysozyme!

Neutrophils can be stimulated to produce lactoferrin.

Innate immunity

This protein works by competing with bacteria for the available iron molecules. Milk contains approximately 6 mg/ml. These immune mechanisms pre-exist in an individual

and are not improved by repeated infection. Like Acute phase proteins appear within hours of tissue damage or infection. The best known one is C reactive and cellular components.

protein which binds to phosphorylcholine on the surface of certain bacteria thus promoting phagocytosis

Humoral

by complement fixation.

Complement

Interferon (IFN)

The word 'complement' is a collective term for 15 or so

plasma proteins (enzymes) which can be activated

These glycoproteins are a family of cell-regulating

sequentially into functional units that then have three

proteins produced by many cell types in response to

major effects:

stimuli. Although viruses are well known as interferon

(IFN) inducers, others include endotoxin, bacteria and

(1)

Production of peptides which are active in inflam-

a variety of other antigenic stimuli. Interferons are clas-

matory processes.

sified as follows:

(2)

Production of C3b which, by its ability to

'opsonize' micro-organisms or other particulate

(1)

IFN-a is produced by leucocytes.

matter, is a powerful incentive for phagocytosis.

(2)

IFN-b is produce by fibroblasts.

(3)

Attachment to cell membranes to create lysis and

(3)

IFN-g is produced by T lymphocytes.

destruction of the target.

Interferon offers a rapid protection to viral infection.

After immunoglobulins, albumin and transferrin, C

It induces an antiviral state in cells by firstly triggering

3 is

the most abundant of bovine serum proteins (in excess

a receptor (IFN-ab share a receptor, IFN-g remains sep-

of 1 mg/ml).

arate). This stimulates the cell itself to produce proteins

How is complement activated? The combination of

to block the transcription of viruses, so protecting them

immunoglobulin with antigen will often cause activa-

from infection.

tion, but enzymes such as trypsin and bacterial composition. Interferon can be produced within hours of stimulation and the antiviral state can last several days. Interferon also has a number of other effects both on the classical pathway and as far as the antibody-immune system (such as stimulation of cytotoxicity antigen reaction is concerned it relies on the availability and increased expression of major histocompatibility of the Fc portion of the antibody molecule. In the complex (MHC) products), as well as other functions cow it used to be thought that of the two major subclasses such as cell division.

classes of IgG (IgG

All three types of bovine IFN have now been genetically engineered and are being investigated for their homologous bovine IgG potential as therapeutic agents. There may well be a

2/complement system,

has

clearly shown that both subclasses can fix complement.

role in diseases such as enzootic calf pneumonia.

Another pathway of complement activation is via the alternative route. This is a more primitive pathway and

Cellular

does not require the Fc portion of the antibody and can

Phagocytic cells

indeed be activated by antibodies such as IgA which is

not effective by the classical route. Certain isolates of

These include neutrophils, macrophages and other cells

E. coli and virus-infected cells can all set the alternative of the reticuloendothelial system. Free-living bacteria

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are fairly easy to kill without antibody or complement,

of 'policing' role within the body, ready to destroy spon-

but bacterial pathogens such as streptococci with large

taneous tumour cells when they arise or help clear up

capsules can alter the surface tension between the

virus infections. Cattle have NK cells, although the term

phagocyte and the bacteria so preventing phagocytosis.

almost certainly covers a heterogeneous population.

It is for this reason that specific antibody and/or com-

Natural killer cells can easily be recruited (stimulated

plement is needed to 'coat' or 'opsonize' the bacteria,

by cytokines such as IFN and IL-2). Interestingly in

altering the surface tension and allowing phagocytosis

cattle, NK activity is quite low in stock reared outside

to proceed. Bovine neutrophils and macrophages/

in extensive systems but is often very high in intensive

monocytes have C3 and Fc membrane receptors.

systems. Whether this is caused by the greater preva-

Cattle have unusually high numbers of monocytes in

lence of microbial infection in such individuals is not

circulation.

clear.

The phagocytic cells have several agents such as acid

Natural killer activity can also be down-regulated by

hydrolases, peroxidase and cationic proteins which can

agents such as prostaglandins. Do NK cells have any in

kill micro-organisms after phagocytosis. The cationic

vivo relevance? They certainly will kill virus-infected proteins come into play first when the pH goes up and

cells such as IBR-infected fibroblasts and tumour cells, in fact these mechanisms are now thought to be at least but the real clinical relevance requires further research.

or even more important than the traditional peroxidase-dependent pathways involving H₂O₂.

Several pathogens such as Brucella abortus, Salmo-

Acquired immunity

nella spp., Listeria spp. and Mycobacterium spp. can live intracellularly and thus pose a problem for the immune

Humoral

system. The solution is by activation of the macrophage, usually by specific protein messengers released espe-

Acquired humoral immunity is mediated through cially by T lymphocytes, called cytokines. Activated immunoglobulin, or antibodies as they are commonly macrophages are basically a lot 'meaner', more efficient known. These are a group of glycoproteins that have and, for example, contain more lysosomes; they are combinations of incredible variety and specificity. The quite capable of dealing with these more awkward

basic units are two identical polypeptide chains, termed pathogens.

light chains, linked to two further identical polypeptide

We must also consider another vital, indeed pivotal, chains, heavy chain, by several disulphide bonds (Fig. 58.1). role for macrophages. This is one we term antigen pres-

entation. These antigen presenting cells (APCs) are

The Fc portion of the molecule is responsible for found throughout lymphoid tissue, the archetypal one linking to the Fc receptor on leucocytes, while the other being the Langerhans' cell in the skin. Other special- end, Fab, carries the actual antigen-combining site itself.

ized APCs are found in the gut, respiratory tract and This latter site is also called the hypervariable region mammary gland. These cells basically phagocytose the simply because it is variation in amino acid sequence in antigen, process it and then deliver it as a neat package this region that produces the incredible diversity of to lymphocytes of the immune system. They combine antibodies.

this 'antigen presentation' with MHC products on their
An explanation of the plethora of titles that accom-
membrane and by the release of a soluble mediator
pany a description of immunoglobulins is given in Table
such as interleukin-1 (IL-1).

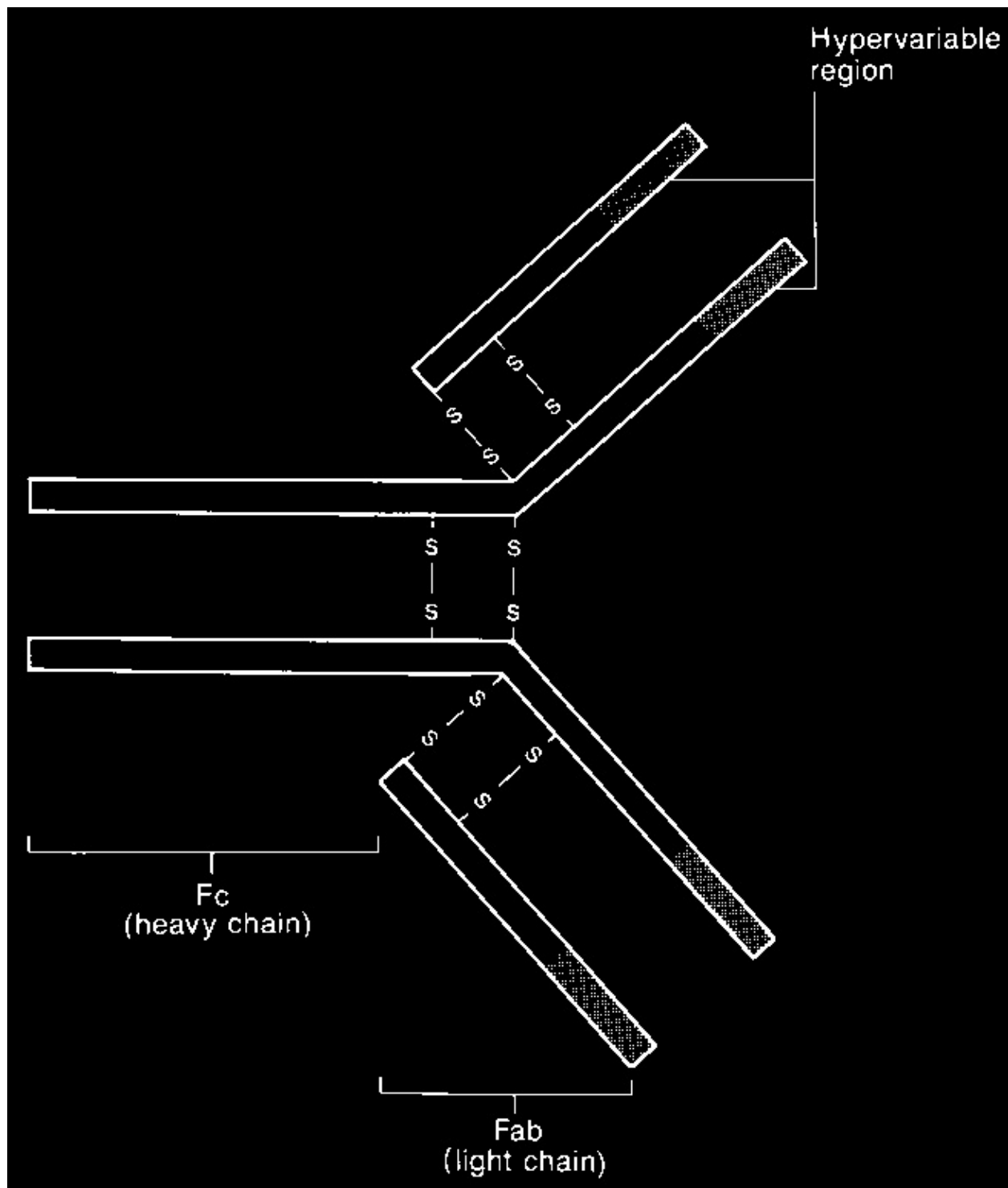
58.1.

Natural killer (NK) cells

IgM

It is only in the last five years or so that natural killer
IgM has five of the basic units forming a pentamer
or NK cells have come into their own. They are basi-
structure and represents an early stage in the evolution
cally lymphocytes with a separate lineage from T and
of antibodies. All vertebrates (with the exception of
B lymphocytes. The genealogy of NK cells remains
cyclostomes such as the lamprey) have an IgM closely
unclear: they share membrane determinants with both
resembling that of mammalian IgM except for the
monocytes and lymphocytes. They have Fc receptors
number of units polymerized into the complete mole-
and are quite large cells, often with noticeable granules.

cule. The level of IgM in normal cattle is in the range
Two fascinating things about NK cells are that: (i) they
of 2–3 mg/ml from the age of 4 months. Levels of IgM
pre-exist within individuals and do not require anti-
in calves is complicated by the amount absorbed in
genic stimulation; (ii) they can kill innumerable targets
colostrum. The actual amount in bovine colostrum is
from virus-infected cells to tumour cells without the
about 6 mg/ml which drops to approximately 0.1 mg/ml
need for antibody. Therefore, they could operate a type
in milk. Interestingly the bovine fetus is quite capable



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tions and stimulate B cells directly. Such antigens, including bacterial lipopolysaccharides, normally stim-

ulate only IgM production. The efficacy of specific IgM should not be underestimated. Not only is IgM highly efficient at aggregating antigen, it can bind with the secretory component (see below under IgA) and act as a protective antibody at mucosal surfaces. In diseases, such as *Escherichia coli* enteritis in calves, IgM has been shown to be a major protective mechanism.

In cattle suffering from infection with trypanosomes, IgM levels can rise in serum to over 20 mg/ml, although the majority of this overproduction is not particularly useful against the trypanosomes. On the other hand, in bovine leukaemia IgM levels can fall quite dramatically.

IgG

IgG predominates over other immunoglobulin classes in serum and contributes in excess of 85 per cent of all circulating immunoglobulins. Its structure is based on the usual immunoglobulin model, and it has a molecular weight of approximately 180 000.

IgG occurs in two subclasses: IgG1 and IgG2. Adult serum contains approximately 11 mg/ml of IgG1 and

Fig. 58.1

Descriptions used for immunoglobulins.

9 mg/ml IgG2. In colostrum the figure is well in excess of 50 mg/ml IgG1 and 3 mg/ml IgG2; this reflects the incredible selective concentration of IgG1 in colostrum (more details are given in the section Maternal im-

Table 58.1

Descriptions used for immunoglobulins.

munity, p. 1002). Twenty-four hours after successful

Class

Five major classes are distinguished in

suckling the calf has approximately 20 mg/ml IgG1 and

mammals: IgM, IgG, IgA, IgE and IgD. These

1 mg/ml IgG2, underlining the obvious and often reiter-

are distinguished in structure and major

ated importance of successful colostral transfer.

variations within the heavy chains.

As mentioned above, both subclasses can fix bovine

Subclass

Several of the individual classes are further

complement; IgG1 somewhat more so than IgG2. As for

split into subclasses, e.g. IgG1 and IgG2.

their reaction with leucocytes exhibiting Fc receptors, a

These are very similar in structure, but vary

somewhat paradoxical situation exists. In experiments

in the amino acid sequence of the heavy

involving freshly purified monocytes or neutrophils,

chain.

IgG2 was always much more efficient at mediating

Allotype

This is a term used to refer to genetic variation

phagocytosis or antibody-dependent cell-mediated

within a given animal species involving alleles

at a given locus; the variants normally occur

cytotoxicity (ADCC). Yet IgG1 is the predominant sub-

within heavy chains. Allotypes in domestic

class that the calf receives from the dam.

animals could well be linked to disease

In the cow and calf IgG1 has a highly significant pres-

resistance/production efficiency.

ence at the mucosal surfaces. While IgA is the pre-

Idiotypic

This term describes the product of a single

dominant immunoglobulin in nasal secretions in the clone of B cells, a unique antibody with adult, IgG1 is the predominant immunoglobulin in the a unique antigen combining site. Monoclonal calf up to at least the 6th week of life. In the lower respiratory tract, for instance the bronchoalveolar secretions, IgG1 becomes the dominant immunoglobulin in all ages. Even infections at mucosal surfaces will of producing IgM from about the third or fourth month produce high levels of circulating and specific IgG1 of gestation. equal to those achieved by intramuscular injection. It is, of course, well established that in a primary When one considers that the main site of the IgG1 synthesis is in the respiratory tract mucosa, it underlines on is IgM. What is not so well recognized is that certain the important potential of this immunoglobulin sub-antigens can bypass many of the normal T cell interaction in local defence mechanisms.

IgA

coli. So by a simple steric hindrance such microbes are unable to attach and are excreted.

IgA is recognized as a major immunoglobulin associ-

*This local stimulation does underline the necessity to
ated with mucosal surfaces. It is basically a dimer (con-
sider vaccination via a local mucosal route when
sisting of two of the basic units) joined by another
dealing with diseases where a major defence mecha-
molecule called the J chain (molecular weight of
nism involves secretory immunoglobulin. Many of the
approximately 15 000). This structure is combined with
currently successful vaccines are given via the mucosal
a secretory component (molecular weight of approxi-
route and even more are under active research.*

mately 60 000) to form secretory IgA or SIgA for short.

Another property of IgA lies in the dimer itself

*Individuals with mucosal surfaces have to have a
rather than the complete SIgA, or as one famous immu-
selective system that can decide which antigen to ignore*

nologist put it 'IgA can be more important beyond its and which to respond to. It is not in the animal's inter-laudable but essentially unglamorous role in intestinal est to respond to dietary antigens, although of course sanitation'. Thus, if a dietary or microbial antigen penetrates the mucosal barrier, a high-affinity IgA attaches mucosal immune system must be able to respond to it. There is no complement fixation or inflammatory quickly and effectively against a pathogen. One of the process, so the whole complex is carried off in the blood most important mechanisms in this decision-making to the liver where the IgA dimer reacts with the process is distinctive epithelial cells called 'M cells' that secretory component on hepatocytes with subsequent overlie Peyer's patches and other mucosal lymphoid release into bile.

aggregates. These cells have a tubulovesicular system with pits in the lumen surface and cytoplasmic

IgE

processes on the opposite surface enfolding lympho-

cytes and other mononuclear cells. These cells can
This immunoglobulin was first described for humans in
actively 'sample' the micro-environment within the
the mid 1960s, and is sometimes called the reaginic anti-
lumen and pass information on to the proactive cells of
body. It is, of course, associated with immediate hyper-
the lymphoid system.

sensitivity reactions. The structure of IgE is based on
The stimulation and subsequent expansion of the
the usual immunoglobulin model but has a higher
plasma cells that are responding to a particular antigen
molecular weight than IgG (196 000 as compared with
take place within the local lymphoid tissue itself. These
180 000). This is due to an additional structure in the
IgA-producing cells migrate to the submucosa where
heavy chain, which is thought to help in the high affin-
they assemble and secrete the complete dimer and J
ity this immunoglobulin has for Fc receptors on mast
chain. The epithelial cells on the microvilli have a recep-
cells. IgE normally exists in very low concentrations in
tor for IgA on their internal side that is probably the

serum (e.g. 20–400 ng/ml in dogs). Reliable and specific secretory component itself. The whole molecule, now reagents are not yet available for the cow but by sensitive in vivo tests like the passive cutaneous anaphylaxis endocytic vesicles and discharged into the mucus itself.

(PCA) test, potent IgE-like activity can be demon-

This process can also take place in the liver where hepatocytes can make secretory component; therefore bile

The antigens that can elicit a specific IgE response has high levels of SIgA.

are called allergens. The allergens are processed in the

What is the function of the secretory component? It

normal way and plasma cells in lymph nodes local to can be found free within normal gut contents or saliva,

the area through which the allergen gained access,

with large amounts in the newborn calf when very little

produce the specific anti-allergen IgE. Mast cell pre-

IgA is being produced. It seems to increase the resist-

cursors also migrate to the lymph node and they take

ance of the complete SIgA to proteolytic enzymes, up the IgE via their Fc receptors. The mast cells now although there is little evidence for any direct antipath-coated with the IgE migrate back to areas underlying ogenic role. One of its major roles may be to help the skin and/or mucosal surfaces. When the allergen anchor the SIgA on the mucous layer on top of the again gains access it reacts with the IgE and this causes epithelium, creating a high concentration of antibody the mast cell to release the contents of its granules, a activity at this crucial site.

whole range of pharmacologically active substances. The IgA part of the SIgA is not particularly effective These latter include 5-hydroxytryptamine, histamine, at 'traditional' roles of antibody such as opsonization, prostaglandins and cytokines such as eosinophil chemo-aggregation and complement fixation. Its major, and tactic factor. The outcome is smooth muscle contrac-highly successful role, is simply to block adherence. tion, increased permeability of capillaries and, through Micro-organisms must attach before penetration of

the chemotactic agents, migration of leucocytes. In fact mucosal barriers, e.g. the filamentous K antigen of E. the latter function is a component of the mechanisms Immunological Fundamentals • 1001

that 'dampen down' the reaction: eosinophils contain monoclonal antibodies has meant a highly specific and many factors such as phospholipase D and histaminase accurate assessment of these lymphocyte subpopulations that will inactivate the products of the mast cells.

tions. These are in today's parlance called CD (or Although such IgE reactions are considered a real cluster determinant) markers, each CD classification clinical nuisance both by humans and their animals, describing an identifiable membrane molecule which there must have been an evolutionary advantage to helps identify subpopulation. The major groups of develop and then retain this hypersensitivity reaction.

lymphocytes to consider are:

One clue for this is a possible mechanism against parasites, especially intestinal nematodes. Although there is

(1)

T lymphocytes, especially the major subsets coded a lot of experimental evidence in rodents, using para- for by the CD4 (T helper) and CD8 (T cytotoxic) sites such as Nippostrongylus brasiliensis, there is markers.

some evidence for nematodes of ruminants such as
(2)

B lymphocytes; lymphocytes involved in delayed- Haemonchus spp. The antiparasite mechanism works by type hypersensitivity.

antigens secreted by the nematode (often those associ-
(3)

An increasingly well-researched group of lym- ated with their mouth parts) being recognized as aller- phocytes lacking the classical T and B cell gens by the host. This results in suitably and specifically markers. The average human adult has about 10¹² sensitized mast cells migrating back to the gut. When lymphoid cells, representing approximately 20 per the worm feeds again the freshly released allergens acti- cent of all leucocytes.

vate the mast cells. The resultant smooth muscle contractions, and increase in permeability allowing through

T lymphocytes

other factors such as IgG, and the possible direct action of the mast cell mediators themselves, cause a mass

The thymus is central to immunological function. The expulsion of the worms. Prostaglandin might be one of T-cell precursors migrate there from the bone marrow the important effector mechanisms. A lot of research via the blood. The thymus extends a complicated but needs to be done to extend our knowledge of this highly effective 'education' of these immature T cells, intriguing immunoglobulin before it could be confirmed after acting through a cascade of thymic-derived hormones. It has been recently stated that it is a main mediator of intestinal immunity. mones such as thymosin and thymopoietin. The pre-

cursor T lymphocytes undergo about eight to ten cell divisions before they leave the thymus. A lot of the cells die within the thymus (maybe up to 90 per cent) and it

IgD

would be a neat solution to claim that this is how the
A brief and quite basic chapter such as this cannot go
host rids itself of all potential self-reactive lymphocytes.
into details of this immunoglobulin which, although
Unfortunately there is much conflicting evidence as to
presumed to occur in the cow, has yet to be convinc-
how exactly the thymus does its job.

ingly demonstrated. IgD is linked to B lymphocytes
What is certain is that when the lymphocytes even-
and appears on the surface of B cells after the initial
tually leave the thymus they can be classified as
expression of IgM. The IgD will co-express with IgM
CD4⁺ve or CD8⁺ve cells, two populations that are
and subsequently other immunoglobulin classes. The
mutually exclusive in peripheral blood. The lympho-
IgD is lost when the B cells respond to antigen by dif-
cytes have an amazing ability to migrate from blood to
ferentiating into immunoglobulin-producing plasma
secondary lymphoid tissue (within lymph nodes, spleen,
cells or memory B cells. The exact role of IgD remains
gut, skin, respiratory tract, etc.) and often back again to

unclear.

blood. This thorough 'mixing and movement' of lymphocytes is essential for them firstly to respond to antigen when it first appears, and then to be able to

Cell-mediated immunity

contact other cells with a view to initiating and finally curtailing an immune response. T lymphocytes tend to This is an often used 'catch-all' phrase that by strict definition mass into their own areas within secondary lymphoid initiation could cover most of the immune system. Early tissues, quite distinct from B lymphocytes.

on in fetal development the ubiquitous haemopoietic CD4 T cells are often called 'helper T cells', which stem cells produce two distinct progenitors: a common does underlie their important role. The antigen-myeloid and a common lymphoid. It is the eventual off-presenting cells or APCs are responsible for the initial spring of the latter cell population that are often considered to constitute cell-mediated immunity.

The latter then further present the antigen to other lymphocytes.

The lymphoid progenitors produce several different phocytes such as the CD8 population and B lymphopopulations of lymphocytes. The rapid development of cytes. The whole business of presentation is not only

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dependent on cytokines such as Il-1 and Il-2 but it is

Maternal immunity (see p. 211)

also MHC-restricted at the class II level. Crucial to the immune reaction is the T-cell receptor (TCR). The identification of the TCR remained a mystery until a few years ago when the TCR was shown to consist of two chains which clearly belong to the immunoglobulin family. This structure is closely linked to between maternal and fetal circulation, so there is little another CD marker: CD3. The human immunodeficiency virus (HIV) preferentially binds to CD4 cells and

*In bovine colostrum the level of IgG is up to a
it is the gradual destruction of this population that is an
massive 75 mg/ml with 4 mg/ml IgA and 6 mg/ml IgM.
important factor in the marked immunosuppression
Why does the neonate actually need all this antibody?
that is such a feature of HIV infection.*

*The newly born calf has a somewhat limited capacity
The CD8 population is also MHC restricted but at
to mount a fully effective immune response. It can
the MHC class I level. This is useful as a major function
produce IgM and some IgG1 at birth; from 36 hours old
of CD8 cells is cytotoxicity. Like other T cells, CD8 cells
it will start producing a lot of IgG1 at a rate of approx-
are highly specific for antigen, and for cytotoxicity to
imately 1 g/day (depending on antigenic challenge).
occur the CD8 cell needs to recognize antigen and
From one to two weeks SIgA starts production and
MHC. As most body cells express MHC class I then any
it is several weeks before IgG2 production starts in
cell expressing say a virus antigen could be attacked and
earnest.*

destroyed by the appropriate CD8 effector cell. In

The neonate is thus basically immunocompetent but

cattle diseases such as Theileria parva or IBR infection, has not 'tuned' its immune system to deal with the envi-these CD8 effector cells, or cytotoxic T lymphocytes

ronment, and it is in this period of tuning and massive

(CTL), have been shown to be important.

primary antibody responses that the neonate needs

A single CTL can kill several individual target cells

passive protection. How does this concentration of

and there is evidence that in virus infection, for

immunoglobulin in the bovine colostrum occur? The

example, recognition and then destruction of the target

major constituent of the immunoglobulins in colostrum

cell can occur before fully infectious virus is assembled

is IgG1 which is selectively transferred from serum. For inside the cell. The molecular basis behind the actual

example, a sheep can produce up to 2500 g of colostrum

destruction itself remains poorly understood. Certainly

in the first 48 hours after parturition of which 90 g will

it is vital to have the all-important recognition and close

be IgG1. When one considers that the concentration of

contact. The killing probably involves the release of IgG1 in her own serum is probably less than 0.015 g/ml, more than one soluble 'mediators'.

it is a major transfer. This transfer of IgG1 occurs via receptors on the heavy chain which binds to the acinar epithelial cells. This receptor interaction is under hor-

B lymphocytes

monal control and only occurs during late pregnancy.

B cells develop initially in the fetal liver but production

There is no such selective concentration for the much then shifts to the bone marrow. The pre-B cell has IgM smaller amounts of IgG2, IgM and IgA.

heavy chains within the cytoplasm. On a commitment

The specificity of the immunoglobulins is also of

to B cell lineage the B cell acquires surface IgM, the

obvious importance, not only reflecting the microenvi-

initial antigen receptor. A second phase of development

ronment of the dam herself, but also as a result of delib-

allows the B cells to express immunoglobulins of a

erate immunization policy. The neonatal calf has a

different class or subclass (plus IgD). On meeting its

greatly increased intestinal permeability due to special-pre-ordained antigen the B cell retains only the ized epithelial cells capable of pinocytosis. These cells immunoglobulin it is programmed actually to produce are activated by the presence of macromolecules and and then enters the terminal stage of immunoglobulin-eventually slough off after releasing their contents into secreting plasma cells. A proportion of the antigen-the circulation. This spontaneous closure starts in the specific cells remain as an enlarged population of calf at 12 hours postparturition with a mean complete memory cells.

closure time of approximately 30 hours. Degradation of Although B and plasma cells produce the this colostrum in the neonatal calf is minimal because immunoglobulins, they are under the tight control of it contains a trypsin inhibitor, and pancreatic activity the CD4 helper T cells for the majority of antibodies; a itself is low. Abomasal pH is between 6 and 7 due to the proportion of T-independent antigens can avoid this delayed development of parietal cells, but by 36 hours

control but at a cost of only IgM production and no postparturition the pH has dropped to between pH 3.0 immunological memory.

and 4.0, and pepsin is then active. A final point con-

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cerning immunoglobulin in colostrum/milk is that in

and, perhaps most importantly it is to a large extent

some diseases in cattle, such as rotavirus in calves, the

an automatic response. The response is switched on in passive protection is better obtained by the ingested

three main areas:

immunoglobulin rather than systemic immunoglobulin

being re-excreted into the gut. Therefore, it would make

(1)

A voluntary motor response, which operates via

more sense to feed diluted colostrum from vaccinated

the lower brain centre AE spinal cord AE peripheral

dams during the first week of life rather than relying

nerves and results in fight/flight, etc.

totally on the protection acquired passively during the

(2)

The adrenal medulla. This works via the autonomic nervous system and adrenal medulla

As well as the immunoglobulins in colostrum there is

releasing catecholamines, inducing the alarm

a high concentration of other effector proteins such as

reaction.

lactoferrin. In addition, there are leucocytes, with the

(3)

The hypothalamus, which is probably the most

macrophage and other mononuclear cells predominant

important for cattle.

in the resting mammary gland and at parturition.

During lactation, however, the predominant leucocyte

This latter mechanism involves the neuroendocrine

is the neutrophil, possibly reflecting the constant,

system, with the hypothalamic stimulating adeno-

although usually non-clinical, bacterial presence. The

hypophysis, which in turn stimulates (via ACTH) the

importance of leucocytes in mammary secretions is as

adrenal cortex, which releases cortisol. The latter

yet unclear for the neonate, but of obvious use for the hormone has an alarmingly high number of effects such as vasoconstriction in skin and intestine, vasodilatation in the working mammary gland will continue in muscles, raising blood glucose, raising pain threshold, to be a goal for researchers.

reducing secretions, etc. The integrity of lysosomal membranes is enhanced by cortisol, so although phagocytosis of bacteria and other particulate matter still

Homeostasis and stress (see Chapter 67)

occurs, it is more difficult for the enzymes to be released from the lysosomes into the phagosomes. Clinically this

In any book involving diseases of cattle, environmental can manifest itself as muscle weakness, trembling,

stress must be a major consideration. Every living

impaired immune response, poor wound healing,

organism in an effort to cope with its environment

weight loss, etc. Cortisol normally inhibits the produc-

attempts to reach homeostasis which can be defined as tion of adrenocorticotrophic hormone (ACTH) in a

the steady state achieved by the optimum action of feedback mechanism; however, this latter mechanism physiological regulation. Stress can be achieved by can be overridden by highly stressful conditions. changes in the animal's environment such as restraint, Summing up this section: the body attempts to handling, housing, weather, etc. Although there are respond to stress by a variety of means to restore home-many definitions of stress, one of the most useful is 'the ostasis. These responses evolved to occur in an open cumulative response of an animal resulting from inter-environment where the individual can move, seek action with its environment via receptors'. shelter, etc. But in a crowded calf pen there is no escape, This is therefore an adaptive response and is prima-the stress conditions remain with the inevitable rily directed at coping with changes in the environment consequences.

Chapter 59

Vaccines and Vaccination of Cattle

I.D. Baker

Definitions

1004

of infectious disease. In cattle practice a vaccine should

Anthrax

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produce herd resistance to minimize the economic

Clostridial disease

1005

effects of infectious diseases. They are usually adminis-

Salmonellosis

1006

tered to healthy animals. There are various types of

Neonatal calf diarrhoea

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vaccine.

Respiratory disease

1007

Bovine respiratory disease complex

1007

Modified live vaccines (MLV): There produce high

PI3 and IBR

1007

levels of immunity from a single inoculation. Their

Bovine respiratory syncytial virus (BRSV) and

Mannheimia haemolytica

1009

virulence is reduced by attenuation, which is achieved

Infectious bovine rhinotracheitis

1010

by either serially passaging the organism through tissue

Haemophilus somnus infection

1010

cultures or by genetic manipulation when those genes

Contagious bovine pleuropneumonia

1011

associated with virulence are removed. These types of

Parasitic bronchitis

1011

vaccine induce immunity by causing a mild infection in

Bovine virus diarrhoea

1011

animals, often showing mild disease signs and occa-

Johne's disease

1012

sionally causing unwanted side-effects.

Rinderpest

1013

Brucellosis

1013

Inactivated vaccines: These are killed vaccines; the

Strain 19 vaccine

1013

infectious agent is inactivated but retains its antigenic

Strain 45/20 vaccine

1014

properties, although these are often weaker than live

Louping-ill

1014

Papillomatosis (warts)

1014

preparations. As a result, two inoculations are needed

Erysipelas

1014

initially to provide adequate immunity. The antigenicity

Leptospirosis

1015

is often enhanced by using various adjuvants. These

Mastitis

1016

adjuvants may cause a local reaction at the site of the

Coliform mastitis

1016

injection.

Foot-and-mouth disease

1016

Ringworm

1017

Genetically modified vaccines: These are vaccines

Rabies

1018

where specific genes have been removed or added to

Other vaccines

1018

the DNA of the infectious agent. Apart from the atten-

There are many vaccines available for use in cattle in uation of vaccines these techniques have enabled the the UK and worldwide. This chapter is an attempt to development of marker vaccines. These are useful in summarize some of those available.

distinguishing between vaccine protected and infected animals.

Definitions

Autogenous vaccine: This is a vaccine prepared from cultures of material derived from a lesion of the animal

Vaccination is the introduction of a vaccine into the to be vaccinated.

body to produce immunity to a specific disease. The vaccine may be administered by subcutaneous, intra-

Toxoid: This is a toxin that has been treated by heat or dermal or intramuscular injection, by mouth, by inhala-chemical agents to destroy its deleterious properties

tion (intranasal) or by scarification.

without destroying its abilities to stimulate the formation of antibody.

Vaccine

A vaccine is a suspension of attenuated or killed micro-

Serovaccine: This is a combination of an antiserum with organisms administered for the prevention or treatment

a vaccine to produce passive and active immunity.

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Vaccines and Vaccination of Cattle • 1005

Anthrax (see p. 717)

Table 59.1

Clostridium spp. and disease syndromes in cattle.

Causative organism

Disease

Many types of vaccines are available internationally. No commercial vaccine is currently available in the UK.

Cl. chauvoei

Blackleg

The disease is notifiable and DEFRA should be con-

Postparturient gangrene

tacted if emergency supplies of this vaccine are needed.

Cl. septicum

Braxy

The vaccine that was available was a live vaccine

Cl. novyi type B

Black disease

prepared from a suspension of living spores of an

Cl. haemolyticum type D

Bacillary haemoglobinuria

encapsulated strain of Bacillus anthracis. The vaccine Cl. tetani

Tetanus

was stable for long periods.

Cl. botulinum

Botulism

Vaccines have been produced by overcoming viru-

Cl. perfringens (welchii)

Enterotoxaemia

lence using saponins or saturated saline to delay

Mixed species of Clostridium

Gas gangrene

absorption (Carbozo vaccines). In the UK the Stern

vaccine contained an avirulent strain.

Dosage and administration

toxoid. Vaccines for Cl. chauvoei are often a mixture of formalin-killed cells and a purified toxoid.

A dose usually of 1 ml is given by subcutaneous injec-

tion. This confers an immunity in seven to ten days that lasts for nine months. Annual booster doses are rec-

Dosage and administration

ommended except in areas of increased risk when they

Usually 2 ml is give by subcutaneous injection. The should be given at six-monthly intervals.

initial course is two inoculations separated by four to six weeks followed by an annual booster unless there is

Contraindications

great risk, when it should be every six months.

The use of antibiotics should be avoided from shortly before to 14 days after vaccination to avoid any inter-

Specific vaccines

ference in the production of immunity. Animals in the last stages of pregnancy should not be vaccinated unless

Blackleg (see p. 723): If this disease is identified at there is a serious risk of infection.

pasture then cattle should be removed from the pasture

There may be a slight reaction at the site of injection until the vaccination course is complete. Some veteri- and animals may have a mild pyrexia for one to two

narians give a concurrent dose of long-acting procaine days and milk yield may be depressed. These side penicillin at the same time as the initial vaccination to effects will subside.

avoid further disease outbreaks during the period of Empty or part-filled vials must be destroyed by developing immunity.

incineration.

Tetanus (see p. 733): Immunity usually lasts for three years following an initial course of vaccination and one

Other vaccines

annual booster. For especially valuable animals, there is

Another vaccine incapable of producing disease is a

a tetanus vaccine that carries a minimum risk of hyper-

cell-free filtrate of a culture of a non-encapsulated

sensitivity; whilst primarily designed for horses it can be

spore-forming strain of B. anthracis. This requires two used in cattle. It is prepared from purified tetanus toxin

injections to produce immunity but it is long lasting.

converted to toxoid by treatment with formalin and

then adsorbed onto aluminium phosphate. A polyvalent

vaccine that is commonly available contains the fol-

Clostridial disease (see pp. 719, 725, 733)

lowing organisms: *Cl. chauvoei*, *Cl. septicum*, *Cl. novyi*, *Cl. haemolyticum* and *Cl. tetani*. It seems eminently The clostridial organisms are common and responsible

sensible to use a polyvalent vaccine such as this instead

for many serious diseases in cattle (see Table 59.1).

of an individual vaccine since all these diseases occur

Vaccines against these diseases are available individually commonly.

ally, for example blackleg and tetanus, but they are

often combined as polyvalent vaccines. In general, the

Botulism (see p. 721): Vaccination is practised in

vaccines are prepared from formalin-killed cells

enzootic areas with either a combined *Cl. botulinum*

adsorbed on to aluminium hydroxide producing a

types C–O or a type-specific toxoid vaccine.

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Salmonellosis (see also Chapter 15)

Dosage and administration

For prophylaxis in calves, up to 20 ml is given subcuta-

Many species of *Salmonella* can cause disease in cattle, neously and repeated at 10–14-day intervals during the

e.g. S. dublin, S. typhimurium, S. newport and S. arizona.

period of risk. For therapy up to 40 ml is given sub-

The two commonest species in the UK are S.

cutaneously and repeated at 24 hours if required. In the

typhimurium and S. dublin and these can be endemic in author's experience these products are not always

a herd. The so-called exotic Salmonella species are not cost-effective.

usually endemic.

Neonatal calf diarrhoea

Live vaccine

A live Salmonella dublin vaccine prepared from an

Neonatal calf diarrhoea is a multifactorial disease

avirulent strain (HWS 51) used to be available in the

complex involving managemental and infective factors.

UK. It was a freeze-dried vaccine, which was reconsti-

Several bacteria, viruses and protozoa are associated

tuted immediately before use. This vaccine was very

with the disease, for example enterotoxigenic E.coli

effective both in calves and in adults. It was especially

(ETEC) (see p. 201), Salmonella spp. (see p. 215),

useful in controlling the disease in the face of an

rotavirus (see p. 191), coronavirus (see p. 199) and cryptosporidia (see pp. 204, 282). Pathogenic infections with *E. coli* are most common in the first four days of life, Inactivated vaccine whereas rotaviruses are most pathogenic from 7–14 days and coronavirus causes disease from 7–21 days.

An inactivated vaccine containing *S. dublin* and *S. typhimurium* is licensed in the UK and can be used in calves in practice; they contain inactivated strains of the vaccine.

Vaccines are available (Table 59.2) and are very successful in practice; they contain inactivated strains of the vaccine.

When the disease has been confirmed, all at-risk stock not showing overt clinical signs.

It is important that the *E. coli* component contains cal salmonellosis can be vaccinated. Calves are given a

both K99 pilus antigen and K99 capsular antigen. The 2 ml dose subcutaneously and can be vaccinated from

K99 antigen enables *E. coli* to adhere to the villi of the 21 days old. A second dose must be given after 14–21

calves' small intestine where rapid bacterial multiplication causes the production of toxins that are responsible for the disease.

Adult cattle require a 5 ml dose; again this is

responsible for the disease.

calves' small intestine where rapid bacterial multiplication causes the production of toxins that are responsible for the disease.

Adult cattle require a 5 ml dose; again this is

responsible for the disease.

repeated after 21 days.

ble for the diarrhoea.

Pregnant cows should be vaccinated six and three weeks before calving. All cows that have not calved

Dosage and administration

within eight weeks of the second inoculation should be vaccinated again three to four weeks before calving.

The pregnant cow is vaccinated between 12 and 4 weeks

This will then confer passive immunity to the calves via of the expected calving date. Calf protection is depend- the colostrum.

ent upon the calf receiving adequate (3 litres)

colostrum within the first six hours of life. They must then continue to receive colostrum or milk from vacci-

Risks

nated cows for the first two to three weeks. This is no

A small number of animals may not show an adequate problem in the suckler herd. In the dairy herd, milk

response to vaccination. Ideally, healthy animals should from the first six to eight milkings should be collected,

be vaccinated. Adequate immune levels will not be

*pooled and stored in a cool place. The calves should
reached until two weeks after the second injection.*

*then be fed, according to size, 2.5–3.5 litres/day of this
Avoid stress when vaccinating pregnant cows or abor-
milk for the first two weeks of life. In a seasonal calving
tion or metabolic disease may occur.*

*dairy herd with a close calving pattern it is common
practice to vaccinate one cow in four and use her
colostrum and milk for the calves. However, the best*

Serovaccines

results are achieved if all the cows in the herd are vac-

*Commercial serovaccines have been available contain-
inated; in this way infection and the level of excretion*

*ing mixed antisera to *S. dublin* and *S. typhimurium* of infected organism are kept
to a minimum.*

*together with polyvalent strains of *E. coli* and *P.**

Oral vaccines have been used and are available in

*multocida. These are designed to give passive immunity some countries (for
example the USA). They are com-to calves for prophylaxis and treatment of
enteric and*

bined MLV of rotavirus and coronavirus and must be

*pneumonic infections caused by *E. coli*, *S. dublin*, *S.**

*given as soon as possible after birth. The suggestion
typhimurium and Pasteurella multocida.*

that colostral antibody may interfere with these oral

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Table 59.2

Neonatal calf diarrhoea vaccines available in the UK, 2003.

Vaccine

Manufacturer

Type of vaccine

Dosage and

Vaccination

Duration of

administration

programme

immunity

Lactovac

Intervet

Inactivated strains bovine

5 ml sc

2 doses 4–5 weeks

Single annual booster

rotavirus (str 1005/78 and

apart to pregnant cows

given 2–6 weeks

Holland), bovine coronavirus

and heifers, 2nd dose

before calving

(str 800)

2–3 weeks before

E. coli K99 and F41

calving

Rotavec K99

Schering-

Inactivated strains bovine

1 ml sc in neck

Single injection 12–4

Single annual booster

Plough

rotavirus, E. coli K99

weeks before calving

Rotavec

Schering-

Inactivated strains bovine

2 ml im in neck

Single injection 12–3

Single annual booster

Corona

Plough

rotavirus (str UK –

weeks before calving

Compton) serotype G6P5

bovine coronavirus

(str Mebus)

E. coli F5 (K99)

Trivacton 6

Merial

Inactivated strains bovine

5 ml sc

2 doses to pregnant

No data but suggested

rotavirus, bovine coronavirus

cows and heifers, 1st

annual single dose

E. coli – enterotoxogenic

dose 4–8 weeks

booster given 2 weeks

(ETEC) strains K99,

precalving, 2nd dose

before calving

F17 (= Y), F41

2–4 weeks later – at

– septicaemic strain 31A

least 2 weeks before

calving

All these vaccines depend upon the calf receiving passive immunization from the cow via the colostrum – adequate colostrum must be fed for at least 10–14 days.

im, intramuscular; sc, subcutaneous.

vaccines has cast doubt on their effectiveness. The in

Following these primary pathogens, damaged lungs

utero vaccination of the fetus has been achieved exper-are invaded by secondary bacterial pathogens of which

imentally with some success, but this is not a practical

the principal organism is Mannheimia haemolytica

method at present.

(formerly Pasteurella haemolytica) which causes severe Serovaccines containing

mixed antisera to E. coli,

lung damage, pneumonia and even death. Further bac-

Salmonella dublin and Salmonella typhimurium used to terial involvement follows, usually Pasteurella multocida available and details of their use can be found under

cida and Arcanobacterium (Actinomyces) pyogenes, salmonellosis in the first edition of Bovine Medicine.

resulting in chronic lung pathology.

BRD can be controlled by the strategic use of vac-

cines. There are many and those that are available in

Respiratory disease

the UK are summarized in Table 59.3. Often the use of one or two vaccines is sufficient to control the disease.

Bovine respiratory disease complex (see

Chapter 17)

PI3 and IBR

Bovine respiratory disease complex (BRD) is a multi-

There are individual live vaccines available against PI3

factorial disease which can be summarized as follows.

and IBR. These are freeze-dried preparations which are

The disease complex is triggered by various stresses

administered intranasally. The calf is held with its head

such as transport, housing, the weather, general cattle inclined and the 2 ml reconstituted dose is instilled into handling or by immunosuppression by bovine virus one nostril. A short time is allowed for the vaccine to diarrhoea virus (BVDV). These stresses lead to infection flow into the upper respiratory tract where it stimulates by primary pathogens which cause lung damage the development of local immunity (IgG) in the upper and disease. These primary pathogens are bovine respiratory mucosa. These vaccines are temperature piratory syncytial virus (BRSV), parainfluenza-3 virus specific and will not replicate at the high temperatures (PI3), infectious bovine rhinotracheitis virus (IBR) or of the inner tissues. The live PI3 vaccine can be used in *Mycoplasma bovis*.

the face of an outbreak as a therapeutic agent.

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Table 59.3

Vaccines against BRD complex available in the UK, 2002.

Name of

Company

Type of

Method of

Vaccination

Comment

Duration

vaccine

vaccine

administration

programme

of immunity

Bayovac

Bayer

Live IBR marker (gE

in

Calves from 2 weeks

Intranasal dosage Booster every

IBR-Marker

negative)

im

of age 2 doses 3–5

if specific local

6 months

Vivum

weeks apart

protection needed

(1 ¥ in, 1 ¥ im) in

acute

infections

Calves from 3 months

For use in herds

of age 2 doses 3–5

with uncertain

weeks apart (2 ¥ im)

infection status

and new reactors

Bayovac

Bayer

Inactivated IBR

sc

Cattle from 3 months

For use in non-

Booster every

IBR-

marker (gE

of age 2 doses 3–5

infected herds

6 months

Marker

negative)

weeks apart

Inactivatum

Bovilis

Intervet

Live avirulent strain

in (preferred)

From 4 weeks of age,

Full protection

Annual booster

IBR

of IBR virus

or im

2nd dose at 12 weeks

in 2 days (in), 7

or single dose from

days (im)

12 weeks

Bovilis

Intervet

Live avirulent strains

in

From 4 weeks 2nd

Annual booster

IBR and PI3

of IBR and PI3

dose at 12 weeks

or single dose from

12 weeks, special

situation for early

protection from 1 week

Bovilis

Intervet

Live IBR marker

in or im

From 2 weeks with

Booster every 6

IBR-Marker

(gE negative)

second dose at or

months

after 3 months, or single

dose given if over 3–4

months old

Bovipast

Intervet

Inactivated strain

sc

From 2 weeks of age

BRSV – 5

RSP

BRSV

2 doses separated

months

PI3

by 4 weeks

M. haemolytica

Mannheimia

6 weeks

haemolytica

serotype AI

Immuresp

Pfizer

Live PI3 virus strain

in

From 3 weeks with

RLB 103

second dose at 10

weeks or single dose

after 12 weeks,

special situation from

1 week

Immuresp

Pfizer

Live PI3 virus strain

in

From 3 weeks with

Animals may

RP

RLB 103 live IBR

second dose at 10

remain

virus strain RLB 106

weeks or single

seropositive to

dose after 12 weeks

IBR

Pastobov

Merial

Inactivated

im or sc

From 4 weeks with

Booster dose

adjuvanted

second dose 3–4

before each risk

Mannheimia

weeks later

period at least

haemolytica type AI

annually

Rispoval

Pfizer

Modified

im

From 7 days of age,

RS

live bovine

repeat doses at

respiratory

intervals of 3 weeks

syncytial

and then at 16 weeks

virus

or over 16 weeks with

second dose after 3

weeks

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Table 59.3

Continued

Name of

Company

Type of

Method of

Vaccination

Comment

Duration

vaccine

vaccine

administration

programme

of immunity

Risposal

Pfizer

Inactivated

in

Single dose from 12

Full protection in

4 Months

Pasteurella

adjuvanted

weeks of age

7 days

Mannheimia

haemolytica biotype

A serotype 1

Rispoval 4

Pfizer

Live strains of BRSV

in

From 3 weeks of age

Full protection in

and PI3 virus inactivated

with 2nd dose 3 weeks

2 weeks

adjuvanted IBR

later and 3rd dose at

and BVD virus

12 weeks or from 12

weeks 2 doses 3–4

weeks apart

Tecvax

Vetoquinol Inactivated

im

From 4 weeks, 2

6 Months

Pasteurella

Mannheimia

doses separated by 3

1/6

haemolytica

weeks, vaccination of

serotypes

pregnant or lactating

A1 and A6

animals is not

recommended

Torvac

Vericore

inactivated

sc

Calves under 9 weeks

ajuvanted

3 doses at 3 week

BRSV

intervals

Calves over 9 weeks 2

doses 3 weeks apart

Tracherine

Pfizer

Live IBR virus

in

From 3 weeks with

Annual booster

strain RLB106

2nd dose at 10–12

weeks or single dose

after 12 weeks

Grovax

Intervet

Polyvalent

sc

Calves from 1–2 days

serovaccine

old or on arrival also

containing

weaner calves

serotypes of E.

Pregnant cows, dosages

coli, Salmonella

repeated at 10-14 days

dublin, Salmonella

or 14 days before

typhimurium

calving, provides active

Pasteurella

and passive immunity,

multocida (Roberts)

increase dose to

type 1, 2, 3 and 4)

debilitated or infected

calves

in, intranasal; im, intramuscular; sc, subcutaneous.

Bovine respiratory syncytial virus (BRSV)

transit or shipping fever . M. haemolytica is a common

and Mannheimia haemolytica

commensal of the pharynx of normal calves. Stress and viral infection lead to the invasion of the lung by
There are both live and attenuated vaccines available
this organism. Multiplication of the organism causes the
against BRSV.

release of leucotoxin which lyses responding leucocytes
Mannheimia haemolytica types A1 and A6 are the
with the corresponding release of toxic cytoplasmic
principal secondary invaders of damaged lung and also
proteins which cause further lung damage. There are
the most frequently isolated organisms from cases of
vaccines available against M. haemolytica. They are

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vivum

form 2 weeks of age im (from 3 months of age in or im)
(live vaccine)

in

3–5 weeks

6 months

6 months

im

apart

inactivatum

from 3 months of age

(killed vacc)

sc

3–5 weeks

6 months

6 months

apart

INITIAL

BOOSTER VACCINATION

Fig. 59.1

The vaccination schedule

IMMUNISATION

EVERY 6 MONTHS

with IBR marker vaccines.

inactivated, can be used at various ages and produce

The vaccine is based on a glycoprotein E (gE) deleted

immunity of varying duration. They are all effective.

herpesvirus 1 strain. A killed virus as well as a modified

Combined and multifactorial vaccines are also produced. The live virus marker vaccine have been developed; this enables differentiation between immunized and BHV-1 infected cattle. The vaccine is based on the principle with the live BRSV vaccine to give a more complete cover. From Table 59.3 it can be seen that there are several multifactorial vaccines available. From the author's experience these are all very effective and a ELISA test, animals can be distinguished by testing serum or milk. This marker vaccine and the serological diagnostic kit can be used to eradicate IBR both on-farm situations. It is important to be sure that full protection is in place before the period of stress or risk; farms and nationally. for example, before the housing of spring born suckler

There are four steps to establishing an IBR-free herd:

calves and their dams in the autumn in the UK.

(1)

Determine the status quo – all animals are tested using gE-ELISA to ascertain the serological status

Infectious bovine rhinotracheitis (see p. 289)

of the herd.

(2)

Immunization – all animals are vaccinated with the Infectious bovine rhinotracheitis (IBR) is caused by marker vaccine and so given protection against bovine herpesvirus 1 (BHV-1) and is responsible for clinical signs (Fig. 59.1). Where there is an acute enormous economic loss in worldwide cattle production risk of infection the live vaccine should be used.

BHV-1 is responsible for various clinical

(3)

Stabilization – natural wastage steadily reduces syndromes:

the number of ‘field virus’ infected animals in the

- *Infectious bovine rhinotracheitis (IBR)*

herd. The natural herd replacements are 'field

- *Infectious pustular vulvovaginitis (IPV)*

virus' free.

- *Infectious balanoposthitis (IBP)*

(4)

IBR freedom – once the last serological positive

- *Abortion*

animals leave the herd, the herd is IBR negative

- *Conjunctivitis and keratoconjunctivitis*

but protected by the vaccine.

- *Meningo-encephalitis (now classified BHV-5)*

- *Enteritis*

- *Dermatitis*

Haemophilus somnus infection

Vaccines against IBR have been available for some

(see pp. 240, 737, 907)

years and are listed in Table 59.3. Whilst they are useful

for the protection of all ages of cattle, they will not

Haemophilus somnus is considered by many to be the

protect cattle incubating the disease and animals may

aetiological agent of *Haemophilus septicaemia* in cattle. remain seropositive after vaccination. The latter point The disease is recognized in the USA and also in the UK is important when the animal's disease status needs to and Europe. A vaccine is available in America. It is a be considered in the future, for example with the export killed bacterin containing whole bacteria adhered on to of animals or in cattle health schemes.

an aluminium hydroxide adjuvant. It is administered by The recent development of IBR-Marker vaccine is a subcutaneous injection and two doses are given at a two real step forward in the control and eradication IBR. to three week interval. Immunity is not long-lasting.

Vaccines and Vaccination of Cattle • 1011

Various vaccination programmes have been tried in least eight weeks old when dosed and a second dose is feedlot conditions and their efficacy is difficult to evaluate. Some success is claimed for protection in the face of exposed to any potential source of lungworm infection an outbreak. No claims are made on the efficacy of the

for at least two weeks after the second dose. During vaccine in the reproductive form of the disease.

the following grazing season exposure to lungworm infection reinforces this initial immunity. Vaccinated stock should not be grazed with unvaccinated animals

Contagious bovine pleuropneumonia

or follow behind unvaccinated stock in a grazing system (see p. 868)

since any increase in pasture lungworm burden may cause a vaccine breakdown. Only healthy calves Contagious bovine pleuropneumonia (CBPP) is a should be vaccinated. Careful consideration should be highly infectious disease causing septicaemia with local- given to calves suffering from any respiratory disease ization in the lungs and pleura. It is common in many before dosing. It is advisable not to use any other live countries of the world. Control by vaccination is possible vaccine for a period of two weeks either side of ble and several vaccines are available. They are all live vaccination.

vaccines and are considered by some people to be

The use of anthelmintics should be avoided for at least two weeks after the second dose, particularly the (p. 873) available include the following:

use of sustained-release boluses. Similarly, the use of

- *Pleural exudate from natural cases. This is not a any avermectin must be avoided for eight weeks before satisfactory vaccine since it can cause severe reaction and ten days after the dosing regimen is complete. The*

tion and spreads natural disease.

routine use of other anthelmintics and parasiticides

- *Vaccines produced from culturing the organism must also be avoided for seven days before and at least*

Mycoplasma mycoides var. mycoides.

ten days after treatment. This is due to the residual

- *Vaccines produced by attenuating the organism in effects of modern anthelmintics.*

avian egg cultures.

Following vaccination it is not uncommon for there to be transient periods of coughing; this occurs after

Administration

some seven to ten days. Indeed on rare occasions vaccination can initiate an outbreak of calf pneumonia. Vaccination in the tail is the route of administration by activating latent infection (see p. 239).

using a high pressure syringe. Severe reactions can

It has been suggested that where ivermectin is used occur in the tail, both generalized and localized.

to control lungworm and gastrointestinal worms in a

Intranasal vaccination is now being developed with

control programme then cattle may enter the second

some satisfactory results. Antimycoplasmal drugs

grazing year with no immunity to lungworm. In this

should not be used during vaccination as they will pos-

case vaccination against lungworm may be carried out

sibly reduce the immune response. However, if the side-

before the second grazing season. This is a suggestion

effects are very severe then treatment may be essential.

only as, at present, no trial work on this has been carried

The immunity to the vaccine varies from six to ten

out.

months for some of the cultured broth vaccines to three

to four years for the avian egg attenuated vaccines. All vaccines against CBPP are light susceptible and should

Bovine virus diarrhoea (see pp. 579, 853)

therefore be stored in the dark.

Bovine virus diarrhoea virus (BVDV) is widespread

Parasitic bronchitis

throughout the UK. At the present time there are two (see p. 272)

vaccines available (see Table 59.4), both containing inactivated strains of BVD virus, and one combined

There is a very successful oral vaccine available. It is a vaccine where inactivated BVDV is combined with live vaccine consisting of an aqueous suspension of IBR, PI3 and BRSV (see Respiratory disease on third-stage infective larvae of *Dictyocaulus viviparus* p. 1007).

partially inactivated by exposure to ionizing radiation.

BVDV is endemic in the UK and in many if not most

Each dose contains at least 1000 irradiated larvae (see countries of the world. The arrival of the inactivated p. 274).

BVDV vaccines has allowed the development of control programmes. The BVDV status can be established by Administration

the ELISA testing of bulk milk or blood samples and

Each dose comes in a bottle that should be shaken well

the results of these tests can direct the control/vaccina-

and the contents then given orally. Calves should be at

tion strategy. Table 59.5 shows the interpretation of bulk

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Table 59.4

Bovine virus diarrhoea vaccines available in the UK, 2003.

Vaccine

Company

Type of vaccine

Route of

Vaccination programme

Duration of immunity

administration

Bovidec

Vericore

Inactivated non-

sc

Two doses 3 weeks apart, to

14 Months – booster

cytopathic strain

protect the fetus the vaccination

vaccination

of BVDV

programme should start in the 3

recommended annually

months prior to service and be

completed 1 week before service

Bovilis

intervet

Inactivated cytopathogenic

im

From 8 months of age 2 doses

Annual

BVD

BVD virus strain C86

4 weeks apart; fetal protection

if primary inoculation is at least 4

weeks before gestation

Rispoval 4 Pfizer

See Table 59.3

im, intramuscular; sc, subcutaneous.

Table 59.5

Interpretation of bulk milk BVDV serology and appropriate action. (After Pritchard, 1998.) **Category**

OD ratio

Approximate %

Interpretation

Action

seropositive

Negative

<0.10

<5

Naive

Maintain high level of herd biosecurity;

test all new arrivals in herd; monitor herd

every 3 months; vaccinate if biosecurity poor

Low positive

0.10–0.35

<25

Basically naive; few

*As above; test pooled samples from 1st and
older animals immune*

2nd calved cows; if naive, vaccinate them

Mid positive

0.35–0.70

25–65

Some naive animals

*(probably youngest half
of herd); some immune
animals (probably older
cows)*

Positive

>0.70

>65

Likelihood of PI animals

*Check for active Infection by checking each
increases with increase
age group; bulling heifers using paired
in OD ratio*

serology of 6 animals; 1st, 2nd, 3rd calvers

using pooled milk samples; vaccinate

whole herd if active infection found

OD, optical density.

milk serology and possible action to be taken. Serology

Johne's disease (see p. 857)

in BVD-positive herds (suckler or dairy) interpretation

and action are given in Table 59.6.

Vaccines are available for the control of the disease. Two

In all circumstances if herd biosecurity is poor con-

vaccines are commonly available: the Vallee vaccine,

sider vaccinating the whole herd. After several years of

*which contains live *Mycobacterium paratuberculosis**

vaccination the older cows, some of which would be

organisms in a paraffin oil/pumice stone vehicle, and the

persistently infected, will have left the herd and the

Sigurdsson killed vaccine. In the UK the only vaccine

temptation would be to stop vaccinating. If this happens

available contains live non-pathogenic strains of

the herd soon becomes naive and unprotected. Under

**Mycobacterium avium* subsp. *paratuberculosis*. This these conditions there must*

be complete confidence in vaccine is only available from the Veterinary Laboratories Agency in Weybridge. The vaccine causes interference in the interpretation of the tuberculin test. It causes a positive reaction to tuberculin, particularly to BVDV, are in use and there is evidence that these the avian component. It obviously also causes a positive reaction to the Johne's intradermal test where it is used.

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Table 59.6

Results of cohort serology in BVD positive herds. (After Pritchard, 1998.) 9–18 m Heifers

1st Lactation cows

2nd Lactation cows

Interpretation and action

All negative

All negative

Some or all positive

Active infection unlikely unless 1st lactation cows are recently calved; PI animals probably died or culled from herd; monitor 1st lactation cows – maintain biosecurity

All negative

Some or all positive

Most or all positive

Active infection (acute infection/PI animals in milking herd but no PI heifers):

(1) vaccinate heifers

(2) test milking herd for PI animals

Some or all positive

Most or all positive

Most positive

Active infection in herd, as above; PI animals present in heifers:

(1) screen heifers for PI animals

(2) test milking herd for PI animals

(3) vaccinate herd

PI, persistently infected.

The vaccine is given to calves under one month of age. It is given by subcutaneous injection, usually in the dewlap. The dewlap is used as an injection site because the vaccine usually produces a localized reaction; indeed such a reaction should be looked for two weeks

Chicken embryo vaccine after inoculation as it indicates that the vaccine has been 'taken'. Johne's disease vaccine has a very short shelf life of 14 days.

of immunity.

***Rinderpest** (see Chapter 43d; pp. 707–10)*

Measles vaccine

Vaccination against this disease is common practice in areas where it is endemic. There are several good related and human measles vaccine will produce good vaccines available and in theory complete eradication

immunity to rinderpest in all ages of cattle. It is especially possible with a vaccination programme.

cially useful in very young calves, which can be vaccinated at an earlier age than with normal vaccines.

Tissue culture vaccine

Measles vaccine is not affected by the calf's colostral immunity.

Attenuated vaccines manufactured from cell culture have largely replaced other attenuated vaccines. They are safe and effective and produce a long-lasting immunity suitable for all cattle.

Brucellosis (see p. 580)

Abortion and infertility in cattle caused by Brucella

Goat-adapted vaccine

abortus can be very successfully controlled by

This is an attenuated vaccine that produces life-long vaccination. There are two vaccines available, strain 19 immunity. It is useful for Zebu-type cattle. It is a fairly (S19) and strain 45/20 (45/20). Once eradication is in virulent vaccine, which produces severe generalized progress the use of either is limited or prohibited as it

reactions in susceptible stock. The signs seen are
interferes with the interpretation of blood and milk
severe gastroenteritis with high temperatures and loss
antibody tests.
of milk.

Strain 19 vaccine

Rabbit-adapted vaccine

*S19 is a live vaccine and is presented in freeze-dried
This vaccine produces a good immunity that lasts for
form, which is reconstituted with diluent immediately
two years. It can be attenuated to a point where it will
prior to use.*

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Dosage and administration

*never reach conclusive levels. Brucellosis has been
eradicated from many countries by a programme of
The dosage is 5 ml given subcutaneously. It can be given
initial vaccination followed some years later by sero-
to any age of stock from two months of age onwards.
logical screening.*

The ideal time is in calves from two to six months of

age; at this time it confers a good immunity for approximately seven years.

Louping-ill (see p. 770)

Inoculation of adult cattle is very successful in controlling severe outbreaks of abortion. In an infected A vaccine is available for use in cattle in tick-infested herd the incidence of abortion will begin to subside areas where louping-ill is a problem. It is an inactivated 40–60 days after vaccination and most herds will be free tissue culture vaccine prepared from the arbovirus and of infection within two years.

is a thick oily emulsion.

Bulls should not be vaccinated with S19; it does not seem to protect them from infection and has been known to be responsible for the development of an Dosage and administration orchitis with *Brucella abortus* S19 present in the semen.

A dose of 2 ml is given subcutaneously. Because of the thickness of the vaccine, prewarming can facilitate its Side-effects

use. This prewarming must not exceed 37°C for 30

minutes because the vaccine is heat labile. Initially, two

Vaccination reactions: Localized swellings at the site of inoculations are given, not less than three weeks and

injection can occur, especially in adult cattle. These

not more than six months apart, with annual boosters.

reactions are sterile, do not rupture and persist for

The initial course should be completed at least two

many months as fibrous nodules. Generalized systemic

weeks before exposure and in pregnant cows before the

reactions can also occur with high temperatures and

last month of pregnancy.

loss of milk in lactating cows. Heavily pregnant cows

Accidental injection of the vaccine into the operator

have been known to abort and should not be vacci-

can cause severe localized reactions and immediate

nated. Idiopathic gonitis (see p. 458) may be caused by

medical attention should be sought. The oily vehicle can

vaccination.

sometimes cause localized vaccination reactions in

animals, which may persist for some time.

Serological reactions: Following vaccination the sero-

logical titres can remain very high for a long period of time and therefore they can interfere with the inter-

Papillomatosis (warts) (see p. 883)

pretation of serological testing in an eradication

programme. The complement fixation test becomes

Autogenous vaccines can be prepared for the treatment

negative sooner than the serum agglutination test and

of warts in individual animals. In the author's opinion

can be used to identify a post vaccinal reaction from an

their efficacy is doubtful as warts have a tendency to

uninfected cow.

regress spontaneously. Individual vaccines can be pre-

pared by removing 5 g of the lesion from the animal,

Zoonotic risk: The vaccine is live and accidental injec-grinding it up and extracting the virus using glycerine.

tion into cattle handlers and veterinary surgeons can

After sterilization the autogenous vaccine is adminis-

cause severe illness (undulant fever) and so great care

tered to the affected animal in two doses, one month

must be taken when handling the vaccine.

apart.

Strain 45/20 vaccine

Erysipelas (see p. 455)

Strain 45/20 is a killed vaccine.

Erysipelas is a rare disease of cattle but has been associated with clinical arthritis of calves and isolated

Dosage and administration

on post mortem from adults. In an outbreak of disease,

Two inoculations are given by subcutaneous injection

vaccination is possible using the available porcine

to animals over six months of age. There are often

vaccines. The vaccine is killed and produced from

large localized reactions at the site of injection.

cultures of Erysipelothrix insidiosa inactivated with Although said to be non-agglutinogenic there is usually

formalin and adsorbed on the aluminium hydroxide

a rise in serum titres, but these are such that they

gel.

Vaccines and Vaccination of Cattle • 1015

Table 59.7

Vaccines against leptospirosis available in the UK.

Vaccine

Company

Type of

Route of

Vaccination programme

Duration of immunity

vaccine

administration

Leptavoid-H Schering-Plough

Formol killed

sc

For vaccination against

Annual; give booster before

L. interrogans

neck, chest

Leptospira interrogans

period of risk, maternal

serovar hardjo

wall

serovar hardjo, prajitno and

immunity interferes

Leptospira borgpetersenii

*before 5 months so if very
serovar hardjo bovis; two
young calves are vaccinated
doses 4–6 weeks apart
the complete course must
completed before period
be repeated at 5 months
of risk; calves from
5 months old*

Spirovac

Pfizer

Inactivated

sc

For vaccination against

Annual; give booster before

L.

neck

leptospirosis caused by

period of risk

borgpetersenii

Leptospira borgpetersenii

serovar hardjo

serovar hardjo bovis; 2 doses

4–6 weeks apart before

period of risk, calves from

4 weeks old

sc, subcutaneous.

Dosage

spring before turn-out. If it is necessary to vaccinate a

herd at other times of the year the first booster dose

A dose of 2 ml is given by subcutaneous injection. Two

should always be given the following spring and there-

doses are given, separated by two to six weeks. If

after repeated annually. Calves can be vaccinated, but

pregnant cows are vaccinated, with a second dose being

if they are less than five months old then a further initial

given three weeks before the expected calving date,

course must be given after five months as maternal anti-

then there is good passive immunity in the calves via

bodies may interfere with their immune response. All

the colostrum.

stock (including bulls) of unknown leptospiral immune

status which are brought into a vaccinated herd should

Leptospirosis

be isolated, treated with antibiotics and vaccinated.

(see p. 735)

Antibiotics are used to remove leptospiral infection

from the kidneys. The recommended antibiotic is dihy-

Leptospira serotypes are found in all farm animals; they

drostreptomycin at a dosage rate of 25 mg/kg (not cur-

are distributed worldwide and are an important zoono-

rently available in the UK) or amoxycillin at a dosage

sis. In cattle the important species are Leptospira inter-

rate of 15 mg/kg of the long acting preparation. Ideally

rogans serovar hardjo (hardjo prajitno) and Leptospira the new stock should be kept isolated until seven days

borgpetersenii serovar hardjo (hardjo bovis) of which L.

after the second vaccination.

borgpetersenii serovar hardjo is the predominant strain.

It has been suggested that the two vaccines are not

It is only recently, using genetic typing techniques, that

compatible and it is not possible to predict what pro-

the strains previously classified as hardjo prajitno and tection will be afforded when there is a switch from one

hardjo *bovis* have been identified as separate species. to another. Cows can be vaccinated during an outbreak. They are both responsible for abortion and they have to good effect; they are less likely to become infected. *Leptospira interrogans* has been identified as one cause of the milk drop syndrome and shed organisms in their urine. The time for immunity can also be involved as a cause of infertility. In the UK there are two vaccines available, both are associated with leptospirosis varies:

activated. These vaccines are summarized in Table 59.7.

When planning a vaccination programme ideally

- The response with milk drop is rapid.

cattle should be vaccinated before the period of greatest risk, which in the UK is when they are out at grass.

- Abortions may continue for some months – as the incubation period associated with leptospiral abortion is 3 months.

Cows in the UK should, therefore, be inoculated in the

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• Infertility – once diagnosed as a cause of infertility

clinical signs. It is best considered as an aid to the the herd should be vaccinated as soon as possible control of coliform mastitis, but there will never be a but it can be 12 months for the full effect to develop. substitute for good dairy cow management. The cost-benefit of its use has been assessed. Herd vaccination Cattle that have been vaccinated may still be carriers of has been shown to be profitable when more than 1 per leptospire as a result of prevaccination infection and cent of cows in the herd are affected with clinical coliform mastitis as such are still a zoonotic risk. They will also show iform mastitis.

positive to diagnostic serology, which may be important when cattle are to be exported or screened in a health scheme.

Foot-and-mouth disease

(see Chapter 43c; pp. 700–707)

Mastitis

This is an acute and very contagious viral disease of

Coliform mastitis *(see pp. 334, 383)*

cloven footed animals. The aetiological agent of foot-

and-mouth disease (FMD) is a picornavirus of which
The development in the USA in the early 1990s of the
there are seven serotypes and very many subserotypes
E. coli J5 vaccine and its subsequent release in the UK
(>80). The three commonest serotypes are A, O and C,
in 2000 has been a great advance in the control of
whilst the others are SAT1, SAT2, SAT3 and Asia1.
coliform mastitis. The principle of the J5 vaccine is the
Vaccines are available and many cattle throughout the
mutation of *E. coli* 0111:B4. This strain lacks the O
world are vaccinated. All the vaccines are produced
antigen found in the outermost part of the cell wall and
from virus grown in tissue culture, on bovine tongue
so the core polysaccharide antigen is exposed. This core
epithelia or more commonly on baby hamster kidney
antigen is strongly immunogenic and is common to
cells. The virus is inactivated using formalin or other
many Gram-negative bacteria. The bacteria most often
agents and adjuvanted.

associated with coliform mastitis are *Escherichia coli*, A single dose gives
immunity in 7 to 21 days; this is

Klebsiella species and Enterobacter aerogenes, but E. coli is predominant.

nity lasts from four to eight months. Young stock can

*The vaccine is given by subcutaneous injection in the
be vaccinated from a very early age (<3 months).*

neck and three doses are recommended (see Fig. 59.2).

However, if they come from vaccinated cows they

*The first dose is given to cows at drying off and heifers
two months before calving. This is followed by a second
dose four weeks later and a third dose two weeks
after calving. The protection afforded by this regime*

Table 59.8

*The response to vaccination with J5. The trial
increases with each inoculation and is 10 per cent, 30–40
involved 460 cows in the first 90 days of lactation.
per cent and 60–80 per cent, respectively. Field studies
in the USA have demonstrated the value of this vaccine*

Vaccinated J5

Non-vaccinated

(see Tables 59.8 and 59.9). In another trial involving 845

cows over one year after vaccination relatively similar

No. of cows

233

227

results were obtained (see Table 59.9).

No. of cows showing

6

29

The vaccine does not claim to totally prevent

clinical coliform

intramammary infection in the first 100 days of lacta-

mastitis

tion, but it does significantly reduce the severity of

Incidence (%)

2.57

12.77

DRYING OFF

CALVING

1st dose

2nd dose

3rd dose

Fig. 59.2

Vaccination schedule for J5

–2 months

–1 month

+2 weeks

Time

vaccine.

Vaccines and Vaccination of Cattle • 1017

Table 59.9

*The number of coliform cases in 845 cows over a
is considered to be sufficient for strategic vaccination
one-year period with or without use of the J5 vaccine.
purposes.*

Vaccinated J5

Non-vaccinated

(placebo)

Disadvantages of vaccination

No. of cows

424

421

(1)

Current vaccines are sometimes not very effective.

No. of confirmed

16

45

A high challenge may overcome immunity and cases of E.

there can be partial vaccination breakdown. This coli mastitis

has occurred in Saudi Arabia where vaccinated

Incidence (%)

3.77

10.69

animals were found to have oral lesions.

(2)

Symptomless carriers can occur, especially where the disease is very common.

(3)

It is difficult to distinguish the immune titre should be revaccinated again at four and five months derived from vaccination from that produced by and possibly again at six months of age. This is due to

natural infection. It is possible, however, by measuring maternally derived colostral antibody.

uring non-structural protein antibody which is

The vaccines are usually marketed as monovalent produced in greater amounts following infection, vaccines, for example type A or type O. Ideally, the but this is not always reliable.

vaccine used should be homologous with the serotype
(4)

Vaccination only protects against FMD for a short or strain of the virus found in an outbreak. In many time (two to six months). The time varies with cases, where a new virulent subserotype is isolated different vaccines and with individual response. the vaccine may be manufactured specifically from this
(5)

Repeated vaccination can lead to anaphylactic locally isolated virus. In countries where more than reactions in some cases, although the reported one serotype causes disease then bi- and trivalent incidence is low.

vaccines are used. Live vaccines are available but

(6)

*On occasions following vaccination lesions similar
are not in common use since there is a narrow safety
to FMD may develop.*

margin between loss of virulence and loss of

(7)

*In an FMD-free country when vaccination is used
immunogenicity.*

*to control and eradicate the disease then the time
taken for international recognition of disease-free*

Vaccination programmes

*status is extended. There are agreed definitions for
the designation of FMD disease-free status. These*

These need to be planned with specific local knowledge.

are laid down in Articles of the Office Interna-

In some countries adult stock are vaccinated annually

tional des Épizooties (OIE). Many countries with

and young stock every six months, whereas in others

endemic FMD have no alternative but to control

such as South America or Saudi Arabia all cattle are

the disease by mass vaccination.

inoculated every four months or less.

Where an outbreak of FMD occurs within a disease-free area a ring vaccination policy can be employed to prevent the spread of infection – for example Holland

Ringworm (see pp. 878–80)

in 2001. Following a single dose of vaccine, immunity

will develop in 7 to 21 days, but if a high potency vaccine

A vaccine is available in Europe; it was originally devel-

is used immunity to aerosol challenge can develop

oped in Russia. It is a live attenuated strain (LTF-130)

within four days. This immunity would be short lived

of Trichophyton verrucosum, presented in a freeze-

but may be sufficient to control the disease for long

dried form. Calves as young as two weeks can be

enough to consider the next steps, which may be to give

vaccinated. The inoculation is given by intramuscular

a second dose of vaccine or to slaughter out all the ‘ring

injection; the neck is the recommended site and two

vaccinates’.

doses are required, 10–14 days apart. Initially the whole

Frontier vaccination can also be employed to prevent herd should be vaccinated and then, in a closed herd, transmission from one country or area to another. A only the young calves need to be injected. All new buffer zone is created between an infected and a non-animals introduced to the herd should be given a full infected area.

vaccination course. Once vaccinated, booster injections In Europe, in January 2000, a European vaccine bank are not required. Heavily pregnant cows (7 months +) was established to hold stocks of FMD virus antigen. should not be vaccinated. Occasionally a small crusty Originally in three locations, but now in two, it holds in lesion may develop at the site of injection. Cattle stock five million doses of type O Manisa vaccine. This infected with or exposed to ringworm should not be

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vaccinated, as such animals can develop very severe Vesicular stomatitis (see Chapter 43e; pp. 710–13) signs of ringworm.

Vesicular stomatitis (VS) is endemic in Central and

*South America and also occurs in the southern and western states of the USA. Its importance really lies in **Rabies** (see Chapter 70; p. 908)*

its confusion with FMD. Serologically there are two principal strains, New Jersey (NJ) and Indiana (IND). Rabies is an invariably fatal neurotrophic disease of all Modified live virus and formalin-killed virus vaccines warm-blooded animals. A vaccine is available, prepared are available against VS-NJ. The dead vaccine reduces from inactivated rabies virus adjuvanted with aluminium phosphate. It is used in dogs, cats and horses as immunity. Inactivated autogenous vaccines prepared well as cattle. Vaccination is advised from six months of from clinical outbreaks have also been used.

age. The primary dose is given by intramuscular injection and boosters are given annually. Calves can be vaccinated from two months of age, but at this age a booster at six months of age is recommended. Immunity develops in 30 days. In cattle, treatment with rabies

References and further reading

nity develops in 30 days. In cattle, treatment with rabies

vaccine after exposure to infection is not successful as

Andrews, A.H. (2000) The Health of Dairy Cattle, pp. 1–359.

death usually intervenes before an effective immunity

Blackwell Science, Oxford.

develops.

Andrews, A.H. Blowey, R.W., Boyd, H. & Eddy, R.G. (1992)

Bovine Medicine, Diseases and Husbandry of Cattle, pp.

1–922. Blackwell Scientific Publications, Oxford.

Bishop, Y.M. (1996) The Veterinary Formulary, 3rd edn, pp.

Other vaccines

413–25. Royal Pharmaceutical Society of Great Britain and

British Veterinary Association, London.

Infectious bovine keratoconjunctivitis (see p. 921)

Blood, D.C. & Studdert, V.P. (1988) Bailliere's Comprehensive Veterinary Dictionary, pp. 1–1124. Ballière Tindall, London.

A vaccine against Moraxella bovis is available in some Brownlie, J. (1985) Clinical aspects of the bovine diarrhoea–

countries. It is made by exposing the pili of Moraxella

mucosal disease complex in cattle. In Practice, 7, 195–

bovis to high frequency sound waves (sonification) and 202.

is used to give passive protection. It is not available in

Brownlie, J., Thompson, I. & Curwen, A. (2000) Bovine virus the UK.

diarrhoea virus – strategic decisions for diagnosis and control. *In Practice*, **22**, 176–87.

Gonzalez, R.N. Cullor, J.S., Jasper, D.E., Farver, T.B., Bushnell, Bluetongue (see Chapter 43a; pp. 691–3)

R.B. & Oliver, M.N. (1989) Prevention of clinical coliform mastitis in dairy cows by a mutant *Escherichia coli* vaccine.

Modified live virus vaccines against bluetongue virus

Canadian Journal of Veterinary Research, **53**, 301–305.

are available in some countries. Ideally, the specific

Kahrs, R.F. (2001) *Viral Diseases of Cattle*, 2nd edn. Iowa State serotype vaccine should be used. The practice of using

University Press, Ames.

one serotype followed by a different serotype one

National Office for Animal Health (2001–2002) *Compendium*

month later is adopted and is more effective than using

of Data Sheets for Veterinary Products, pp. 1–866. NOAH, mixed vaccines. However, vaccination interferes with

Enfield.

diagnostic serology used in health schemes and for

Pritchard, G.C. (1998) Making the best use of bulk milk anti-export purposes.

body tests. *Cattle Practice*, **6**, 133–7.

Radostits, O.M., Gay, C.C., Blood, D.C. & Hinchcliff, K.W.

(2000) *Veterinary Medicine*, 9th edn. pp. 1–1877. W.S.

Rift Valley fever (see pp. 769–70)

Saunders, London.

Rinehart, C.L., Lucas, M.J., Cornell, C.P., Rzepkowski,

This is a condition found only in Africa, where vaccines

R.A., Ho, C.H., Peñaredondo, C.C. & Simonson, R.R.

are available. One vaccine is prepared from neuro-

(1996) *Efficacy against bovine mastitis using a J5*

trophic virus passaged through mouse brain. It is known

*Escherichia coli bacterin: Enviracor. In Proceedings of the to cause abortions. A
killed vaccine is also available and*

*XIX World Buiatric Congress, Edinburgh, 8–12 July 1996, is said to produce
good immunity.*

Vol. 1, pp. 285–8.

Chapter 60

Antiparasitics

M.A. Taylor

Introduction

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recently introduced anthelmintics have a broad spec-

Endoparasiticides

1019

trum of activity against a wide range of target helminth

Anthelmintics

1019

parasites.

Classification of anthelmintics

1019

The external ectoparasites affecting cattle are pre-

Others (group 5)

1024

dominantly arthropods and include insects (flies and

Treatment of parasitic gastroenteritis in cattle

1025

lice) and arachnids (ticks and mites). Ectoparasitic

Treatment of parasitic bronchitis

1027

treatments (ectoparasiticides) can be classed as pesti-

Treatment of trematode infections in cattle

1028

Treatment of cestode infections in cattle

1028

cides, insecticides or acaricides according to their activ-

Antiprotozoals

1029

ity. The more recent introduction of the endectocide

Ectoparasiticides

1030

antiparasitics, which have activity against both endo-

Introduction

1030

and ectoparasites, has led to new approaches in the

Ectoparasiticide groups

1030

treatment and control of parasite infections.

Introduction

ENDOPARASITICIDES

The control of parasitic diseases in cattle, as with other

Anthelmintics

domestic animals, relies heavily on the use of antipara-

sitic drugs. A vast array of drugs has been developed

Anthelmintics are widely used in both the treatment

for their antiparasitic activity and these are often clas-

and prevention of internal parasite infections of cattle.

sified according to the target parasites they control. For

Their use has been documented from ancient times

the purposes of this chapter, parasites are classed as

when it was first recognized that certain disease condi-

either internal parasites (endoparasites) or those on the

tions were associated with parasitic helminth infections.

outer body, the skin, dermal tissues or hair (ectopara-

Up to 1938, before the discovery of phenothiazine, few

sites). Endoparasites include a wide and diverse range

useful drugs existed for the treatment of nematode and

of organisms from simple, single-celled protozoa

trematode infections of domestic animals. Since then,

through to large metazoan helminths.

and particularly over the last 30 years, considerable

Protozoan parasites are responsible for several major

advances have been made in the development of safer

economic diseases globally and are classified into

and more effective drugs. Modern, highly effective

several main groups, the most important of which are

anthelmintics with increased safety margins and ease of

the Sarcomastigophora (flagellates and amoebae) and

application have superseded the potentially toxic Apicomplexa (coccidia). Protozoa are found in the chemicals in use less than half a century ago. The introduction in 1961 of thiabendazole, the first of the (GI) tract of cattle. Antiprotozoal agents are generally benzimidazoles, saw the beginning of broad-spectrum referred to by target parasites and include anticoccidi-anthelmintic therapy that has since led to the development of drugs with activity against GI nematodes, lung-Helminths are divided into three main groups: worms, tapeworms and liver fluke. The discovery of the roundworms (nematodes), which parasitize the GI tract avermectins in 1976 has led to the subsequent introduction of a number of endectocidal compounds that GI tract and liver, and the tapeworms (cestodes), which has taken parasite therapy one step further. parasitize the intestines as adult stages or the muscles and viscera as larval forms. Drugs used in the treatment

of helminth infections are generally referred to as

Classification of anthelmintics

anthelmintics, although these can be further subdivided into nematocides, flukicides or taenicides according to On the basis of chemical structure and mode of action, the target parasites they control. Some of the more anthelmintics can be divided into several main groups

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(Table 60.1). These include the broad-spectrum groups mical compounds, with the exception of clorsulon comprising the benzimidazoles (group 1), imidazothiazoles (available in combination with ivermectin) which is a zoles and tetrahydropyrimidines (group 2) and aver-sulphonamide derivative. Flukicides may be formulated mectins and milbemyrcins (group 3).

with a broad-spectrum anthelmintic to increase their Broad-spectrum anthelmintics are highly effective range of activity to include nematodes and flukes. against the majority of nematode species reported in cattle (Table 60.2). Some of the benzimidazoles may

Benzimidazoles and probenzimidazoles

also have activity against liver fluke (Fasciola hepatica) (group 1-BZ)

and cestodes (Moniezia spp.). Avermectins and milbe-

mycins (often collectively referred to as endectocides)

Most anthelmintics available for cattle belong to a

are active against both endoparasites (nematodes and

group of chemicals referred to as the benzimidazoles.

some larval insects) and certain ectoparasites (species

Since their appearance in the early 1960s, the benzimi-

of mites and lice) of cattle.

dazoles have been subjected to continuous structural

Narrow-spectrum anthelmintics are more specific in

modifications to improve their safety and spectrum of

their activity, and are available specifically for liver

activity. Thiabendazole was the first to be introduced

fluke, although individual compounds may also have

in 1961 and was followed by parbendazole (1967),

activity against some nematode species (Table 60.2).

oxibendazole (1973), fenbendazole (1974), oxfendazole

Anthelmintics in this group mainly belong to the

(1975), albendazole (1976), triclabendazole (1981) and

salicylanilide and substituted phenol groups of chericobendazole [albendazole oxide] (1987). Three other chemicals, febantel, netobimin and thiophanate, usually referred to as probenzimidazoles, are also included in this group because they are metabolized in the body by

Table 60.1

Broad-spectrum anthelmintic groups.

ruminal and hepatic pathways to active benzimidazole metabolites. Netobimin is unusual because, as an ionic

Group 1

Benzimidazoles

1-BZ

Affect tubulin

salt, it shows good solubility in water, which offers flex-

Probenzimidazoles

polymerization

leading to cell

ibility for its administration. The benzimidazole group

starvation

is derived from substitution of various side chains and

Group 2

Imidazothiazoles

2-LM Act on

radicals on the parent benzimidazole nucleus, giving

Tetrahydropyrimidines

acetylcholine

rise to individual members of the group. Modification

receptors causing

of a particular benzimidazole can affect the pharmacoparalysis

kinetic behaviour of the drug through changes in rela-

Group 3

Avermectins

3-AV

Affect glutamate-

tive insolubility, slowing the elimination of the parent

Milbemycins

gated Cl⁻ channels

drug and/or active metabolites (Fig. 60.1). The greater

and GABA

efficacy, and wider spectrum of activity, of the most

receptors causing

recently introduced (second-generation) benzimidazole
paralysis

zoles appears to be due to the relative insolubility of

Table 60.2

Classification and activity of anthelmintics available for cattle.

Chemical group

Gut worms

Lungworms

Liver flukes

Tapeworms

Ectoparasites

Broad-spectrum

Benzimidazole and

+

+

±

±

-

probenzimidazole

Imidazothiazoles

+

+

-

-

-

Tetrahydropyrimidines

+

-

-

-

-

Avermectins/milbemycins

+

+

-

-

+

Narrow-spectrum

Salicylanilides and

±

-

+

±

±

substituted phenols

Diphenoxyalkyl ethers

-

-

+

-

-

Organophosphates

+

-

-

-

+

Chlorinated hydrocarbons

-

-

+

-

-

Piperazines

±

±

-

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Others

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clabendazole lacks activity against roundworms and

FEBANTEL

OXFENDAZOLE

(Fenbendazole sulphoxide)

tapeworms but is highly effective against immature and

adult liver fluke. All benzimidazoles and probenzimida-

zoles are ovicidal to nematode eggs. This group of chem-

FENBENDAZOLE

Fenbendazole sulphone

icals is amongst the least toxic of all the anthelmintics available. However, several members of the group have been found to be teratogenic in cattle (parbendazole, NETOBIMIN

ALBENDAZOLE

oxfendazole, albendazole and netobimin) and therefore

Albendazole sulphoxide

Albendazole sulphone

have limitations on their use in pregnant animals.

RICOBENDAZOLE

Benzimidazoles are poorly soluble and are generally given orally as a suspension or as an active ingredient

Fig. 60.1

In vivo metabolic pathways of the common benzimidazole and probenzimidazole anthelmintics in cattle. in a bolus. Netobimin can be solubilized and administered via drinking water. Fenbendazole can also be incorporated into drinking water using specialized these chemicals, which affects the absorption, transport, metering equipment. Benzimidazoles have also been

passage along the GI tract and metabolism within incorporated into a range of controlled release devices, the host, and ultimately the final excretion of the which will be discussed in more detail later (see section anthelmintic compound from the host. The longer on Anthelmintic delivery devices; p. 1026); these the persistence in the animal body, the more effective include pulse-release devices, containing oxfendazole, the anthelmintic appears to be.

and several sustained-release devices available in various countries that contain either fenbendazole or Pharmacokinetics: Following oral drenching, benzimidazole.

dazole anthelmintics usually pass to the rumen which acts as a reservoir, allowing gradual release and absorp-

Mode of action: All members of the benzimidazole tion of the drug into the bloodstream. Thereafter, con- (BDZ) class have a similar mode of action and act by concentrations of the principal circulatory metabolites can disrupting energy metabolism in worms by binding to be found in the plasma from where they are extensively

parasite tubulin, a constituent protein present in micro-recycled between the circulation and the gut wall, along tubules (MT) and in plasma and mitochondrial membranes the whole length of the intestinal tract. Nematodes branes. The formation of MT is a dynamic process attached to the gut wall may be more exposed to the involving the polymerization of tubulin rings at one end recycled drug than to a drug present in the digested (Fig. 60.2) and depolymerization at the other end. BDZ food passing down the intestinal tract.

anthelmintics bind to β -tubulin causing capping and Host physiological factors may affect the efficacy of inhibition of further MT formation. The resultant effect benzimidazoles administered orally. For example, the is starvation of the parasite due to inhibition of glucose oral administration of anthelmintic solutions may result uptake, protein secretion and MT production. There is in closure of the oesophageal groove in some animals, also a reduction in enzyme activity such as acetyl- with subsequent bypass of the rumen and delivery of cholinesterase secretion and in carbohydrate catabo-

the anthelmintic to the abomasum. From here absorption by the fumarate reductase system.

tion, metabolism and excretion of the drug may be

The mode of action of triclabendazole on Fasciola rapid, resulting in reduced exposure of the parasite to

hepatica is at present unknown. It appears to have

the drug and lowered efficacy. In some disease and

limited tubulin-binding properties, unlike other mem-

nutritional states, the pH of the abomasum may be

bers of this group, and it must therefore act along alter-

tered. This may not only reduce exposure of parasites

native pathways.

in the abomasum to some of the anthelmintics, but it

may also reduce solubilization of relatively insoluble

Imidazothiazoles (group 2-LM)

drugs and thus reduce absorption.

Benzimidazoles are highly effective against most of

The two anthelmintic drugs in this chemical group are

the GI nematodes of cattle. The range of activity

tetramisole and levamisole. Tetramisole is a racemic

of second-generation benzimidazoles available for

mixture of dextro and levo forms; levamisole is the cattle, notably albendazole, fenbendazole, netobimin, levo-isomer and it is with this form that anthelmintic oxfendazole and ricobendazole (albendazole oxide), potency resides. The dose rate of levamisole is therefore also includes arrested (hypobiotic) larvae of nematodes half that of tetramisole, and it has twice the safety index. such as *Ostertagia* and also lungworms and tapeworms. Levamisole has good activity against a range of adult Albendazole, netobimin and ricobendazole have activity against adult liver fluke at increased dose rates. Tri- but not against arrested larvae. It is also highly

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Tubulin

polymerization

Benzimidazoles prevent the polymerization of β -tubulin to microtubules leading to cellular dysfunction, inhibition of glucose uptake, glycogen

Fig. 60.2

Benzimidazoles – mode of

depletion and death

Benzimidazole

action.

effective against lungworms. Unlike the benzimidazoles

anthelmintic properties, levamisole has been shown to

it is not ovicidal. Levamisole is non-teratogenic and is

stimulate the mammalian immune system by increas-

therefore safe to use in pregnant animals. The thera-

ing cellular activity. The relationship between the

peutic index in relation to other anthelmintics is,

immunostimulatory and nematocidal properties of lev-

however, low. Animals given levamisole may be hyper-

amisole is unknown.

active for a few minutes after receiving the recom-

mended therapeutic dose. Toxic signs, due to a stimulant

Tetrahydropyrimidines (group 2-LM)

effect on nerve ganglia, may manifest as salivation,

bradycardia, muscular tremors and, in extreme cases,

Pyrantel and morantel are the only members of

death from respiratory failure. Injectable levamisole the tetrahydropyrimidine group available for cattle. may cause inflammation at the site of injection.

Morantel, as its tartrate salt, is the more potent and the Levamisole can be administered orally, by injection most widely available in the form of a sustained-release or pour-on, according to the formulation, and is active bolus. Morantel has higher activity against adult gut against GI worms and lungworms, but not against tapeworms and luminal stages, but not against mucosal or worms or liver fluke. A levamisole sustained-release arrested stages or against established lungworm infection is also available in certain countries. It is combinations. Like levamisole, it has no activity against tapeworms and flukes. Neither morantel nor pyrantel are (oxyclozanide or triclabendazole) to form a broad-spectrum drench for worms and fluke.

and young animals.

Pharmacokinetics: Levamisole is rapidly absorbed and

Pharmacokinetics and mode of action: The pharmacokinetics and mode of action of this group of chemicals are similar to those of levamisole. They act as nerve ganglion stimulants, causing a spastic reversible paralysis of the worms that results in rapid expulsion from the host.

Unlike the benzimidazoles it is therefore not as essential to maintain high drug levels over a protracted

period.

Macrocyclic lactones: the avermectins and milbemyccins (group 3)

Mode of action: Levamisole appears to act as a selective agonist, mimicking the action of acetylcholine (ACh), and affecting transmission of nerve impulses at synaptic and extrasynaptic nicotinic ACh receptors on

Avermectins differ from each other chemically in side chain substitutions on the lactone ring, whilst milbemycin differ from the avermectins through the absence of a sugar moiety from the lactone skeleton. The avermectins, it has also been shown to affect nematode metabolism by inhibition of the fumarate reductase system. and ivermectin, and are active against a wide range of nematodes and arthropods. Moxidectin is a milbemycin anthelmintic efficacy is unknown. In addition to its activity, it has also been shown to affect nematode metabolism by inhibition of the fumarate reductase system. The extent to which this contributes to overall activity is unknown. In addition to its activity, it has a similar wide ranging activity.

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Nicotinic acetylcholine (Ach)

Presynaptic neuron

neuromuscular synapse in

nematode muscle cell

Release of Ach causes depolarisation

*of the post-synaptic membrane
leading to influx of Na^+ and muscle
Imidazothiazoles mimic the
contraction. Ach is removed by the
Ach
action of Ach causing an
action of acetylcholinesterase
Ach receptor
open channel block and
allowing membrane repolarisation
 Na^+
depolarisation of the post-
Acetylcholinesterase
synaptic membrane leading
to (reversible) paralysis*

Fig. 60.3

*Mode of action of imidazothia-
Postsynaptic neuron
zoles.*

*With the exception of eprinomectin, which is only
Injectable and pour-on preparations provide protec-*

available as a pour-on formulation, these groups
tion of 7–42 days for lungworms and 7–35 days for
of drugs are available as both injectable and pour-
stomach worms, depending on the product and formu-
on preparations for cattle. Drench formulations of
lation. The ivermectin bolus provides protection against
abamectin, and a sustained-release device containing
GI worms and lungworms for up to 135 days following
ivermectin, are also available for cattle in some
administration. The prolonged half-life of these com-
countries. As well as being effective against adult and
pounds also determines levels of residues in meat and
larval GI roundworms (including arrested *Ostertagia*
milk, and subsequent compulsory withdrawal periods
ostertagi) and lungworms, most have activity against
following treatment. For meat these may vary between
other helminths of cattle such as the eyeworm,
14 and 42 days according to the product and method of
Thelazia rhodesii, and the filarial parasite, *Parafilaria* administration. With the
exception of eprinomectin,
bovicola. None of these compounds has activity against which has a zero milk
withdrawal period, treatment

tapeworms or liver fluke, although ivermectin is available with this class of compounds cannot be given to lactating cows, or during the last two months of pregnancy. Ectoparasite activity

includes warbles (*Hypoderma* spp.), sucking lice

(*Haematopinus*, *Linognathus*, *Solenopotes* spp.) and Mode of action: The mode of action of the avermectins

mange mites (*Psoroptes*, *Sarcoptes*, *Chorioptes*). More has been studied but has still not been completely elucidated information on the efficacy of the endectocides

identified. Ivermectin is known to act on γ -aminobutyric acid (GABA) neurotransmission at two or more sites against ectoparasites is provided in the section on ectoparasitocides (see p. 1030).

in nematodes, blocking interneuronal stimulation of excitatory motor neurones and thus leading to a flaccid

Pharmacokinetics: Avermectins and milbemycins are paralysis (Fig. 60.4). It appears to achieve this by stimulating the release of GABA from nerve endings and

stored in fat tissue from where they are slowly released, metabolized and excreted. Ivermectin is absorbed systemically following oral, subcutaneous or dermal administration, but is absorbed to a greater degree, and

Presynaptic neuron

has a longer half-life, when given subcutaneously or

These compounds act on

glutamate-gated chloride

dermally. A temporary depot appears to occur in the fat

ion channels on post-

and liver, from which there is a slow release. Excretion,

synaptic membrane of

of the unaltered molecule, is mainly via the faeces, with

inhibitory neurones

less than 2 per cent excreted in the urine. The reduced

Glutamate

or neuromuscular end plate

GABA

absorption and bioavailability of ivermectin when given

Cl⁻

GABA receptor

Glutamate receptor

orally in cattle may be due to its metabolism in the rumen. The affinity of these compounds to fat explains their persistence in the body and the extended periods of protection afforded against lungworms and stomach worms. Individual variances in these periods of protec-

Postsynaptic neuron

tion reflect differences in drug distribution, metabolism and excretion.

Fig. 60.4

Mode of action of avermectins/milbemycins.

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enhancing the binding of GABA to its receptor on

Mode of action: Salicylanilides and substituted phenols the postsynaptic membrane of an excitatory motor

uncouple oxidative phosphorylation and therefore

neurone. The enhanced GABA binding results in an

decrease the availability of high-energy phosphate

increased flow of Cl⁻ ions into the cell, leading to hyper-

compounds such as adenosine triphosphate (ATP) and

polarization. In mammals, GABA neurotransmission

reduced nicotinamide-adenine-dinucleotide (NADH-) is confined to the central nervous system; the lack of in the mitochondria. They have also been shown to effect of avermectin on the mammalian nervous system inhibit succinate dehydrogenase activity and the at therapeutic concentrations is probably because, fumarate reductase system, which is associated with being a large molecule, it does not readily cross the oxidative phosphorylation. Because of the long half-life blood–brain barrier. More recent evidence suggests of the plasma protein-bound molecules, the parasites that ivermectin may exert its effect through action on experience prolonged exposure to the drugs which glutamate-gated Cl^- ion conductance at the postsynap- reduces the energy available to the parasites. Plasma tic membrane or neuromuscular end plate. binding reduces incorporation of the drugs into host tissues and accounts for the selective parasite toxicity. Looseness of faeces and slight loss of appetite may be Salicylanilides and substituted phenols (group 4) seen in some animals after treatment at recommended

*With the exception of niclosamide, the salicylanilides dose rates. High doses may cause blindness and signs of and substituted phenols are usually marketed as uncoupled oxidative phosphorylation, i.e. hyperventilation, being highly effective against adult and, to a lesser extent, immature *F. hepatica*. Some also possess matel death. activity against blood-sucking nematodes such as *Haemonchus*. Most drugs in this group are given orally to cattle, although nitroxylin is usually given by subcutaneous injection.*

Others (group 5)

The salicylanilides, substituted phenols and

Piperazines

bisphenols can be regarded as close analogues and include the bromsalans, clioxanide, oxyclozanide, bro-Diethylcarbamazine is still marketed in certain parts of tianide, rafoxanide and closantel (salicylanilides), the world, for the treatment of lungworm infections nitroxylin (substituted phenol) and bithionol, hexa-

in cattle. It is primarily active against immature chlorophene, niclofolan (bisphenols).

Niclosamide

lungworms and because it has to be given over a period (salicylanilide) is highly effective against tapeworms of three days to achieve its effect, more modern and possibly against immature paramphistomes.

anthelmintics such as the benzimidazoles, levamisole and the avermectins/milbemycins have tended to

Pharmacokinetics: Salicylanilides and substituted replace it. It is also active against microfilariae, the phenols appear to be extensively bound to plasma mode of action being incompletely understood, but it is proteins (>99 per cent), which may explain their high thought to enhance phagocytosis of the microfilariae by efficacy against blood-feeding parasites. Fasciolicidal the host immune system. The action of diethylcarbamazine on immature lungworm larvae is thought to be persist in the plasma. Rafoxanide and closantel have a flaccid paralysis due to hyperpolarization of neuronal

long plasma half-lives when compared with oxy-
postsynaptic membranes resulting from an increased
clozanide. Evidence suggests that the apparent efficacy
flow of Cl^- ions into the cell. This mode of action, similar
of these drugs, particularly against immature *F.*
to the GABA agonist action of piperazine, is probably
hepatica, may be due more to their persistence in the
unrelated to its antiparasitic action.

plasma and the effect they have on maturing adult flukes
when they reach the bile ducts, rather than the effect

Organophosphates

they have on the immature stages themselves. Young
flukes probably ingest mainly liver cells, which contain
Several organophosphorus compounds have been mar-
little anthelmintic. As they grow and migrate through
keted for use in cattle and are still available in a limited
the liver they cause extensive haemorrhage and come
number of countries for the treatment of GI nematode
into contact with anthelmintic. Finally, when the flukes
infections. Haloxon and metrifonate are usually effec-
reach the bile ducts they are in contact with even greater

tive against the adults of some species of GI nematodes, concentrations of anthelmintic as the bile ducts are but not against arrested larvae and lungworms. The important in the excretion of these compounds, as evidenced by the high proportion of these, and their greater detail in the section on ectoparasiticides (see p. 1032).

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Clorsulon

Strategies for controlling helminth infections of cattle (see p. 271 onwards)

The sulphonamide clorsulon is available as an injectable solution for cattle and is highly effective

Strategies for the control of parasitic GI nematode against both adult and immature liver flukes over eight

infections of cattle are generally targeted at the first weeks of age. It has been shown to inhibit the glycolytic

year grazing animal, although increasingly control

*enzyme phosphoglyceromutase in *F. hepatica* by*

measures are being applied to cattle in their second competition with 3-phosphoglycerate and 2,3-grazing season. Currently employed control strategies diphosphoglycerate, leading to a gradual suppression of are based on detailed epidemiological studies con-motility and paralysis.

early part of the grazing season, as a means of limiting pasture contamination and subsequent exposure to Praziquantel is a quinoline-pyrazine compound and is infective larvae later in the season. This approach is a highly effective cestodicidal and trematocidal drug for particularly used in temperate climates where there is use in both human and veterinary medicine. The drug a fairly predictable cyclical appearance of GI nematode appears to modulate cell membrane permeability, species on the herbage throughout the year. The major leading to both spastic muscle contraction and disintegration of strategic single-dose treatments is

gration of the parasite tegument. Praziquantel is active against larval cestodes and schistosomes found in cattle, the cattle. As such, the advent of novel methods of but the high dosage required is probably uneconomic presentation of anthelmintics and those with greater in these infections.

persistence in the body has meant that strategic control of PGE in calves can be achieved with the

Treatment of parasitic gastroenteritis in

minimal handling of stock. The use of pour-on preparations, for example, is convenient, less traumatic to both animal and handler and is gaining widespread

The activities of the available anthelmintics have been acceptance. The most important development over described under their respective anthelmintic groups recent years, and for the foreseeable future, has been and are summarized in Tables 60.2 and 60.3. All are the introduction of controlled-release devices which highly effective against adult and developing larval

deliver anthelmintic at targeted times throughout the stages of the common GI nematodes. When selecting an grazing season.

anthelmintic for treatment of parasitic gastroenteritis (PGE) in calves during the summer months there is little difference, in terms of efficacy, between any of Strategic anthelmintic dosing (see p. 271 onwards)

the currently available anthelmintics. Abamectin, doramectin, eprinomectin, ivermectin and moxidectin

On farms where 'clean grazing' in the form of new leys injections and respective pour-on formulations have or aftermaths is available, GI nematodes can be controlled by integrating systems of grazing management todes and lungworms and can be used at extended with parasite control strategies. These clean grazing treatment intervals in strategic dosing strategies. For systems can be designed for the effective control of treatment of type II ostertagiosis, or for treating PGE, but offer little protection for the control of lung- animals on housing, it is advisable to use a product with

worm because of the unpredictable nature of the high activity against arrested fourth-stage larvae. An anthelmintic that lacks such efficacy may necessitate repeated treatment as it will only remove adult worms where cattle are grazed on permanent pasture, control can be achieved through strategic anthelmintic dosing and developing larvae, leaving arrested larvae to resume development and cause damage to the abomasal wall. Here there is some variability in efficacy between products and evidence suggests that at 0, 8, 13 days post turn-out (sometimes referred to as the 'poor man's clean grazing system'). Further developments of this system include the 3, 8, 13 day dosing strategy for ivermectin, the 0, 8 dosing strategy for doramectin, and the use of boluses (see p. 271). Calves dosed with a bolus or treated strategically with doramectin, eprinomectin, ivermectin and moxidectin anthelmintic should remain set-stocked on the pasture

are more active against arrested fourth-stage larvae or moved to clean pasture, when available, for than other anthelmintics. The activities of the probenz-maximum benefits. Many of the anthelmintic strategies imidazoles, febantel and thiophanate are increased if described claim to protect calves against lungworm they are given in feed over several days.

disease during their first season at grass. There is much

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Table 60.3

Anthelmintics effective against helminth parasites of cattle.

Parasite group

Parasite genus/species

Anthelmintics

Gastrointestinal worms

Ostertagia, *Trichostrongylus*, *Cooperia*, *Albendazole*, *ricobendazole*, *febantel*, *fenbendazole*,

Haemonchus, *Nematodirus*, *Chabertia*,

netobimin, *oxfendazole*, *thiophanate*, *levamisole*, *morantel*,

Bunostomum, *Oesophagostomum*,

abamectin, *doramectin*, *eprinomectin*, *ivermectin*, *moxidectin*

Strongyloides

Closantel and nitroxynil (Haemonchus, Bunostomum,

Oesophagostomum only)

Lungworms

Dictyocaulus viviparus

Albendazole, febantel, fenbendazole, netobimin, oxfendazole,

levamisole, abamectin, doramectin, eprinomectin, ivermectin,

moxidectin

Diethylcarbamazine (available in some countries)

Cestodes

Moniezia expansa, M. benedini

Albendazole, febantel, fenbendazole, netobimin, oxfendazole

Oxyclozanide (some activity against proglottids)

Taenia saginata (metacestode

Praziquantel

Cysticercus bovis)

Echinococcus granulosus (hydatid)

Praziquantel

Trematodes

Fasciola hepatica (adults)

Albendazole, ricobendazole, netobimin, triclabendazole,

nitroxynil, oxyclozanide, clorsulon, closantel

Rafoxanide, bithionol, niclofan

Fasciola hepatica (immatures)

Triclabendazole, nitroxynil (>6 weeks), closantel

Fasciola gigantica

Triclabendazole, clorsulon

Fascioloides magna

Triclabendazole, clorsulon, closantel

Dicrocoelium dendriticum

Netobimin

Schistosoma spp.

Praziquantel

debate as to whether these strategies compromise an ideal site for the location of these devices, which immunity to both GI worms and lungworms and, in can release anthelmintic over an extended period part, explain the increasing reports of lungworm in either continuously (sustained-release devices, SRD), older animals seen over the last few years in parts of or at pulsed intervals (pulse-release devices, PRD). western Europe. On farms where lungworm is endemic,

Ruminal retention is achieved either on the basis of or if there is a high risk of introduction of disease, density or the variable geometric configuration of the then vaccination is the recommended method of device.

control (see p. 1011).

Several devices are available for cattle in certain

The two grazing strategies commonly used for the countries and include:

control of PGE in first-year cattle are commonly referred to as 'preventative' and 'evasive'. In a preven-

- A morantel sustained-release device (MSRD);*
- tative strategy clean grazing is provided at the start of*
- A levamisole sustained-release bolus (LSRB);*
- the grazing season, usually in the form of new leys.*

- A fenbendazole sustained-release bolus (FSRB);*

When weaned, parasite-naïve calves are turned out

- An ivermectin sustained-release bolus (ISRB); and*
- onto the clean pasture where they should remain set-*
- Oxfendazole pulse-release devices (OPRB).*

stocked for the whole grazing season and should not

require any anthelmintic dosing. In an evasive strategy, These range in shape and design, each being administered by the use of specially provided dosing guns. Care from mid-summer onwards (see p. 271).

is required when administering as dosing injuries can easily occur or the bolus may be regurgitated if not swallowed properly.

Anthelmintic delivery devices

The MSRD consists of a coiled trilaminar sheet Intraruminal devices represent a new approach to composed of a central core matrix of morantel tartrate. administering anthelmintics for the control of parasitic The entire sheet is punched with holes which allow the helminths and are becoming a popular labour-saving controlled release of morantel into the rumen from the means of worming cattle. The reticulo-rumen provides edges of the perforation as well as the perimeter.

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The device is retained in a rolled shape by a special cal disease in the following spring. In this situation

tape. Following administration, the tape releases and calves may require dosing in the autumn while still at the device unrolls and is retained in either the first or grass, or on housing, to remove arrested burdens.

second stomach of the animal. The device remains

With some delivery system designs the dose/weight active for about 90 days, after which it gradually dis- ratio reduces as the animals grow, whilst at the same integrates and is eliminated.

time release rates may decline with time. Both of these

The LSRB bolus consists of a compressed polyvinyl factors may result in considerable selection pressure chloride (PVC) matrix containing levamisole encased for the development of drug resistance and since these in an outer impermeable shell. Anthelmintic is released systems are recommended as a form of clean grazing from the internal surface area through a single outer system, the build-up of resistant over susceptible pore over a period of up to 90 days.

species could rapidly occur.

The FSRB is a cylindrical bolus consisting of ten flat-

faced tablets in two magnesium alloy tubes joined and enclosed by alloy rings. The bolus is designed to release Anthelmintic resistance

fenbendazole by galvanic erosion over a period of 140 Although widely reported in sheep, anthelmintic resistance days.

ance appears less of a problem in cattle. This may be a The ISRB consists of an external semipermeable reflection of the relative frequency of treatment and membrane surrounding a semisolid formulation of ivermectin. The bolus is retained in position in the first or between the two hosts. It may also reflect the prolonged second stomach by the presence of an iron densifier at survivability of free-living larval stages within the one end. The bolus is designed to release anthelmintic bovine faecal pat, thus ensuring a supply of susceptible over a period of up to 135 days.

worms.

The OPRB consists of either five or seven annular

The effects of intraruminal devices on the develop-

tablets of oxfendazole, mounted on a central metal
ment of resistance, given the selection pressures they
core with a stainless steel weighted end. The release
exert, are not yet apparent. Until the inheritance of
of each annular tablet is determined by the corrosion
anthelmintic resistance is completely understood, this
rate of the central core. The first dose of oxfendazole
will remain a matter for speculation. In the meantime,
is either released within 24 hours (for use in animals
recommendations aimed at limiting the development of
already at grass) or at approximately three weeks after
resistance in sheep nematodes should be followed
administration for either first (seven tablet) or second
wherever possible. The ultimate choice of anthelmintic
year animals (five tablet) prior to turnout. Remaining
product may therefore be influenced by a number of
doses are released at regular intervals thereafter
factors of which group type or mode of action may
(approximately 21 days) giving the boluses active lives
become one of the overriding factors, when considered
of 105, 126, or 147 days depending on the number of

mazine (available in some countries) is primarily active of the grazing season, although some can be given later against immature larvae if given over a three-day period. in the season. If the animals are turned out early, or Some degree of control of parasitic bronchitis in moved to pasture grazed by untreated animals during calves can be achieved by early season suppression of late summer, subsequent protection from infective pasture contamination in much the same way as for the larvae may be lost, as will any benefits of anthelmintic control of GI nematodes. However, the epidemiology treatment. The result may be that either the calves of lungworm infection is complex and still not fully experience sufficient challenge to develop clinical understood, and it is the opinion of the author that the disease in the autumn or, alternatively, they acquire vaccination of calves with an irradiated larval vaccine large numbers of arrested larvae that may cause clinical is the most reliable form of control (see p. 1011).

of *F. gigantica*. Only triclabendazole and clorsulon are **cattle** (see pp. 276–80)

effective against both mature and immature stages of *F. gigantica*. *Fascioloides magna*, a parasite of deer that Fasciolosis can affect cattle, is susceptible to triclabendazole, clorsulon and closantel.

drugs against *F. hepatica*. All are effective against adult Only limited information is available on the treat-fluke and are therefore suitable for the treatment of

ment of paramphistomes in cattle (see p. 279). Niclochronic fasciolosis in cattle. Few compounds have activity against immature fluke stages, limiting the choice in have only variable activity against intestinal juvenile suspect cases of acute fasciolosis. As a general recommendation for cattle in temperate countries, housed apart, has been shown to be effective against adult cattle should be dosed once in mid-winter in average flukes in the rumen.

rainfall years. Wherever possible, the most effective

*drugs with efficacy against mature and immature flukes
Schistosomes (see p. 280)*

*should be used. Further treatment should then be
unnecessary. If a flukicide with activity against mature
In contrast to the chemotherapy of schistosomes in
flukes only is used, a second dose at turnout may be
humans, little information is available on the treatment
necessary to remove any fluke missed by the first
of schistosomosis in cattle. For economic reasons,
treatment. Where animals were housed for at least eight
anthelmintic treatment is not suitable for the control
weeks prior to the first treatment, the second dose
of schistosomosis in cattle, except during clinical out-
should be unnecessary.*

*breaks when praziquantel may be administered orally
In outwintered stock, two doses of anthelmintic may
at 15–20 mg/kg. Repeat doses two to three days apart
be necessary. The first is given in early winter to remove
are usually required or high dose rates (25/mg per kg)
adult and immature fluke burdens acquired from the
at three to five weeks apart.*

autumnal flush of pasture metacercariae. A second dose in early spring may be required to prevent heavy

Treatment of cestode infections in cattle

pasture contamination and infection of the intermediate snail host, especially if rainfall has been heavy in

Adult stages of tapeworms parasitizing cattle are

previous years. In these situations the treatment inter-

usually of little consequence. Several of the benzimida-

val will depend on the activity of the flukicidal drug

zoles used in the treatment of GI nematodes are also

against immature stages.

effective against cestodes (Tables 60.2 and 60.3).

Certain flukicidal drugs also have cestocidal activity

(Table 60.4). Larval stages of cestodes affecting cattle,

Other *Fasciola* spp. (see p. 277)

notably *Echinococcus* spp. and *Taenia saginata*, are The drugs and dose rates given for the treatment of *F.*

generally refractory to treatment with anthelmintics.

hepatica are also generally applicable for the treatment However, praziquantel has been shown to have activity

Table 60.4

Activities of anthelmintics against liver fluke in cattle.

Chemical group

Chemical

Recommended

Active

Comments

dose rate

against

(mg/kg)

A

B

C

Salicylanilides

Rafoxanide

7.5

+

+

-

No longer available in many countries

Closantel

NR

+

+

±

Available for cattle in some countries

Oxyclozanide

10

+

-

-

Some activity against tapeworm segments

Substituted phenols

Nitroxynil

10

+

+

-

Also active against blood nematodes

Bithionol

30

+

-

-

Also has anticestodal activity

Niclofolan

3

+

-

-

Benzimidazoles

Albendazole

10

+

-

-

Also active against nematodes and cestodes

Triclabendazole

12

+

+

+

Do not give to dairy cows within 7 days of calving

Probenzimidazoles

Netobimin

20

+

-

-

Do not administer to cattle in the first 7 weeks of

Sulphonamide

Clorsulon

7

+

+

±

pregnancy

A, adult fluke; B, 6–12 week old fluke; C, 1–6 week old fluke.

NR, no recommended dose rate.

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against the intermediate stages of both these parasites,

*causative organism, *Cryptosporidium parvum* (see*

although the cost of treatment may preclude its use.

pp. 204, 286), is a significant cause of disease in neona-

tal calves and is also an important zoonosis.

Antiprotozoals

Haemoparasites

Ruminants carry large numbers of protozoa in their

Cattle are infected with a number of haemoprotozoa in the stomach and intestines, the vast majority of which are entirely harmless. Some species of protozoa, however,

piroplasms (Babesia and Theileria) (see p. 748) and the are significant as causes of disease in cattle or because

haemoflagellates (Trypanosoma) (see p. 756). Trans- mission of their potential for zoonotic transmission. Antiproto- mission of these blood parasites normally occurs via a zoonal compounds are listed in Table 60.5.

biting tick or insect vector. Babesia species occur in Protozoal diseases of economic importance through-most parts of the world where there are ticks. They are

out various regions of the world include trypano- of greatest importance in the tropics, but some species somosis, theileriosis, babesiosis and trichomoniasis.

occur in temperate climates where they can cause

Coccidiosis in cattle is caused by parasites of the genus disease in non-immune cattle. The merozoites of Eimeria spp. and can be a serious cause of disease

Babesia occur in the erythrocytes of infected animals in young animals (see p. 282). A protozoan disease

where they multiply asexually by binary fission and of increasing importance is cryptosporidiosis. The are described as 'large' and 'small', depending on size.

Table 60.5

Antiprotozoal compounds for cattle.

Compound

Trade name

Manufacturer

Dose rates

Comments

(mg/kg)

Route

Diminazene aceturate

Berenil

Intervet

3.5–7.0

For treatment of trypanosomosis (*T. congolense*,
im or sc

T. brucei, *T. vivax* [nagana]) and babesiosis (*B.*

bigemina, B. bovis and B. divergens)

Imidocarb

Imizol

Schering-Plough

1–3

For treatment of babesiosis (B. bigemina, B.

(Mallinckrodt

sc

bovis and B. divergens). Prophylactic effect for up Veterinary)

to 8 weeks depending on dose and species

involved. Causes cholinergic effects and is slowly

metabolised and excreted

Homidium chloride

Novidium

Merial

1.0

Treatment of trypanosomosis (T. congolense,

im

T. brucei, T. vivax [nagana])

Isometamidium chloride

Samorin

Merial

0.25–1.0

Treatment or prevention of trypanosomosis

im or iv

(T. brucei, T. congolense, T. vivax [nagana]).

Toxic above 2 mg/kg

Suramin

Naganol

Bayer

10

Treatment of trypanosomosis (T. brucei, T.

iv

congolense, T. vivax [nagana])

Amicarbalide

Diampron

Merial

5–10

Treatment of babesiosis (B. bigemina, B. bovis, im

B. divergens)

Buparvaquone

Butalex

Schering-Plough

2.5

Treatment of East Coast fever (Theileria parva)

(Mallinckrodt

2 doses at 48 h

and Mediterranean Coast fever (T. annulata)

Veterinary)

intervals

Parvaquone

Clexan

Schering-Plough

10–20

Treatment of East Coast fever (T. parva) and

(Mallinckrodt

2 doses at 48 h

Mediterranean Coast fever (T. annulata)

Veterinary)

intervals

Halofuginone

Stenoral

Intervet

1.2

Treatment of East Coast fever (T. parva) and

po 2 doses at

Mediterranean Coast fever (T. annulata)

48 h intervals

im, intramuscular; sc, subcutaneous; iv, intravenous; po, per os (oral).

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Several species are highly pathogenic in cattle and

tion to hygiene. Raising of food and water troughs, for

include Babesia bigemina, found in Africa, Australia,

example, can help avoid contamination by reducing the

Europe and Central and South America, and B. bovis,

levels of infection. Young animals should be kept off

found throughout most of the world and being particu-

heavily contaminated pastures when they are most

larly common in southern Europe. Transmission occurs

susceptible. The anticoccidial decoquinate is licensed

following a bite from an infected tick of the genera

in many countries for the prevention of coccidiosis

Boophilus, Rhipicephalus, Haemaphysalis and Ixodes, in calves. Other compounds, such as monensin and

the species involved varying with geographical location.

amprolium, are available in some countries.

The species found throughout temperate Europe is

Babesia divergens (a small piroplasm), transmitted by

Cryptosporidiosis (see pp. 204, 286)

the tick Ixodes ricinus. Clinically affected animals

develop a haemolytic anaemia leading to haemo-

Bovine cryptosporidiosis is often seen as part of the

globinuria, fever and death.

neonatal diarrhoea complex, symptoms of which are

A number of Theileria species have been reported in

profuse watery diarrhoea, dehydration, and death if left

cattle and are significant causes of disease in many tropi-

cal countries. Infections are usually, although not always,

associated with the presence of other pathogens such

and erythrocytic cells and are transmitted by the bites

as Salmonella spp., E. coli, rotavirus and coronavirus.

of various species of ticks. T. parva (the cause of East Specific antiparasitic

therapy is not available for

Coast fever) and T. annulata (Mediterranean Coast

cryptosporidiosis and treatment relies on the use of fever) are the main pathogenic species occurring in East supportive antidiarrhoeals and fluid therapy for animals and North Africa, southern Europe and parts of the showing dehydration. Recently halofuginone lactate former Soviet States, respectively. Transmission of these has been licensed for the prevention of diarrhoea parasites occurs following the bite from an infected tick caused by *Cryptosporidium parvum* by treating daily belonging to various species of the genus *Hyalomma*. for seven consecutive days.

Trypanosomosis is one of the most important diseases of domesticated cattle in central parts of Africa.

ECTOPARASITICIDES

Trypanosomes are broadly divided into two groups

Introduction

depending on the site of development in the insect (see Chapters 45, 50)

vector. The salivarian trypanosomes, transmitted in the saliva of biting flies, are responsible for 'nagana' in

Ectoparasites can cause intense irritation leading to loss

domestic ruminants (see p. 757), as well as diseases such of condition and effects on performance such as weight as 'surra' of horses and other animals, and African loss, reduced milk yield or hide damage. In addition Sleeping Sickness in humans. The stercorarian try- they may be responsible for transmission of disease. panosomes are transmitted by contamination with The choice and use of ectoparasiticide chemicals and infected insect faeces and include diseases such as the method for their control depend to a large extent Chagas disease in humans in Central and South on husbandry and management practices as well as the America, which is transmitted by reduviid bugs. type of ectoparasite causing the infestation. Parasites that live permanently on the host, such as lice and mites, are relatively easily controlled and, once Coccidiosis (see p. 282) eradicated, reinfestation only occurs from contact with At least 18 different species of coccidia are known to infested stock. Non-permanent parasites (ticks and infect cattle worldwide. Bovine coccidiosis is primarily

flies) are less easily controlled because only a small proportion of young animals aged between three weeks and nine months, but can occur in older cattle. The and other hosts may maintain them.

disease is usually associated with a previous stressful

Most infestations in cattle are seasonal and pre-situation such as shipping, overcrowding, changes of dictable and can be countered by prophylactic use of feed, severe weather or concurrent infections. Clinical ectoparasiticides. For example, biting and nuisance flies signs, often presenting as bloody diarrhoea, are associated with the presence of *Eimeria zuernii* or *E. bovis*, tick populations increase in the spring and autumn, and which occur in the lower small intestine, caecum and lice and mites may increase in winter-housed stock.

colon. Another species of coccidia, *E. alabamensis*, has been reported to cause enteritis in yearling calves in

Ectoparasiticide groups

some northern European countries.

The incidence of coccidiosis can be reduced by avoid-

Available ectoparasiticides act either systemically or by direct contact with the target parasites and have an effect on the ectoparasite's nervous system. Systemic facilities, in addition to labour requirements for such ectoparasiticides may be given parenterally or applied topically to the skin from where the active ingredient is absorbed percutaneously and taken up into the blood circulation. Topical ectoparasiticides have a direct effect on the target parasite on the surface of the skin.

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Organochlorines (OCs) are no longer in general use but several compounds are still available in some countries. Different formulations of a drug preparation may be indicated for different target parasites (Table 60.6).

OCs fall into three main groups:

Both topical and systemically acting formulations can be applied by various methods including dips, sprays,

(1)

Chlorinated ethane derivatives such as 'pour-ons', 'spot-ons', dusting powders or ear tags. Typically, a number of formulations are available as liquid (dichlorodiphenyltrichloroethane), DDE (dichlorodiphenyldichloroethane) and DDD concentrates requiring dilution with water to produce (dicofol, methoxychlor); an emulsion for application by spray or dip. However,

(2)

The cyclodienes that include chlordane, aldrin, more convenient ready-for-use pour-on and spot-on dieldrin, heptachlor, endrin, tozaphene; application methods provide a broad choice of systems

(3)

Hexachlorocyclohexane (HCH), also known as depending upon individual circumstances, for example benzene hexachloride (BHC), which includes the availability of spraying, dipping and animal handling g-isomer lindane.

Table 60.6

Chemicals active against cattle ectoparasites.

Ectoparasite

Parasite species

Ectoparasiticides

group

Biting flies

Haematobia (Lyperosia) irritans, horn fly

Ivermectin, *doramectin*, *eprinomectin*, *moxidectin*, *a*

Stomoxys calcitrans, stable fly

cypermethrin, *deltamethrin*, *fenvalerate*, *permethrin*

Simulium spp., blackfly

Nuisance flies

Hydrotaea irritans, head fly

Cypermethrin, *deltamethrin*, *fenvalerate*, *permethrin*

Musca autumnalis, face fly

Musca domestica, house fly

Morellia simplex, sweat fly

Warble flies

Hypoderma bovis

Abamectin, *doramectin*, *eprinomectin*, *ivermectin*, *moxidectin*,

Hypoderma lineatum

famphur, *fenthion*, *phosmet*

Sucking lice

Haematopinus eurysternus

Abamectin, doramectin, eprinomectin, ivermectin, moxidectin,

Linognathus vituli

phosmet, cypermethrin, deltamethrin, fenvalerate, permethrin,

Solenopotes capillatus

Amitraz

Closantel

Biting lice

Bovicola (Damalinia) bovis b

Doramectin,b eprinomectin,b ivermectin,b moxidectin,b famphur, fenthion,
phosmet, cypermethrin, deltamethrin, fenvalerate,

permethrin, amitraz

Mites

Psoroptes bovis

Abamectin,c doramectin,c eprinomectin,c ivermectin,c

Sarcoptes scabiei

moxidectin,c phosmet, amitraz

Chorioptes bovis

Ticks

Ixodes spp.

Amitraz

Boophilus spp.

Flubenzuron

Rhipicephalus spp.

Closantel

Ambylomma spp.

Dermacentor spp.

Hyalomma spp.

*a Pour-on preparations with activity and protection against horn flies [
Haematobia (Lyperosia) irritans]*

*b Efficacy varies depending on method of application. Injectable preparations
aid in the control of biting lice.*

*c Efficacy varies depending on method of application. Injectable preparations
aid in the control of chorioptic mange.*

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Chlorinated ethanes cause inhibition of sodium con-

Pyrethrins and synthetic pyrethroids

ductance along sensory and motor nerve fibres by

Natural pyrethrins are derived from pyrethrum, a

holding sodium channels open, resulting in delayed

mixture of alkaloids from the chrysanthemum plant.

depolarization of the axonal membrane. This state

Synthetic pyrethroids (SPs) include bioallethrin, cypermethrin, deltamethrin, fenvalerate, flumethrin, lambda-cyhalothrin, phenothrin and permethrin. The content of potential in a fully repolarized neuron.

some SPs is also expressed in terms of the drug isomers;

The cyclodienes appear to have at least two com-

*for example, cypermethrin preparations may contain
ponent modes of action:*

varying proportions of their cis and trans isomers. Thus,

- Inhibition of γ -amino butyric acid (GABA)-*

cypermethrin (cis : trans 60 : 40) 2.5 per cent is equivalent to cypermethrin (cis : trans 80 : 20) 1.25 per cent. In

- Interference with Ca^{2+} flux.*

*general terms, cis isomers are more active than the corresponding trans isomers.
The mode of action of SPs*

*The resultant inhibitory postsynaptic potential leads
appears to be an interference with sodium channels of
to a state of partial depolarization of the postsynaptic
the parasite nerve axons, resulting in delayed repolar-*

membrane and vulnerability to repeated discharge. A
ization and eventual paralysis. The lethal activity of
similar mode of action has been reported for lindane,
pyrethroids seems to involve action on both peripheral
which binds to the picrotoxin side of the GABA recep-
and central neurones, while the knockdown effect is
tor resulting in an inhibition of GABA-dependent Cl-
probably produced by peripheral neuronal effects only.
flux into the neuron.

Pour-on, spot-on, spray and dip formulations are
available in many countries with activity against biting
and nuisance flies, lice and ticks. Some compounds are
Organophosphates and carbamates
active against sarcoptic and chorioptic mange mites.
For non-permanent ectoparasites, treatments are gen-
Organophosphates (OPs) are neutral esters of phos-
erally applied during periods of anticipated peak chal-
phoric acid or its thio analogue and act by inhibiting the
lenge. Treatments for lice are best administered in
action of acetylcholinesterase (AChE) at cholinergic
the autumn. Periods of protection against flies vary

synapses and at muscle end plates. This results in an
between products and methods of application, but gen-
accumulation of acetylcholine (ACh) at the postsyn-
erally last for up to one month for most species of flies.
aptic membrane, leading to neuromuscular paralysis.
Some SPs are also available as ear tags for use on cattle,
OP compounds include azamethiphos, chlorpyrifos,
providing protection against biting and nuisance flies
coumaphos, dichlorvos, diazinon, fenitrothion, hep-
for several months during the fly season. One tag is
tenophos, metrifonate, phoxim, propetamphos or
usually sufficient but in severe cases of head or face fly
tetrachlorvinphos and there are many preparations
attack, both ears can be tagged.

available worldwide for cattle. Carbamate insecti-
cides are closely related to the OPs and are also
anticholinesterases but, unlike OP compounds, are
Amidines

spontaneously reversible. The two main carbamate
The main member of this group is amitraz, which acts
compounds in use in veterinary medicine are carbaryl

at octopamine receptor sites in ectoparasites giving rise and propoxur. OP compounds can be extremely to increased nervous activity. When applied as a spray toxic in animals and humans. Inhibition of AchE, and it is active against mites, lice and ticks in cattle. In severe other cholinesterases, can occur in higher vertebrates as cases of mange, lice and tick infestations, treatments well and accounts for host toxicity seen at higher dose may have to be repeated during the risk period. rates. Typical signs of overdosage, or overexposure, include salivation, miosis, diarrhoea and muscle Avermectins and milbemycins tremors. Death may occur through respiratory failure. Toxic clinical signs can be treated with atropine. Avermectins and the structurally related milbemycins Chronic exposure may lead to damage to the nervous have been discussed earlier under anthelmintics. As system and clinical signs including headaches, anxiety well as being effective against GI roundworms and and irritability in operators. OP compounds are gener- lungworms, all have activity against warbles, sucking

ally active against warble fly larvae, flies, lice, ticks and

lice and mange mites. Control against the biting louse,

mites on cattle, although activity will vary between

Bovicola (*Damalinia*) *bovis*, the mite, *Chorioptes bovis*, compounds, formulations and against different target

and the hornfly, *Haematobia* (*Lyperosia*) *irritans* varies parasites.

between products and the method of administration.

Antiparasitics • 1033

Insect growth regulators

Caldow, G.L., Taylor, M.A. & Hunt, K. (1989) Comparison of two early season anthelmintic programmes on a commer-Insect growth regulators (IGRs) are a group of chemi-

cial beef farm. *Veterinary Record*, **24**, 111–14.

cal compounds that do not kill the target parasite

Campbell, W.C. (1985) Ivermectin: an update. *Parasitology*

directly, but interfere with growth and development.

Today, **1**, 10–16.

IGRs act mainly on immature stages of the parasite and

Campbell, W.C. (1986) The chemotherapy of parasitic infec-

as such are not usually suitable for the rapid control of

tions. *Journal of Parasitology*, **72**, 45–66.

established adult populations of parasites. Where para-

Craig, T.M. & Hucy, R.L. (1984) Efficacy of triclabendazole sites show a clear seasonal pattern, IGRs can be applied

against *Fasciola hepatica* and *Fascioloides magna* in natu-at the start of the grazing season as a preventative

rally infected calves. American Journal of Veterinary Research, **46**, 1644–5.

measure. Based on their mode of action they can be

Donald, A.D. (1985) New methods of drug application for

divided into chitin inhibitors and juvenile hormone

control of helminths. Veterinary Parasitology, **18**, 121–37.

analogues. Chitin inhibitors appear either to interfere

Eagleson, J.S. & Bowie, J.Y. (1986) Oxfendazole resistance with the assembly of chitin chains and microfibrils or to

in *Trichostrongylus axei* in cattle in Australia. Veterinary inhibit the deposit of chitin into the cuticle. The juve-Record, **119**, 604.

nile hormone analogues mimic the activity of naturally

Egerton, J.R., Suhayda, D. & Eary, C.H. (1986) Prophylaxis of occurring juvenile hormones and prevent metamor-nematode infections in cattle with an indwelling rumino-

phosis to the adult stage.

reticular ivermectin sustained release bolus. Veterinary

IGRs are widely used for flea control in domestic pets

Parasitology, **22**, 67–75.

and for blowfly control in sheep, but have limited

Eysker, M. (1986) The prophylactic effect of ivermectin treatment in cattle. The phenoylbenzyl urea flubenzuron is ment of calves three weeks after turnout, on gastrointestinal helminthiasis. *Veterinary Parasitology*, **22**, 95–103.

marketed in Australasia as a tick development inhibitor.

Fairweather, I. & Boray, J.C. (1998) Mechanisms of fasciolin-When applied as a pour-on it provides long-term procide action and drug resistance in *Fasciola hepatica*. In taction against the one-host tick *Boophilus microplus*.

Fasciolosis (ed. by J.P. Dalton), pp. 225–76. CABI Publishing, Wallingford.

Fairweather, I. & Boray, J.C. (1999) Fasciolicides: efficacy, actions, resistance and its management. *The Veterinary*

Further reading

Journal, **158**, 81–112.

Geerts, S., Brandt, J., Kumar, V. & Biesemans, L. (1987)

Anderson, N. (1985) Controlled release technology for the

Suspected resistance of *Ostertagia ostertagi* in cattle to control of helminths in ruminants. *Veterinary Parasitology*, levamisole. *Veterinary Parasitology*, **23**, 77–82.

18, 59–66.

Grimshaw, W.T.R., Weatherley, A.J. & Jones, R.M. (1989)

Armour, J., Bairden, K., Pirie, H.M. & Ryan, W.G. (1987)

Evaluation of the morantel sustained release trilaminar in

Control of parasitic bronchitis and gastroenteritis by

the control of parasitic gastroenteritis in first season grazing strategic prophylaxis with ivermectin. Veterinary Record, cattle. Veterinary Record, 124, 453–6.

121, 5–8.

Hart, R.J. (1986) Mode of action of agents used against

Barth, D. (1987) Treatment of inhibited Dictyocaulus vivipa-arthropod parasites. In Chemotherapy of Parasitic Diseases rus in cattle with ivermectin. Veterinary Parasitology, 25, (ed. by W.C. Campbell & R.S. Rew), pp. 585–602. Plenum

61–6.

Press, New York.

Bauer, C., Holtemyer, H. & Schid, K. (1997) Field evaluation Hashemi-Fesharki, R. (1991) Chemotherapeutic value of

of a fenbendazole slow release bolus in the control of

parvaquone and buparvaquone against Theileria annulata

nematode infections in first-season cattle. Veterinary

of cattle. Research in Veterinary Science, 50, 204–207.

Record, 140, 395–9.

Jacobs, D.E., Fox, M.T. & Ryan, W.G. (1987) Early season par-Bennett, D.G. (1986) Clinical pharmacology of ivermectin.

asitic gastroenteritis in calves and its prevention with iver-

Journal of the American Veterinary Medical Association, mectin. Veterinary Record, 120, 29–31.

189, 100–104.

Jacobs, D.E., Thomas, J.G., Foster, J., Fox, M.T. & Oakley, G.A.

Bishop, Y. (ed.) (1998) *Drugs used in the treatment and*

(1987) *Oxfendazole pulse release intraruminal devices*

control of parasitic infections. In The Veterinary Formulary, and bovine parasitic bronchitis: comparison of two control

4th edn, pp. 185–248. Pharmaceutical Press, London.

strategies in a field experiment. Veterinary Record, 121, Boersma, J.H. (1985) Chemotherapy of gastrointestinal nema-221–4.

todiasis in ruminants. In Chemotherapy of Gastrointestinal Jones, R.M. (1983) Therapeutic and prophylactic efficacy of

Helminths (ed. by H. vanden Bossche, D. Thienpont & morantel when administered directly into the rumen of

P.G. Jannsens), Handbook of Experimental Pharmacology, cattle on a continuous basis. Veterinary Parasitology, 12, vol. 77, pp. 407–42. Springer-Verlag, Berlin.

223–32.

Boray, J.C. (1986) Trematode infections of domestic animals. In Kassai, T. (1999) Anthelmintic therapy and control. In Veteri-Chemotherapy of Parasitic Diseases (ed. by W.C. Campbell nary Helminthology, pp. 141–61. Butterworth Heinemann,

& R.S. Rew), pp. 401–25. Plenum Press, New York.

Oxford.

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Martin, R.J. (1997) Modes of action of anthelmintic drugs. The phosphoglyceromutase. Molecular and Biochemical Para-Veterinary Journal, 154, 11–34.

sitology, **5**, 321–32.

Martin, R.J., Robertson, A.P. & Bjorn, H. (1997) Target sites Seibert, B.P., Guerrero, J., Newcomb, K.M., Ruth, D.T. &

of anthelmintics. *Parasitology*, **114**, S111–S124.

Swites, B.J. (1986) Seasonal comparisons of anthelmintic

Peregrine, A.S. (1994) Chemotherapy and delivery systems:

activity of levamisole pour-on in cattle in the USA. *Veteri-haemoparasites. Veterinary Parasitology*, **54**, 223–48.

nary Record, **118**, 40–2.

Prichard, R.K. (1985) Interaction of host physiology and

Sharma, N.N. & Mishra, A.K. (1990) Treatment of bovine

efficacy of antiparasitic drugs. *Veterinary Parasitology*, **18**, tropical theileriosis with buparvaquone. *Tropical Animal*

103–10.

Health Production, **22**, 63–5.

Prichard, R.K. (1986) Anthelmintics for cattle. *Veterinary Taylor, M.A. (1996) Bovine anthelmintics: an overview of*

Clinics of North America: Food Animal Practices, **2**, 489–501.

current thinking. *Veterinary Practice*, **28**, 7–8.

Prichard, R.K., Hennessy, D.R. & Steel, J.W. (1978) Prolonged Taylor, M.A. (1997) Treatment and control of ectoparasites in

administration: a new concept for increasing the spectrum

cattle. *Cattle Practice*, **5**, 279–82.

and effectiveness of anthelmintics. *Veterinary Parasitology*, Taylor, M.A. (1999). *Anthelmintics for cattle: a review. Cattle* **4**, 309–15.

Practice, **7**, 157–67.

Prichard, R.K., Hennessy, D.R., Steel, J.W. & Lacey, E. (1985) Taylor, M.A. & Catchpole, J. (1994) Coccidiosis of domestic Metabolic concentrations in plasma following treatment of

ruminants. *Applied Parasitology*, **35**, 83–6.

cattle with five metabolites. *Research in Veterinary Science*, Taylor, M.A. & Webster, K.A. (1998) Recent advances in

39, 173–8.

the diagnosis of *Cryptosporidium*, *Toxoplasma*, *Giardia* and Rew, R.S. & Fetterer, R.H. (1986) Mode of action of anti-other protozoa of veterinary importance. *Research in*

nematocidal drugs. In *Chemotherapy of Parasitic Diseases*

Veterinary Science, **65**, 183–93.

(ed. by W.C. Campbell & R.S. Rew), pp. 321–37. Plenum

Taylor, S.M., Malbo, T.R. & Kenny, J. (1985) *Comparison of Press*, New York.

early season suppressive anthelmintic prophylactic methods

Rolfe, P.F. & Boray, J.C. (1987) *Chemotherapy of param-*

for parasitic gastroenteritis and bronchitis in calves. *Veteri-phistomosis in cattle. Australian Veterinary Journal*, **63**, 328–

nary Record, **117**, 521–34.

32.

Wall, R. & Shearer, D. (1997) *The control and treatment*

Ryan, W.G., Crawfoes, R.J., Gros, S.J. & Wallace, D.H. (1997) of ectoparasite infestation, pp. 331–9. Chapman & Hall,

Assessment of parasite control and weight gain after use of
London.

an ivermectin sustained-release bolus in calves. Journal of Yazwinski, T.A.,
Featherstone, H., Presson, B.L., Greenway,

the American Veterinary Medical Association, 21, 754–6.

T.E., Pote, L.M. & Holtzen, H. (1985) Efficacy of injectable Schulman, M.D. &
Valentino, D. (1982) Purification, charac-clorsulon in the treatment of immature
bovine Fasciola

terisation and inhibition by MK-401 of Fasciola hepatica
hepatica infections. Agripractice, 6, 6–8.

Chapter 61

Antimicrobial Agents

A.H. Andrews

Introduction

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on other organisms. These chemical compounds are

Identification of the causal agent

1035

the metabolic products of bacteria and fungi. Most

Sampling

1035

commonly, antibiotics are effective against bacteria

Sensitivity testing

1036

but some have activity against viruses, Rickettsia,

Penetration

1036

Mycoplasma, fungi and helminths (see Table 61.1).

Antibiotic resistance

1038

At present all prescribing of antimicrobial agents for

Dosage

1038

therapy, prophylaxis or control of disease is under vet-

Route of administration

1038

Duration of therapy

1038

erinary control and prescription in the UK. The level of

Spectrum of drug activity

1040

control in some countries is greater than in Britain, e.g.

Bacteriostatic or bactericidal activity

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Sweden, although in many parts of the world it is less

Drug combinations

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or almost non-existent.

Cost

1042

Toxicity

1042

Drug withdrawal times

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Identification of the causal agent

Legal considerations

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Frequency of dosing

1042

Some decisions regarding therapy can only be made

Possible antibacterial therapy for cattle

1044

once a diagnosis is reached. Clinical examination is

used to identify the nature of the infection, its site and

Introduction

possible cause. The signs of some diseases are so clear-

cut that diagnosis is easy to establish and the organism

The choice of a suitable antimicrobial agent is very dif-

is readily identifiable, e.g. wooden tongue, actinomyco-

difficult. It first depends on deciding whether the problem

sis. In such cases the choice of drug is easy to make as

encountered is an infection and even then whether it

well as its dosage, rate and duration of treatment. The

warrants such therapy. The site of infection needs to be

likely outcome of therapy can also be predicted with

located and the type of microbe established. Then the

some confidence. However, in many cases the disease

in vitro antimicrobial sensitivity of the organism and the signs are not clear-cut, the area of infection is difficult

minimal inhibitory concentration need to be deter-

to localize or the sensitivity pattern of the organism is

mined. The likely ability to penetrate the area, the

irregular. In such cases the identification of the causal

action of the antibiotic in the local environment and the

agent is important not only for treatment of the maintenance of an effective concentration at the area individual animal affected but also for possible future must be considered. The selection also has to include action on a herd basis. However, in many situations, the the possibility of any potential toxic side-effects as well site of infection and the cause of the condition are not as the meat and milk withholding times. Once all these known. In such instances laboratory diagnosis, other factors have been taken into consideration, the drug to than haematological examination to indicate that an be used, its dosage, route and frequency of administra- infection is present, is of no value. Therapy in these tion can be determined. All the above, however, has to cases usually involves the use of one or more anti- be undertaken on an economic basis.

microbial agents to ensure a broad spectrum of activity.

There are various ways of defining antimicrobial

Subsequent progress will depend on evaluation of the preparations. Chemotherapy can be defined as the use effect of the chosen treatment.

in the treatment of a disease of pure chemicals that have a special antagonistic effect(s) on the organism causing the disease.

Sampling

Antibiotics are complex organic chemicals synthesized by micro-organisms during their growth and
Wherever possible sampling should be undertaken to which, in minute quantities, have a detrimental effect
try to determine the causal organism. This will be

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Table 61.1

Non-antibacterial activity of some antibiotics.

Table 61.2

Drugs with reasonable penetration of various fluids and tissues.

Mycoplasma

Chlortetracycline, doxycycline,
erythromycin, fluoroquinolones,

Bile

Erythromycin

lincomycin, methacycline,

Bone

Chlortetracycline, doxycycline, fucidin,

oleandomycin, oxytetracycline,

lincomycin, methacycline,

spiramycin, tetracycline, tylosin

oxytetracycline, tetracycline

Rickettsia

Chloramphenicol, chlortetracycline,

Cerebrospinal

Chloramphenicol, florfenicol,

doxycycline, fluoroquinolones,

fluid

fluoroquinolones, sulphonamides,

methacycline, oxytetracycline,

trimethoprim and sulphonamides

tetracycline

Eye

Amoxycillin, ampicillin, cephalexin,

Protozoa

Monensin, sulphonamides, tetracycline

cephaloridine, cephalothin,

Fungi

chloramphenicol, lincomycin

Candida albicans Amphotericin, nystatin

Milk

Benzylpenicillin, erythromycin, lincomycin

Dermatophytes

Griseofulvin, natamycin

(but toxic), oleandomycin, penethamate,

Ectoparasites and

Ivermectin

spiramycin, tylosin

endoparasites

Prostate

Erythromycin, trimethoprim

Serosal fluids

Chloramphenicol, dihydrostreptomycin,

erythromycin, framycetin, gentamicin,

kanamycin, lincomycin, neomycin,

paromomycin, streptomycin

considered academic by many, but if it is looked at

objectively, it has much to commend it. At the first

Sensitivity testing

examination and, hopefully, before any therapy has

been initiated is the best time to obtain samples to

Identification of the causal organism allows a decision

establish the likely cause of the problem. In addition,

to be made as to whether or not to test for anti-

the cost of micro-organism determination is usually

microbial sensitivity. In many cases there is no need to

less, or at least no more, than the cost of appropriate

undertake this. Thus organisms of *Clostridium* spp. are therapy. In almost all cases the value of the individual

sensitive to high doses of penicillin G. However, in cases

animal will be sufficient to make it worthwhile and this

involving *Escherichia coli* and *Salmonella typhimurium* is multiplied many times when problems are encoun-there will be considerable variations in the sensitivity

tered on a herd basis. There is virtually no justification

pattern, bearing in mind the origin of the organism.

for not sampling problems of a herd nature when infec-

The Kirby–Bauer disc sensitivity test is that most

tions are suspected. In addition, the treatment of

commonly used for in vitro testing. The aim is to deter-chronic conditions or

those that have responded poorly
mine if an organism is likely to be affected by specific
is important. Besides positively determining the cause,
antimicrobial agents at the usual therapeutic levels.
sampling may help to eliminate other potential agents.
While the drug concentration reflects levels achieved
The method of sampling is important in that it should
in the plasma, it does not take into account quantities
involve fresh clinical cases rather than those of a
in various tissues such as the brain or udder. The test
chronic nature. The sample should be taken from as
organism is usually classified as sensitive, intermediate
near the inflamed area as possible. This is difficult with
or resistant to the specific antimicrobial agent. The test
problems such as salmonellosis, as organisms in the
was produced for use in human medicine where it pro-
faeces may be diluted by those further down the gut,
vides a quantitative assessment. However, in animals
which in themselves may be much faster growing. Sam-
it is a useful guide. Quantitative tests involve determin-
pling of pneumonias is always difficult as the organisms

ing the minimal inhibitory concentration and usually present in the upper respiratory tract may be unrepresentative of those in the lung. In general, an organism consist of tube sensitivity tests.

sensitive in vitro may be sensitive in vivo, but one resistant-Direct examination of the sample after staining is

ant in vitro is also likely to be resistant in vivo.

of use as in some circumstances organisms will not subsequently grow. Rapid staining methods are now available and these allow the shape of the organism

Penetration

and whether it is Gram-positive or Gram-negative to be determined. This in turn can be useful in making a The ability of a drug to penetrate the infected area needs quick decision about the drug most likely to be to be considered (see Table 61.2). Penetration depends effective.

on the degree of the pathological process as is seen with Antimicrobial Agents • 1037

abscess or thrombus formation. It also depends on the high turnover rate, usually in the lysis of cells and

tissue involved and whether there are natural limits to release of the organisms into fluids containing the penetration such as in the brain (see p. 897) or in milk. antimicrobial agent. Lipid solubility does have advantages for organisms that spend most of their time within whether it is intracellular or extracellular. cells or behind tissue barriers. Inflammation can also The ability of an antimicrobial drug to cross biological membranes depends on many factors. These include central nervous system by many drugs is minimal in the protein binding, lipid solubility, pH of the drug in relation to environmental pH, molecular size and in some healthy animal although penicillin G and other antibiotics can enter during inflammation. However, the cases specific cellular transport mechanisms. Antibiotic effect of inflammation on the penetration of many is absorbed into the circulation, partly becoming bound drugs is unknown. Thus it can be the local environment to plasma protein. Binding varies, e.g. from less than 50

in which the organism is present that is important.

per cent with amoxycillin, streptomycin or oxytetracycline to more than 80 per cent with cloxacillin or erythromycin. Binding is readily reversible and the bound conditions with a slightly alkaline pH. As such conditions do not exist around staphylococcal abscesses this form. Protein binding can be a disadvantage, as with group of drugs is relatively inactive in such circumstances. Activity of antibacterial agent in urine is often purulent fluids often contain much protein, sulphamides can be relatively inactive.

Water-soluble drugs are useful for extracellular

Some antibiotics do have more effect on anaerobic bacteria than others under such conditions (see Table 61.4).

Table 61.3

Antibacterial drug activity in urine. Urinary infections are relatively uncommon in cattle but can lead to therapeutic problems.

Not affected by urine pH

Most active in alkaline urine

Most active in acid urine

Cephalexin

Colistin

Carbenicillin

Cephaloridine

Dihydrostreptomycin

Chlortetracycline

Cephalothin

Erythromycin

Doxycycline

Chloramphenicol

Framycetin

Methacycline

Nalidixic acid

Gentamicin

Nitrofurantoin

Sulphonamides

Kanamycin

Oxytetracycline

Trimethoprim and sulphonamides

Neomycin

Penicillin G

Paromomycin

Tetracycline

Polymyxin B

Streptomycin

Acidification

D-Methionine (oral)

Alkalinization

Ascorbic acid (oral)

Sodium bicarbonate (oral)

Table 61.4

Antibiotics effective in vitro against anaerobic bacteria.

High efficacy

Moderate efficacy

Low efficacy

Amoxycillin

Chlortetracycline

Dihydrostreptomycin

Ampicillin

Erythromycin

Framycetin

Carbenicillin

Oxytetracycline

Gentamicin

Chloramphenicol

Procaine penicillin G

Kanamycin

Clindamycin

Tetracycline

Neomycin

Metronidazole

Paromomycin

Streptomycin

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Antibiotic resistance

Table 61.5

Penicillinase (b-lactamase) active antimicrobials.

Penicillins

Cloxacillin, methicillin

The advent of antimicrobial drugs has resulted in the

Cephalosporins

Cefoperazone, cephalexin,

emergence of resistance to these drugs (see Table 61.5).

cephalonium, cephaloridine,

The use of antibiotics in therapeutic concentration

cephalothin

results in an alteration of the microflora within the

Aminoglycosides

Amikacin, framycetin, gentamicin,

host. There is a tendency for a loss of the sensitive

kanamycin, neomycin, paramomycin

strains with the resistant ones remaining. This selection

Macrolides

Erythromycin, oleandomycin,

allows multiplication of resistant strains within the

spiramycin, tylosin

less competitive environment. The resistant organisms

Nitrofurans

Furazolidone, nitrofurantoin,

can remain in the animal and also contaminate

nitrofurazone

the environment, thereby allowing passage to other

Fluoroquinolones

Danofloxacin, enrofloxacin,

animals. The prevalence of the resistant organisms

marbofloxacin

Others

Bacitracin, chloramphenicol, florfenicol,

tends to reduce the longer the period since the drug was

lincomycin, novobiocin, sulphonamide

used.

and trimethoprim, vancomycin

Spontaneous mutation of organisms can occur but it

is probably only of limited importance in practice.

However, transferable drug resistance is of significance.

This resistance, as determined in the form of plasmids

or transposons, is transferred to susceptible strains.

levels that are effective against most susceptible organ-

Plasmids are extrachromosomal genetic material that

isms, with minimal side-effects. In most cases the recombinant plasmids can replicate independently of the chromosome. These plasmids can be transferred by conjugation within or between bacterial species and they can also act as vectors for transposons. This allows the transfer of single or multiple antibiotic resistance and has produced many of the resistant bacteria found in enteric calf infections including both *E. coli* and *S. typhimurium* as well as in *Staphylococcus aureus* and low toxicity. However, when the antimicrobial agent is of high toxicity any increase in dosage should be undertaken with caution. Different dosages may also

animals is restricted or banned because of the usage in influence the drug withdrawal time. It should be humans and also due to potential human toxicity from remembered that when different companies produce handling it. Increasing concern has been expressed the same antibiotic it may well contain different about microbial resistance to antibacterials in human amounts of the drug and the vehicle in which it is beings caused by contact directly with, or indirectly presented may vary.

from food produced from, farm animals. While the risks appear to be minimal in creating human microbial resistance compared with other causes, all veterinarians

Route of administration

should bear the problem in mind when treating or prescribing for cattle.

The choice of administration route depends on the Table 61.5 gives examples of antimicrobials which speed with which infection has to be counteracted as still have activity in the presence of penicillinase. well as the volume to be given, ease of administration,

etc. The principal routes used are given in Table 61.6.

Dosage

Duration of therapy

When an antimicrobial agent is selected it must be in a high enough concentration and used for a long enough

In general terms, most injections are used for three to period to result in contact with the organism and its

five days and for at least one day after normality is

consequent demise. Various factors influence this,

reached. However, there is a tendency not to treat for

including the dose, formulation and rate of administra-

long enough in chronic conditions such as some cases

tion. Doses are usually based on body weight. The rec-

of chronic joint or thoracic infections when therapy

ommended dose usually provides good blood and tissue

should be continued for at least 10–14 days. If it was a

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Table 61.6

Routes of administration of antimicrobial drugs.

Intravenous (iv): always give slowly

Disadvantages

Advantages

May provoke marked fluid reaction

Rapid high blood and tissue levels, therefore useful for

Only slowly absorbed

septicaemias

Speed of absorption less than im

Always higher circulating levels than im or oral

Intra-articular

Increased levels at areas where levels usually low, e.g.

Increased level in joint

necrotic areas, chronic abscesses

Must be sterile technique

Useful for large volumes

Useful for painful injections

Intrapleural

Useful for irritant injections

Increased level in thorax

Only method of obtaining accurate blood and tissue

Must be non-irritant

levels

Must be a sterile technique

Disadvantages

Subconjunctival

Acute toxic reactions more common

Useful for ocular conditions

Specific formulations necessary

Must be non-irritant

Requires good technique

Must be sterile technique

Perivascular and intravascular thrombosis can occur

Oral (water or feed)

Intramuscular (im)

Advantages

Advantages

Easiest method of administration

Most common route

Can be undertaken by owner

Only 20 ml per injection site

Disadvantages

Peak blood and tissue levels within 2 hours

Blood and tissue levels less than for comparable parenteral dose Slightly less irritant injections than those given iv can be

Higher doses usually used

given by deep im injection

Absorption is variable (depends on gut motility, type of feed, Allows a reasonable level to be maintained over a period

volume of feed, drug binding, e.g. calcium, kaolin impair

of time

absorption of tetracycline, oxytetracycline, chlortetracycline, Disadvantages methacycline, doxycycline, ampicillin, lincomycin)

Muscle damage

Blood levels do not peak for 12–18 hours

Try not to do within 3 weeks of slaughter

Antibiotics may influence normal ruminal flora

Severe reactions to some drugs

Some antibiotics not absorbed, e.g. streptomycin,

Some injections painful

dihydrostreptomycin, neomycin, framycetin,

Accurate blood and tissue levels not obtained

phthalylsulphathiazole

Some antibiotics destroyed, e.g. benzylpenicillin

Intraperitoneal (ip)

Topical

Advantages

Ointments, powders, aerosol sprays

Speed of absorption similar to im injection

Usually well absorbed

Ophthalmic preparations

Useful for peritonitis

Ointments and drops. Limited retention at site except some

Useful if very toxic

special preparations

Useful for irritant drugs

Aural preparations

Useful in severe respiratory distress (especially

Drops and ointments

tetracyclines)

Large volumes can be given

Intramammary (use aseptic technique)

Advantages

Disadvantages

Convenient

Possible sepsis

Good efficacy

Accurate blood and tissue levels not obtained

Disadvantages

Subcutaneous (sc)

Often diffusion reduced by blocked lactiferous ducts, etc.

Advantages

Used for clinical treatment

Little used for antimicrobials except sulphonamides

Dry-cow therapy used for subclinical mastitis and prophylaxis

Large volumes can be given

If intramammary tube used in one quarter all milk should be

Less irritant than im

withheld

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*human or small animal there would often be no dispute
activity against Mycoplasma, Rickettsia and Chlamydia.*

about such a length of therapy. Many antibacterial

*The antibacterial activity of drugs can also vary (see
preparations are now available that can be injected at*

*Table 61.7): some have a narrow spectrum, only inhibit-
extended intervals to provide adequate drug concen-*

ing either Gram-negative or Gram-positive organisms;

trations for several days. However, in some cases the others have most activity against Gram-positive duration of effective therapy can be overestimated. organisms but will inhibit some Gram-negative agents. However, some have a broad spectrum inhibiting both Gram-positive and Gram-negative bacteria.

Spectrum of drug activity

Antibacterial drugs vary in their degree of activity

Bacteriostatic or bactericidal activity

against various classes of organism. They are usually considered narrow spectrum if they are only active At times it is important to know the activity of a against bacteria, but broad spectrum if they also have drug. Some drugs are bactericidal, e.g. penicillin,

Table 61.7

Antibacterial activity other than for anaerobic bacteria.

Gram-positive

Gram-negative

Broad spectrum: Gram-positive

bacteria

bacteria

and Gram-negative bacteria

Penicillins

Penicillins

Penicillins

Cloxacillin

Carbenicillin

Amoxycillin

Methicillin

Ampicillin

Penicillin G

Polymyxins

Clavulanic acid and amoxycillin

Penicillin V

Colistin

Polymyxin B

Sulphonamides

Macrolides

Many forms

Erythromycin

Aminoglycosides

Oleandomycin

Amikacin

Tetracyclines

Spiramycin

Dihydrostreptomycin

Chlortetracycline

Tylosin

Framycetin

Doxycycline

Gentamicin

Methacycline

Others

Kanamycin

Oxytetracycline

Bacitracin

Neomycin

Tetracycline

Lincomycin

Paromomycin

Novobiocin

Streptomycin

Cephalosporins

Tiamulin

Cefquinone

Vancomycin

Others

Ceftiofur

Apramycin

Cephalexin

Spectinomycin

Cephaloridine

Cephalothin

Nitrofurans

Furazolidone

Nitrofurantoin

Nitrofurazone

Fluoroquinolones

Danofloxacin

Enrofloxacin

Marbofloxacin

Others

Chloramphenicol

Florfenicol

Halquinol

Sulphonamide and trimethoprim

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Table 61.8

Bactericidal and bacteriostatic drugs.

Bactericidal

Bacteriostatic

Penicillins

Halquinol

Amoxycillin

Novobiocin (high concentration)

Ampicillin

Spectinomycin (occasionally)

Carbenicillin

Sulphonamide and trimethoprim

Cloxacillin

Vancomycin

Methicillin

Penicillin G (benzylpenicillin)

Macrolides

Penicillin V (phenoxypenicillin)

Carbomycin

Erythromycin (low concentration)

Macrolides

Oleandomycin

Erythromycin (high concentration)

Spiramycin

Tilmicosin

Nitrofurans

Tylosin

Furazolidone (high concentration)

Nitrofurantoin (high concentration)

Sulphonamides

Nitrofurazone (high concentration)

All types

Cephalosporins

Nitrofurans

Ceftiofur

Furazolidone (low concentration)

Cephalexin

Nitrofurantoin (low concentration)

Cephaloridine

Nitrofurazone (low concentration)

Cephalothin

Tetracyclines

Polymyxins

Chlortetracycline

Colistin

Doxycycline

Polymyxin B

Methacycline

Oxytetracycline

Aminoglycosides

Tetracycline, etc.

Amikacin

Dihydrostreptomycin

Others

Framycetin

Apramycin

Gentamicin

Chloramphenicol

Kanamycin

Clindamycin

Neomycin

Florfenicol

Paromomycin

Lincomycin

Streptomycin, etc.

Novobiocin (low concentration)

Fluoroquinolones

Spectinomycin (usually)

Danofloxacin

Tiamulin

Enrofloxacin

Trimethoprim

Marbofloxacin

Others

Bacitracin

Panfloxacin

Whether a drug is bactericidal or bacteriostatic needs to be known as problems can occur with combinations.

aminoglycosides, while others are bacteriostatic and

Drug combinations

require the animal's own defence mechanism to help in

the organisms' removal (Table 61.8). These definitions
These are frequently used in veterinary practice but are
are only relative and some agents are bactericidal in
often best avoided. They are of used where infection
high concentrations but only bacteriostatic at low levels.
is due to mixed organisms. They can be helpful where

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synergism has been shown with the combination, e.g.
biotic testing. Where they have occurred they involved
penicillin and aminoglycosides, or trimethoprim and
usage of milk. Another possible hazard is drug resist-
sulphonamides. Although the bacteriostatic or bacteri-
ance in humans but adequate documented evidence for
cidal nature of many drugs is dependent on the
the occurrence of this following consumption of milk or
concentration used, it is still a good ploy to combine
meat is still required. The main effect of antibiotics in
antibiotics with a similar activity. This is of particu-
milk is a commercial one in that it can affect the starter
lar importance when treating immunocompromised
cultures for cheese or yoghurt.

animals. Besides the broader spectrum of activity
Various factors influence the withdrawal time and
attained with some combinations, cost may be less and
these include the drug dose rate, age, disease, duration
in some cases there may be a reduction in toxicity.
and role of therapy. The injection site will obviously
When using combinations it is best to give each
contain higher levels than other sites. Depending on
drug individually at the correct dose with an interval
how the drug is metabolized, the kidney and liver
between administrations. When two drugs are com-
may have higher levels than other tissues. In all
bined in commercial preparations then the dosage of
cases it is the duty of the veterinary surgeon to ensure
each is fixed regardless of the needs of the particular
that any withdrawal time is observed and that the
situation or the interval between doses. Care should be
owner of the animal is made aware of this. It is always
taken in the choice of antimicrobial agent when used in
best to write the time down either on the drug vial if
combination with corticosteroids.

doses are to be provided for administration by the farmer, or on an invoice or note to the farmer. Where a withdrawal time is not known then the manufacturer

Cost

or distributor of the drug should be contacted.

However, in general, when no withholding time is

This is a major drawback in that often the best therapy

stated then milk must be discarded for seven days

cannot be instituted because of the expense. Repeat

and cattle may not be slaughtered for human con-

doses also cause problems, especially as oral therapy is

sumption for 28 days. In the USA veterinarians can

not often advisable in ruminants. The problem can be

obtain information on withdrawal times and residues by

overcome by long-acting preparations or providing

contacting the Food Animal Residue Avoidance Data

combination injections for the farmer or stockworker

book (FARAD).

to administer. It must be remembered there is an indi-

rect cost, particularly to the dairy farmer, in the length

of time the milk has to be withheld after the end

Legal considerations

*of treatment. The beef farmer encounters a similar
The availability of antimicrobial agents in most coun-
problem regarding meat withdrawal time but to a lesser
tries is first dependent on their being licensed for use
extent.*

*by a regulatory authority. This can be the Veterinary
Medicines Directorate, Food and Drug Administration*

Toxicity

*or another similar body. Some antibiotics are available
in certain countries but are banned in others, e.g. chlo-
ramphenicol, which is prohibited from use in North
Except where there is routine usage of antimicrobial
America and most of Europe.*

*drugs in adult animals, few problems of toxicity
arise. Some of the more common are give in Table
61.9.*

Frequency of dosing

Drug withdrawal times

*The frequency of administering therapeutic prepara-
tions varies. Some compounds are placed in slow-*

Following use of an antimicrobial agent there is now release boluses to facilitate this. The rate of absorption usually a period between finishing therapy and the use also varies between different preparations, dependent of the milk or the slaughter of the animal for meat. Milk on the particle size; thus those with micronized particles are rapidly absorbed. The form of the antibiotic health risk. There is the possibility of allergy to penicillins will also influence the level of activity. Thus with cillins or other antibacterials following medical pre-penicillin, benzylpenicillin is rapidly absorbed (in minutes) to reach peak levels but only persists for almost non-existent in countries with adequate antibiotic about 6 hours. However, procaine benzylpenicillin

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Table 61.9

Toxicity of antimicrobial drugs.

Penicillins

Very low toxicity, hypersensitivity. Occasional severe adverse reactions

Sulphonamides

Toxicity rare, rapid intravenous injection causes collapse and respiratory distress, crystalluria

Pain and reaction following sc or im injections. Crystalluria not a problem in large animals. Erythromycin and tylosin irritant following injection

Lincomycin

Diarrhoea, reduced milk production (oral)

Aminoglycosides

Ototoxicity (unlikely), cardiovascular damage, hypotension (for toxic and shocked animals), renal toxicity

Tetracyclines
Acute collapse syndrome (iv). Alimentary tract dysfunction (if oral). Deposition in bone and teeth

Chloramphenicol
Little toxicity. Superinfection (oral). Anaemia in humans

Polymyxyn B Colistin

Usually only used topically as toxic shock and death can occur following parenteral use

Nitrofurans
Acute – nervous signs

Chronic – haemorrhagic syndrome

Table 61.10

Some possible antibacterial therapy for infections in cattle.

Calves

Diarrhoea (acute) bacterial

Amoxycillin, ampicillin, (chloramphenicol), chlortetracycline, clavulanic acid and amoxycillin, danofloxacin, enrofloxacin, framycetin, marbofloxacin,

neomycin, oxytetracycline, trimethoprim and sulphonamide

Diarrhoea (subacute) bacterial

Neomycin, nitrofurazone, oxytetracycline, sulphonamides

Joint ill

Lincomycin, oxytetracycline

Meningitis

(Chloramphenicol), danofloxacin, enrofloxacin, marbofloxacin, trimethoprim and sulphonamide

Navel ill

Amoxycillin, ampicillin, clavulanic acid and amoxycillin, oxytetracycline, trimethoprim and sulphonamide

Pneumonia

Amoxycillin, ampicillin, cefquinone, ceftiofur, cephalixin, clavulanic acid and amoxycillin, danofloxacin, enrofloxacin, florfenicol, marbofloxacin, oxytetracycline, penicillin and streptomycin, tilmicosin, tylosin

Ringworm

Griseofulvin, natamycin

Salmonella spp.

Amoxycillin, ampicillin, apramycin, (chloramphenicol), clavulanic acid and amoxycillin, danofloxacin, enrofloxacin, marbofloxacin, neomycin, nitrofurazone, trimethoprim and sulphonamide

Septicaemia

Amoxycillin, ampicillin, (chloramphenicol), clavulanic acid and amoxycillin, danofloxacin, enrofloxacin, marbofloxacin, trimethoprim and sulphonamide

Older cattle – injections

Actinomycosis

Amoxycillin, cephalixin, oxytetracycline, penicillin, sulphonamides Anthrax

Amoxycillin, oxytetracycline, penicillin and streptomycin

Blackleg

Benzylpenicillin

Clostridial infections

Oxytetracycline, penicillin

Diarrhoea (acute)

Sulphonamide

Infectious bovine keratoconjunctivitis

(Chloramphenicol), oxytetracycline

Leptospirosis

Amoxycillin, oxytetracycline, streptomycin

Mastitis (peracute) (parenteral)

Amoxycillin, ampicillin, cefquinone, cephalixin, oxytetracycline, trimethoprim and sulphonamide

Metritis

Amoxycillin, ceftiofur, cephalixin, clavulanic acid and amoxycillin, oxytetracycline Peritonitis

Sulphonamides, trimethoprim and sulphonamides

Pneumonia (acute)

Amoxycillin, ceftiofur, danofloxacin, enrofloxacin, marbofloxacin, oxytetracycline, trimethoprim and sulphonamide

Pneumonia (subacute)

Amoxycillin, ampicillin, ceftiofur, oxytetracycline, penicillin and streptomycin, trimethoprim and sulphonamide

Pyelonephritis

Procaine penicillin

Salmonella spp.

Amoxycillin, ampicillin, (chloramphenicol), danofloxacin, enrofloxacin, marbofloxacin, neomycin, trimethoprim and sulphonamide

Tetanus

Benzyl and procaine penicillin

Wooden tongue

Amoxycillin, oxytetracycline, penicillin and streptomycin, sulphonamide Note:
Not all these drugs are registered for the species or for the use suggested.

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Box 61.1

Advantages and disadvantages of slow-release

*reaches a maximum level in 2–4 hours and persists for
antibiotic preparations.*

*24 hours, benethamine benzylpenicillin has a slow
absorption (6–24 hours) and may be effective for three*

Advantages

to four days, and benzathine benzylpenicillin is slowly

Often used in prophylaxis

Post-operatively

absorbed over 6–24 hours and is often effective for four

Post calving where there has been intervention

to seven days.

Dry cow therapy: most remain about 3–4 weeks in udder but

The advantages and disadvantages of slow-release

some persist six weeks or longer

antibiotic preparations are given in Box 61.1.

Less stress to animal

Reduced handling

Less pain to animal

Possible antibacterial therapy

Convenience

for cattle

Disadvantages

Animals may not be examined as regularly as when injected

A survey of some of the drugs used in the treatment of

often

cattle is given in Table 61.10. It is emphasized that this

Thus animal less monitored

list is not complete and some preparations are not per-

*Usually tissue and blood levels lower than routine injections
mitted in all countries.*

*Increased withholding time for milk or injection-to-slaughter
time*

Chapter 62

Inflammation and Pain

J.L. Fitzpatrick, A.M. Nolan, P. Lees and S.A. May

The nature of inflammation

1045

tially, local blood flow increases. Vascular endothelial

Mediators and modulators of inflammation

1046

cells change their shape, with the formation of inter-

Classification of anti-inflammatory drugs

1048

endothelial cell gaps through which plasma leaks. This

Non-steroidal anti-inflammatory drugs (NSAIDs)

1050

leads to the formation of protein-containing exudate

Classification and mechanism of action

1050

(oedema fluid) in the interstitial compartment. At this

Actions and interactions of NSAIDs and general uses

1051

stage blood flow slows and eventually becomes static.

Pharmacokinetics of NSAIDs

1052

The contents of the microcirculation include plasma

Side-effects and toxicity of NSAIDs

1055

Residues

1056

and the cellular elements of blood. The phagocytic leu-

Steroids

1056

cocytes (neutrophils and monocytes) emigrate from the

Classification and general properties

1056

circulation during the inflammatory process and engulf

Mechanism of action

1057

*the tissue irritant, including micro-organisms, in the
Administration and pharmacokinetics of steroids*

1058

inflammatory exudate. The initial step is margination

Side-effects of steroids

1059

and adherence of leucocytes to the vascular endothelial

General uses of steroids

1059

wall, followed by pavementing, the process whereby the

Therapy with anti-inflammatory drugs

1060

cells roll along the wall to the inter-endothelial cell gaps.

Respiratory diseases

1060

Passage through the wall and directional movement to

Mastitis and endotoxaemia

1061

the site of injury occurs by the process of diapedesis.

Metritis

1063

Neutrophils are the first cell type to enter the lesion, fol-

Calf scours

1063

Hypersensitivity reactions

1063

lowed after several hours by mononuclear phagocytes.

Arthritis and joint diseases

1063

Other circulating cells (platelets, eosinophils and

Foot-and-mouth disease

1063

basophils) may also migrate to the extravascular space.

Pain

1063

Inflammation, if associated with infection, may be

Conclusions

1064

accompanied by fever, as the result of resetting of the

‘thermostat’ in the hypothalamus. These changes at the

level of the tissue or organ comprise Celsius’ four clas-

The nature of inflammation

sical signs of inflammation, rubor et calore cum tumor

et dolor (redness and heat with swelling and pain) . To Inflammation (from the Latin inflammare, to burn) is

these Virchow added a fifth sign, functio laesa (loss of common to many diseases involving injury to living

function), many centuries later. The essential features

tissues. Inflammation is, in fact, the basis of most pathol-

of the acute inflammatory process are outlined in Fig.

ogy. Acute or chronic inflammation occurs as a conse-

62.1.

quence of chemical, physical or microbiological damage

The suffix ‘-itis’ is appended to the name of the

to tissues. Acute inflammation has been described

affected tissue to describe organ-based inflammation in

as the response of the living microcirculation and its

the conditions of mastitis, bursitis, colitis, cystitis, metri-contents to injury. A range of defence mechanisms

tis, arthritis, tendonitis, laminitis and many others.

including the immune system may be utilized. If the

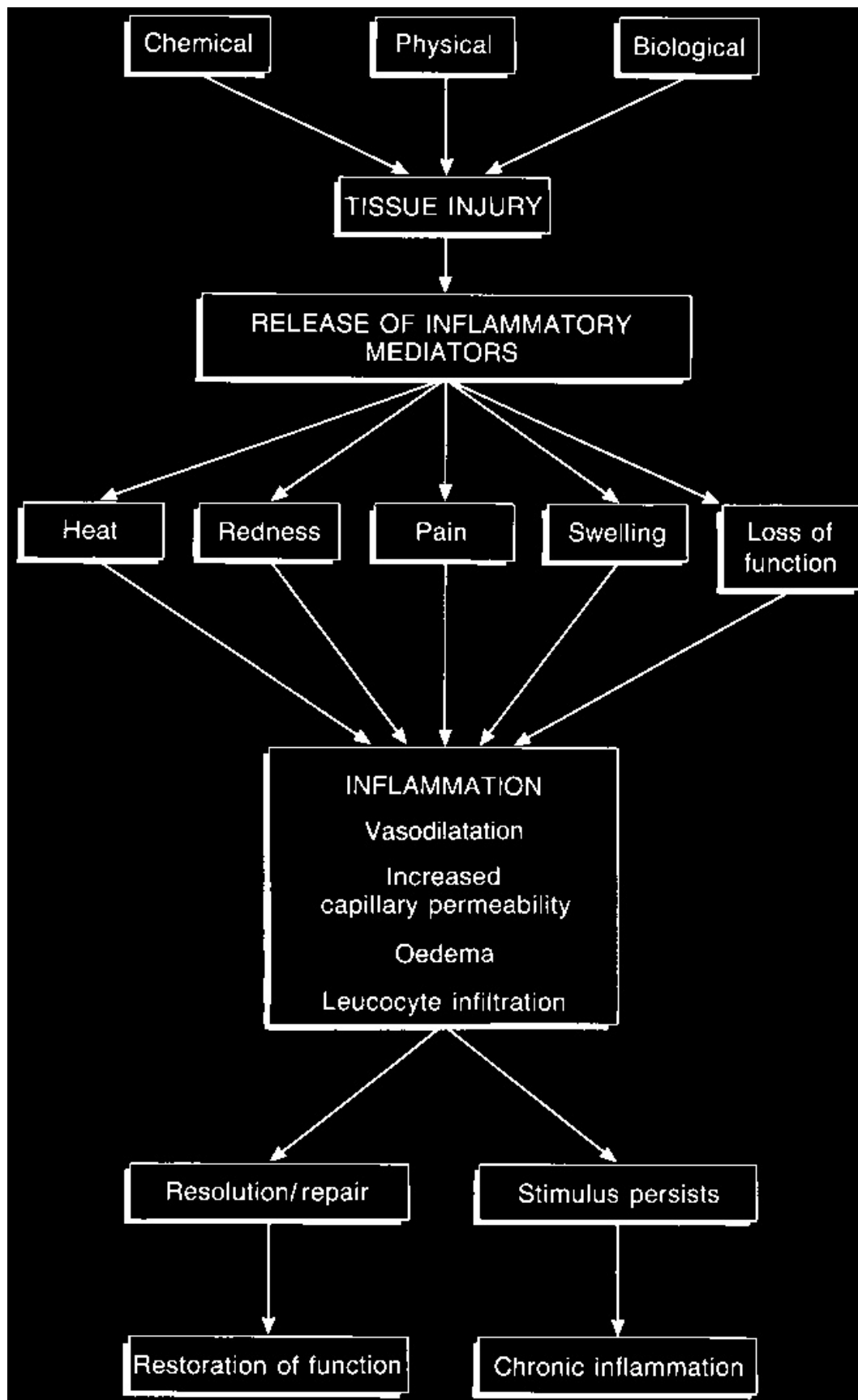
Severe local inflammation or more widespread inflam-

inflammation is caused by micro-organisms, tissue

mation, particularly associated with infection, may be damage may proceed as far as host cell death. An avas- accompanied by fever.

cular tissue like cartilage, as with dead tissue, cannot The outcome of acute inflammation may be resolu- respond to injury by mounting an acute inflammatory tion and hence restoration of normal tissue architecture response, although in some circumstances it may and function. However, particularly if the stimulus per- degrade.

sists, tissue destruction may occur with fibrous tissue The microcirculation therefore plays a crucial role in formation and repair, potentially compromising normal tissue response to injury. Blood vessels dilate and, ini- function. In some cases, there may be delayed healing



lesions, including sole ulceration, white line disease and acute digital tissue infection, leading to lameness. The pain component of inflammation arises from activation and sensitization of fine unmyelinated and some myelinated sensory fibres to the actions of chemicals released from a variety of sources during inflammation (Ferreira, 1983). Information from the periphery is conducted to the spinal cord and from there to the brain, where pain is perceived. Inflammation induces alterations in nociceptive information processing which may have serious consequences for the animal. Allodynia (perception of innocuous stimuli as noxious) and hyperalgesia (exaggerated responses to noxious stimuli) are common features of inflammatory pain (Coderre & Melzack, 1987; Nolan, 2000).

Mediators and modulators of inflammation

The characteristic pathophysiological changes of acute and chronic inflammation are produced by the action

of chemicals, either released from cells such as mast cells, macrophages and fibroblasts or formed by de novo synthesis. If there is an immune basis to the inflammatory response, lymphocytes are also involved. Compounds like histamine, that act directly to produce microvascular changes, or like bradykinin, to cause pain are called inflammatory mediators or, if their role has not been firmly established, they are termed putative mediators.

The number of putative mediators identified is con-

Fig. 62.1

The inflammatory process.

siderable. Moreover, there are some species differences in mediators, so that, for example, 5-hydroxytryptamine (5-HT) is probably an acute inflammatory mediator in or even persistent chronic inflammation, with local pro-rodents only. Some mediators, such as complement lification of connective tissue and mononuclear cell fragments C5a and C3a, not only act directly on the accumulation. The granulation tissue in chronic inflammation but also cause mast cell degranulation,

mation comprises highly vascularized connective tissue.

leading to histamine release and hence reinforcement

In some forms of chronic inflammation lymphocytes are

of the inflammatory response. In addition, there is a

present, suggesting an immune response superimposed

sequential release of mediators, such that histamine, 5-

on the inflammatory response.

HT and bradykinin are involved in the early part of the

Pain associated with inflammatory disease is proba-

acute phase, whereas lysosomal and other proteolytic

bly the major source of pain in ruminant species. The

enzymes are involved subsequently. Much is now

measurement of pain in animals is fraught with diffi-

known about acute inflammatory mediators; informa-

culties and, to date, pain research in animals has focused

tion on the mediators responsible for the transition to,

primarily on mechanisms of acute pain (e.g. Lascelles et

and the subsequent persistence of, chronic inflamma-

al., 1997; Dolan & Nolan, 2000). Pain is a complex expe-tion is more sparse.

Examples of mediators inducing

rience, dependent not only on the severity of the insult

vascular leakage are bradykinin, histamine and platelet to pain pathways and the degree of tissue or nerve activating factor (PAF), which act directly, and damage, but also on previous pain experiences and on leukotriene B4 (LTB4) and complement fragment C5a, social position within a herd. The most significant which exert their effect through neutrophil-dependent inflammatory diseases likely to be associated with pain mechanisms (Williams, 1983). Mediators of cell migration in ruminants are mastitis and inflammatory limb tion and accumulation include C5a, PAF, LTB4,

Inflammation and Pain • 1047

interleukin-1 (IL-1) and many other chemokines and markedly both the pain produced by kinins such as cytokines, whilst mediators responsible for producing bradykinin and the increased vascular permeability tissue damage include the neutral proteases and the produced by simple amines, like histamine (Davies et al. , 1984). Those compounds that amplify the actions of hydroxyl radical. others are described as modulators of inflammation.

Mediator interactions are complex, and there are
 The properties and sources of some important media-
 compounds whose principal role is to interact synergis-
 tors and modulators of inflammation and joint disease
 tically with inflammatory mediators. For example, the
 are described in Table 62.1. It should be noted that the
 vasodilator prostanoid prostaglandin (PGE₂) enhances
 list is not comprehensive!

Table 62.1

Mediators and modulators of acute and chronic inflammation.

Putative mediator/modulator

Properties and source

Histamine

An amine produced from histidine by decarboxylation. Stored in mast cells, basophils and platelets. Mast cell degranulation leads to release into extracellular environment in immune and non-immune inflammation. Causes vasodilatation and increased permeability of small postcapillary venules. Local administration causes pain and itching 5-Hydroxytryptamine (serotonin)
 Another simple amine stored in platelets and mast cells. Increases permeability of small venules in rodents but probably at most a minor mediator in other species Bradykinin

A peptide containing nine amino acids that act on bradykinin receptors. Synthesized from plasma kinins de novo. Produces vasodilatation, increased vascular permeability and pain Neuropeptides

Main peptides involved are substance P, serokinin A (both tachykinins) and calcitonin gene related peptide. Induce mediator release, contract smooth

muscle and induce vasodilation Complement system

Several complement components including C3a, C5a, C8, C9 exert proinflammatory actions, e.g. vasodilatation, increased vascular permeability, mast cell degranulation, leucocyte chemotaxis and cytolysis

Eicosanoids

PGE2

Derived from cell membrane phospholipid, following release of the precursor arachidonic acid.

Produces prolonged dilatation of small arterioles and potentiates the increased vascular permeability and pain produced by other mediators like histamine and bradykinin PGI2

A potent dilator of small arterioles. Possesses similar properties to PGE2 but with shorter duration of action

PGI2

A derivative of PGD2. Believed to play a role as anti-inflammatory prostanoid in the resolution of acute inflammation

LTB4

Potent chemoattractant for polymorphonuclear leucocytes and mononuclear cells. Enhances vascular permeability produced by other mediators such as PGE2, PGI2 and bradykinin Lipoxin A

Produced from cell membrane phospholipid by polymorphonuclear leucocytes. Stimulates leucocytes to generate superoxide radical and causes release of lysosomal enzymes. Lipoxins also possess anti-inflammatory properties and are probably involved in resolution of acute inflammation

Platelet activating factor (PAF)

An ether-linked analogue of phosphatidylcholine. Derived from cell membrane phospholipid.

Produces platelet and neutrophil aggregation, bronchoconstriction, vasodilatation, increased capillary permeability and chemotaxis

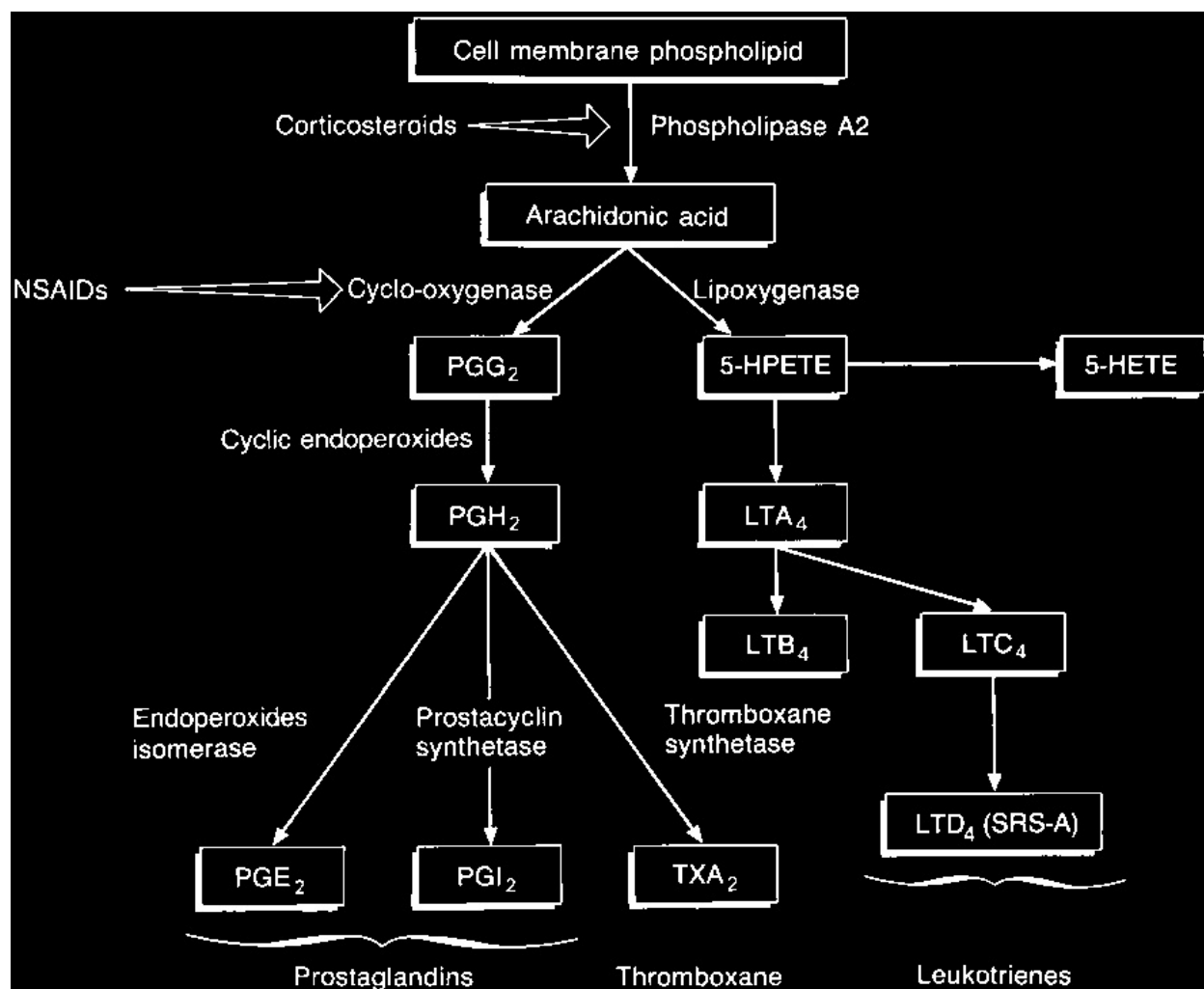
Interleukins and other

Produced by macrophages, chondrocytes and synovial cells.

cytokines, e.g. IL-1, TNF- α

Chemotactic for leucocytes and possess mitogenic and pyretic properties. Stimulate release of PGE₂ and collagenase by chondrocytes, synovial cells and dermal fibroblasts. Possibly involved in cartilage breakdown. Other cytokines possess anti-inflammatory properties Enzymes

Lysosomal enzymes such as lysozyme and acid phosphatase concerned with lysis of foreign and host cells in acute and chronic inflammation. Non-lysosomal metalloproteinases such as stromelysin, concerned with proteoglycan breakdown in arthritis



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Fig. 62.2

The arachidonic acid cascade illustrating pathways of generation of eicosanoids and proposed sites of action of steroids and NSAIDs.

The eicosanoid group of mediators and modulators

Classification of

is particularly important to a consideration of the two **anti-inflammatory drugs**

most important anti-inflammatory drug groups in cattle medicine, steroids and non-steroidal anti-inflammatory drugs (NSAIDs). Eicosanoids are formed from the 20-

In view of the complexity of the inflammatory process carbon unsaturated fatty acid substrate arachidonic and the involvement of many mediators and modulators in determining the time course of inflammatory reactions, it is inevitable that those anti-inflammatory component of the phospholipid of cell membranes. The drugs with activity against a single mediator have only release is catalysed by phospholipase A2, an acylhydro-limited clinical effectiveness. With currently available lase enzyme that is activated by tissue injury. Arachidonic acid may act as a substrate for two enzyme flow and oedema associated with acute inflammation groups, the cyclo-oxygenases (COX) and lipoxygenases

than it is to suppress chronic inflammation or the cartilage breakdown that characterizes degenerative joint disease. However, there have been some interesting recent advances in the latter field.

PGG₂ and PGH₂ (Fig. 62.2). Lipoxygenases, of which Table 62.2 gives a general classification of drugs that there are three groups, 5-LO, 12-LO and 15-LO, on the either possess anti-inflammatory properties or that other hand, are responsible for the synthesis of hydroxy-yeicosatetraenoic acids (HETEs), leukotrienes and that inhibit COX are the classical aspirin-like drugs lipoxins (Fig. 62.2). The importance of the arachidonic acid pathway in inflammation in general, and in veterinary therapeutics in particular, lies in the fact that the first compound of this class in clinical use was salicylate, derived initially from the bark of willow and

steroids and NSAIDs act, at least partially, by inhibiting enzymes in the arachidonic acid pathway (Fig. 62.2) is the acetyl derivative of salicylate and was introduced (Higgs et al. , 1981). The NSAIDs act by inhibiting COX into clinical medicine by Hoffman in 1898.

and thus they decrease the release of PGE₂ and TXA₂, More recently introduced are the drugs that inhibit since COX catalyses the conversion of arachidonic acid both 5-LO and COX enzymes, so-called dual inhibitors. released from cell membranes when they are damaged By inhibiting the synthesis of leukotrienes such as LTB₄ (Vane, 1971).

as well as prostanoids like PGE₂, these novel com-

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Table 62.2

Classification of anti-inflammatory drugs and drugs used in the treatment of joint disease.

Drug class

Examples

General properties

Antihistamines

Tripelenamine, mepyramine,

Classical histamine (H)1-receptor antagonists. Produce some

diphenhydramine

suppression of acute inflammation but limited to early phase of acute inflammation

COX inhibitors

Aspirin, phenylbutazone, flunixin,

Provide analgesia in inflammatory pain and some suppression of carprofen, meloxicam, tolifenamic

vascular changes in inflammation. Little or no effect on leucocyte acid, vedaprofen, ketoprofen

migration. Gastrointestinal ulceration may occur and blood

dyscrasias; hepatotoxicity and renotoxicity may arise

occasionally

Dual inhibitors of

Tepoxalin

Inhibit most changes in acute inflammation, possess fewer side-COX/5-LO

effects than pure COX inhibitors

Corticosteroids

Hydrocortisone, cortisone,

Suppress all components of acute and chronic inflammation but

dexamethasone, betamethasone,

side-effects associated with medium- to long-term use (adrenal prednisolone, prednisone,

atrophy, immune suppression, decreased bone strength,

triamcinolone

Cushinoid symptoms, delayed wound healing, muscle wastage,

etc.)

Disease modifying agents

Penicillamine, gold salts

Retard the degenerative changes in immune-based arthritides.

Very slow onset of effects (several weeks). Potentially severe side-effects, little veterinary use

Cytotoxic agents

Cyclophosphamide,

Used in rheumatoid arthritis in the dog. Immune suppression

chlorambucil

and other toxic effects to rapidly dividing cells limit clinical value Free radical scavengers

Orgotein, copper-containing

Possess superoxide dismutase activity. Scavenge toxic free

compounds, DMSO, some

radicals, e.g. superoxide, hydroxyl

NSAIDs

Miscellaneous

Hyaluronic acid, polysulphated

Inhibit prostaglandin synthesis and proteolytic enzymes, e.g.

glycosaminoglycan, pentosan

*stromelysin. Administration by intra-articular injection may be sulphate
required for some drugs*

*pounds may provide the basis for the introduction of
inhibit induction of one isoform of COX, described as
agents with a broader spectrum of activity than
COX-2 (see below).*

inhibitors of COX enzymes alone. They may be of use

*Of other drug classes listed in Table 62.2 several,
in immune-based diseases such as asthma, chronic
including immune stimulants, disease modifying agents
obstructive pulmonary disease and insect bite allergies.
and cytotoxic agents, have been used extensively to
However, only one compound of this class, tepoxalin, is
treat rheumatoid arthritis in man. They slow the pro-
currently in veterinary use in the dog. An interesting
gressive degenerative changes in cartilage and soft*

feature of dual inhibitors is their excellent gastrointestinal inflammation in this disease. Some have also intestinal tolerance.

been used for similar therapy in veterinary medicine, Corticosteroids are the anti-inflammatory agents that notably in the dog, but their use has been limited by provide a greater control of all elements of both acute several factors such as slow onset of action and potential and chronic inflammation than any other class of drug. tially severe side-effects have prevented extensive veterinary use. They exert membrane-stabilizing properties on lysosomal and cell membranes. Through this and other

Other drugs which have been used in the therapy of actions, they suppress the release of inflammatory joint disease, especially in the horse, are the free radical mediators and act to preserve cell integrity. Their mode scavengers orgotein and dimethyl sulphoxide (DMSO) of action is multifactorial. It is attributable partly to and drugs with uncertain mechanisms of action, their action on the cell nucleus to promote, by protein

hyaluronic acid and chondroprotective agents. Again, transcription, the synthesis of a group of endogenous the use of these compounds in bovine medicine has polypeptides with antiphospholipase activity, the been very restricted to date, mostly due to lack of lipocortins or annexins (Fig. 62.2). However, they also licensed products for use in food producing species.

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Non-steroidal

produce hyperalgesia, inflammation, pyrexia and

anti-inflammatory drugs (NSAIDs)

platelet coagulation (Fig. 62.2). Differences in relative potency between drugs for these activities are probably

Classification and mechanism of action

due to tissue differences in enzyme structure, i.e. the existence of isoenzymes. Two forms of COX have been The organic acid drugs in this important group of com- identified, COX-1, a constitutive enzyme, and COX-2, pounds can be divided into two subclasses, carboxylic which is expressed constitutively in some tissues, but is acids ($R-COOH$) and enolic acids ($R-COH$).

also inducible. The existence of COX isoenzymes may
Further subdivisions can be made on the basis of chem-
also explain species differences in plasma concentra-
ical structure. For example, the main enolic acid groups
tions required for efficacy. Therapeutic concentrations
are pyrazolone derivatives, such as phenylbutazone and
of phenylbutazone in the horse are of the order of
oxicams, like piroxicam; the carboxylic acid groups

10–30 mg/ml (Gerring et al., 1981), however, in cattle include the propionic acid
drugs such as ketoprofen.

even higher concentrations produce only limited COX

Most non-steroidal anti-inflammatory drugs (NSAIDs)
inhibition (Arifah & Lees, 2002).

possess three principal types of pharmacological activ-

All NSAIDs can have antithrombotic effects by

ity: peripheral analgesic and anti-inflammatory actions

blocking platelet COX-1, and thus inhibiting produc-

and central antipyretic, anti-inflammatory and analgesic

tion of the proaggregatory eicosanoid, TXA₂. However,

actions (Table 62.3). Some differences between drugs

at therapeutic doses most NSAIDs do not impair clot-

have been reported for the three types of activity.
ting mechanisms or prolong bleeding time. Aspirin is
Paracetamol possesses weak peripheral actions and
unique in this respect since it irreversibly binds to COX
does not produce significant gastrointestinal irritation.
so that production of TXA₂ is inhibited for the life-span
However, it is a relatively weak anti-inflammatory drug
of the platelet. Consequently, aspirin is a potent anti-
and is principally a centrally-acting analgesic (Lees et
thrombotic drug.
al., 1991; McKellar et al., 1991).

COX-1 may also contribute to the inflammatory
An important action of NSAIDs, underlying their
process and in particular to the pain component and,
therapeutic uses, is inhibition of COX and consequent
consequently, COX-2 selective inhibitors may not be
inhibition of production of the eicosanoids, which
as efficacious as mixed inhibitors in their anti-

Table 62.3

Principal therapeutic and toxic effects of NSAIDs.

Site of action

Pharmacological or toxicological action/therapeutic use

Therapeutic effects

CNS

Antipyretic. Lowering of body temperature in patients with fever but not in normothermic subjects. Action on temperature regulating centre in hypothalamus

Analgesic and anti-inflammatory. Mechanisms poorly understood

Peripheral

Anti-inflammatory in acute inflammation and analgesic against inflammatory, but not against non-inflammatory pain

Antithrombotic through inhibition of platelet COX. Aspirin, irreversible inhibitor of COX

Anti-endotoxic. Some effects on endotoxin due to release of eicosanoid synthesis being inhibited by NSAIDs

Toxic effects

Gastrointestinal tract

Ulcerogenic following parenteral as well as oral administration. Blood or plasma loss. Melaena. Emesis in simple-stomached animals only

Kidney

Nephropathies such as renal papillary necrosis

Liver

Both cholestatic and parenchymal cell toxicity

Blood cells and

Various dyscrasias including aplastic and haemolytic anaemias and

agranulocytosis described.

cardiovascular

Methaemoglobinaemia, hypoprothrombinaemia and prolongation of bleeding time. Small vein phlebopathy.

system

Alteration of acid-base and electrolyte balance

Skin

Urticaria, erythema

Fetus

Teratogenic and embryopathic effects with some drugs. Delayed gestation at term

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inflammatory actions.

COX-2 is constitutively

inflammatory agents and this suggests that the princi-

expressed in kidney, and mice which have the COX-2

pal mechanism of action, of at least some drugs, is due

gene deleted suffer from renal failure and are poorly

to some other effect. Carprofen, for example, has been

viable, while displaying inflammatory responses similar

shown to inhibit secretion of interleukin 6 (IL-6)

to normal mice. Both COX-1 and COX-2 are constitu-

(Armstrong & Lees, 2002).

tively expressed in the central nervous system (CNS)

In general, NSAIDs do not, at clinical dose rates,

and their relative expression varies depending on

affect all components of the inflammatory response.

species. More work is required to elucidate the role of

They do not affect cellular events or minimize tissue

these enzymes in CNS function. COX-2 generated PGs

damage, although high doses of some drugs do possess

are also involved in ovulation, parturition and may be

free radical scavenging properties, suggesting tissue

involved in the resolution phase of acute inflammation

healing mechanisms may be impaired, but more

and wound repair.

research in this field is required.

Until recently, it was considered that the PGs that

Other actions of NSAIDs have been described.

mediate inflammation, fever and pain were produced

Recent evidence suggests that some NSAIDs can act by

by COX-2, while the PGs that are important in gas-

mechanisms other than COX inhibition; for example,

gastrointestinal and renal function are produced via COX-
aspirin inhibits the transcription factor NF- κ B pathway

1. This led to the belief that the NSAIDs exerted their
(Kopp & Ghosh, 1994), and recently the R(-)- and
therapeutically beneficial effects primarily by inhibiting
S(+)-enantiomers of ibuprofen were reported to inhibit
COX-2, while inhibition of COX-1 was considered to

activation of NF- κ B (Scheuren et al., 1998), an effect be responsible for some of
the toxic side-effects associated independent of specific COX inhibition. By
preventing

associated with these drugs, e.g. gastric ulceration and renal
the translocation of NF- κ B into the nucleus, NSAIDs
papillary necrosis.

modify the expression of genes such as the COX-2 gene,

Recent work has indicated that the distinction

which is involved in the cellular response to acute

between the two roles of COX-1 and COX-2 is not as
inflammation. Furthermore, activation of the glutamate
clear as was once considered (for review see Wallace,
receptor, N-methyl D-aspartate (NMDA), a key step in
1999). Most commercially available NSAIDs at present
both the induction and maintenance of inflammatory

licensed for use in animals inhibit both enzymes. There hyperalgesia, activates NF- κ B (Guerrini et al., 1995). is much controversy over the selectivity of these compounds. An action on prostanoid receptors has been described for meclofenamic acid (see above), while some drugs on the test systems used for assessing COX-1 and COX-2 inhibit glucuronidase release from neutrophils. Recent work in humans has indicated a role for COX-2 inhibition in the therapy of colonic cancer. This may represent a new role for NSAID use in the future.

not being highly potent. Currently, there are no truly selective/specific COX-2 inhibitor drugs licensed for

Actions and interactions of NSAIDs and

use in veterinary medicine. The balance of evidence

general uses

currently available suggests that phenylbutazone, flunixin and ketoprofen are non-selective, whereas car-

The analgesic action of most NSAIDs on the CNS is

profen, meloxicam and tolafenamic acid are COX-2 weaker than that provided by narcotic analgesic drugs preferring, depending on the test systems used. There of the morphine type. To what extent the central anal- is, however, evidence of specific differences on COX1 : gesic effect accounts for the clinical efficacy of NSAIDs COX2 selectivity in ruminant species.

is unclear, but there is increasing recognition of its While most NSAIDs inhibit COX very effectively, importance for some drugs. The peripheral analgesic other actions, both on the arachidonic acid pathway and effect is thought to be due to blockade of synthesis on other unrelated enzymes, may contribute to their of PGs, which exert hyperalgesic actions through syner- effects. For example, some fenamate derivatives inhibit gism with pain-inducing mediators like bradykinin some (but not all) actions of PGs by blocking activity (Ferreira, 1983). Suppression of pain produced by tis- at PG receptors in addition to blocking their synthesis. sue damage is a major use of NSAIDs, together with High doses of most NSAIDs also block 5-LO, and thus

suppression of the acute inflammation associated with suppress leukotriene synthesis. However, it is doubtful tissue trauma. Post surgical use of NSAIDs to provide whether this action contributes significantly to the clinical analgesia and to reduce oedematous swelling that can lead to wound breakdown is now widespread. These exceptional in this respect. Finally, some NSAIDs drugs are clearly of value in the former circumstance, (carprofen is one example), although relatively weak but the doses required for anti-oedematous actions may COX inhibitors, are nevertheless effective anti-

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medicine. NSAIDs have been used increasingly alone to per cent per day. Longer inhibition can occur if significant amounts of aspirin penetrate to the developing diseases but, more usually, they are combined with megakaryocytes in bone marrow.

antibacterial agents. Endotoxic shock may result from a

This action of aspirin explains its use in preventing number of disease states when endotoxin derived from clot formation in humans who may be prone to throm- Gram-negative bacterial cell walls is released into the boembolic diseases. It is of interest that low doses circulatory system. This may occur, for example, in cases of aspirin are preferred in these subjects, because in- of equine colic, acute and peracute mastitis, metritis hibi- tion of synthesis of PGI₂, an endogenous anti- and in septic peritonitis. Some of the ensuing effects on aggregatory agent released from endothelial cells, by cardiovascular function and the respiratory system, high aspirin doses could offset blockade of synthesis together with metabolic and gastrointestinal changes, of TXA₂ by platelets. Low aspirin doses, administered are produced by the release of eicosanoids such as PGI₂ once daily or every second day, very effectively inhibit and TXA₂. Hence, the pathophysiological changes asso- platelet COX, without seriously impairing COX in ciated with endotoxaemia are generally suppressed, endothelial cells. The full potential for the antithrom-

though rarely abolished, by NSAIDs such as flunixin. The analgesic action of aspirin in veterinary medicine has not been fully explored, although it is recommended for use in prevention of aortic embolism in cats and may be of value in conditions such as laminitis, navicular disease and disseminated intravascular coagulation. However, the effectiveness of these drugs once shock is established is likely to be less than when pre-treatment is possible. The clinical value of NSAIDs therefore remains to be established; however, The use of two, or more, NSAIDs in combination generally has no clear advantages over the administration of a single drug at a higher dose rate. Most NSAIDs anti-endotoxaemic effects have been demonstrated for flunixin meglumine at doses considerably less than those required for anti-inflammatory or analgesic activity (approximately 0.25 mg/kg).

apeutic effects. Non-steroidal anti-inflammatory drugs
Prostaglandin E2 is a pyretic agent released as a result
have been licensed for the treatment of inflammation
of the action of endogenous pyrogens such as IL-1. The
in animal species and, more recently, their potent anti-
local release of PGE2 in the anterior hypothalamus
hyperalgesic activity has been recognized (Welsh &
leads to resetting of the thermoregulatory centre.
Nolan, 1994; Cheng et al., 1998). Although the meas-
NSAID inhibition of PGE2 synthesis accounts for their
urement of pain in ruminants has not been described
antipyretic action when infections are accompanied by
frequently, workers have described the occurrence of
fever. The reduction in body temperature gives symp-
hyperalgesia in animals suffering from lameness (Ley et
tomatic relief in such patients. However, NSAIDs may
al., 1995; Whay et al., 1998; Dolan & Nolan, 2000) and theoretically influence
adversely viral diseases, since the
mastitis (Fitzpatrick et al., 1998). While the magnitude production and release of
interferons are increased in
of hyperalgesia does not appear to be related to the
pyrexia. In spite of this, NSAIDs have been used in the

stimulus intensity, the duration of hyperalgesia does treatment of respiratory disease produced by parainfluenza type 3 (PI3) virus in calves and in the therapy of acute interstitial pneumonia caused by Ascaris suum by Fitzpatrick et al., 1998; Dolan & Nolan, 1999; Dolan in pigs (Selman et al., 1984; Selman, 1988). Both the

& Nolan, 2000). Inflammatory conditions that induce anti-inflammatory action in the lungs and the central hyperalgesia therefore probably induce spontaneous antipyretic action may underlie the therapeutic pain in the first instance, and contribute to adverse response in these diseases.

welfare over a longer duration (Dolan & Nolan, 2000).

Aspirin differs from other NSAIDs in that its

Thus, measurement of hyperalgesia is potentially a inhibitory action on COX is irreversible (Lees et al., useful tool in validating disease severity and since it is

1987). It covalently acetylates the enzyme, which is, a consequence of altered pain processing, it provides a in consequence, permanently inhibited. In most body

pathophysiological link between disease and pain.

cells new COX enzyme can be synthesized in the cell nucleus. As aspirin concentration falls the cell's ability

Pharmacokinetics of NSAIDs

to form the products of COX metabolism is restored.

However, the platelet cannot do this. The platelet is

The pH of fluids within the gastrointestinal tract varies

anuclear and the enzyme is thus inactivated perma-

but at all sites it is usually more acidic than plasma.

nently, i.e. for the lifespan of the platelet. Partial inhi-

In the stomach of monogastric species, fluid pH can be

bition may persist for several days since platelet

as low as 1.0. This marked difference from plasma

numbers are replaced at the rate of approximately 10

increases, by the mechanism of ionic trapping, the rate

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at which NSAIDs are absorbed. Being weak organic

Thus many NSAIDs are retained at the site of inflam-

acids they are relatively less ionized in gastrointestinal

mation for periods long in excess of those predicted by

tract liquor than in plasma and this facilitates absorp-

the plasma half-lives. In consequence, there are significant differences in dosing schedules between species. As well as species differences in pharmacokinetics, the rumen, where liquor pH is also acid relative to plasma. However, the normal pH, approximately 4–5, is less acid than the gastric juice of monogastric species. Moreover, differences occur for any NSAIDs in other species, other factors are involved in determining rates of absorption. Low drug solubility in acidic conditions for some NSAIDs, such as aspirin, can lead to precipitation and safe dosage schedules. in the stomach and this prolongs and slows absorption. A further factor relevant to the activity of some NSAIDs is the formation in vivo of active metabolites.

an important factor in horses and ruminants, since some

The conversion of aspirin to salicylate is one example.

NSAIDs may be adsorbed on to hay. Such binding to

Another is the hydroxylation of phenylbutazone in

feed, with subsequent release of the drug by fermenta-

several positions. One such metabolite, oxyphenbuta-

tive digestion within the large intestine of the horse, has

zone, is of similar potency to phenylbutazone and is

been suggested as a cause of the ulcerogenic action of

formed in the liver in quantities that are sufficient to

phenylbutazone in the distal parts of the gastrointesti-

contribute about one-fifth of the overall activity in the

nal tract (Lees & Higgins, 1985; Maitho et al., 1986). In horse. Plasma concentrations achieved in the goat may

spite of these factors, absorption of most NSAIDs is

also be significant, but in both young and adult cattle,

generally good after oral dosing in all species and

concentrations are low or undetectable and no contri-

bioavailability generally exceeds 50 per cent.

bution of oxyphenbutazone to efficacy can therefore be

Another factor that may potentially markedly influ-

assumed in cattle.

ence NSAID absorption following oral dosing in rumi-

Age may also affect NSAID pharmacokinetics. In

nants is operation of the oesophageal groove reflex.

general, both urinary excretion and hepatic biotrans-

Closure of the groove can direct orally administered

formation of drugs are less efficient in very young

substances directly to the abomasum and this will gen-

animals, aged less than two to six weeks depending on

erally speed absorption into plasma, since dilution

species. Aged animals may also metabolize and excrete

within the vast fluid volume in the rumen inevitably

NSAIDs less readily than young adults. There is some

extends the time course of absorption. Double peaks in

evidence in support of this for phenylbutazone in the

the plasma concentration–time curve of the NSAID

horse (Lees et al. , 1985). Reduced capacity to metabo-meclofenamate after oral dosing in sheep have been

lize drugs in the liver is likely to be of greater signifi-

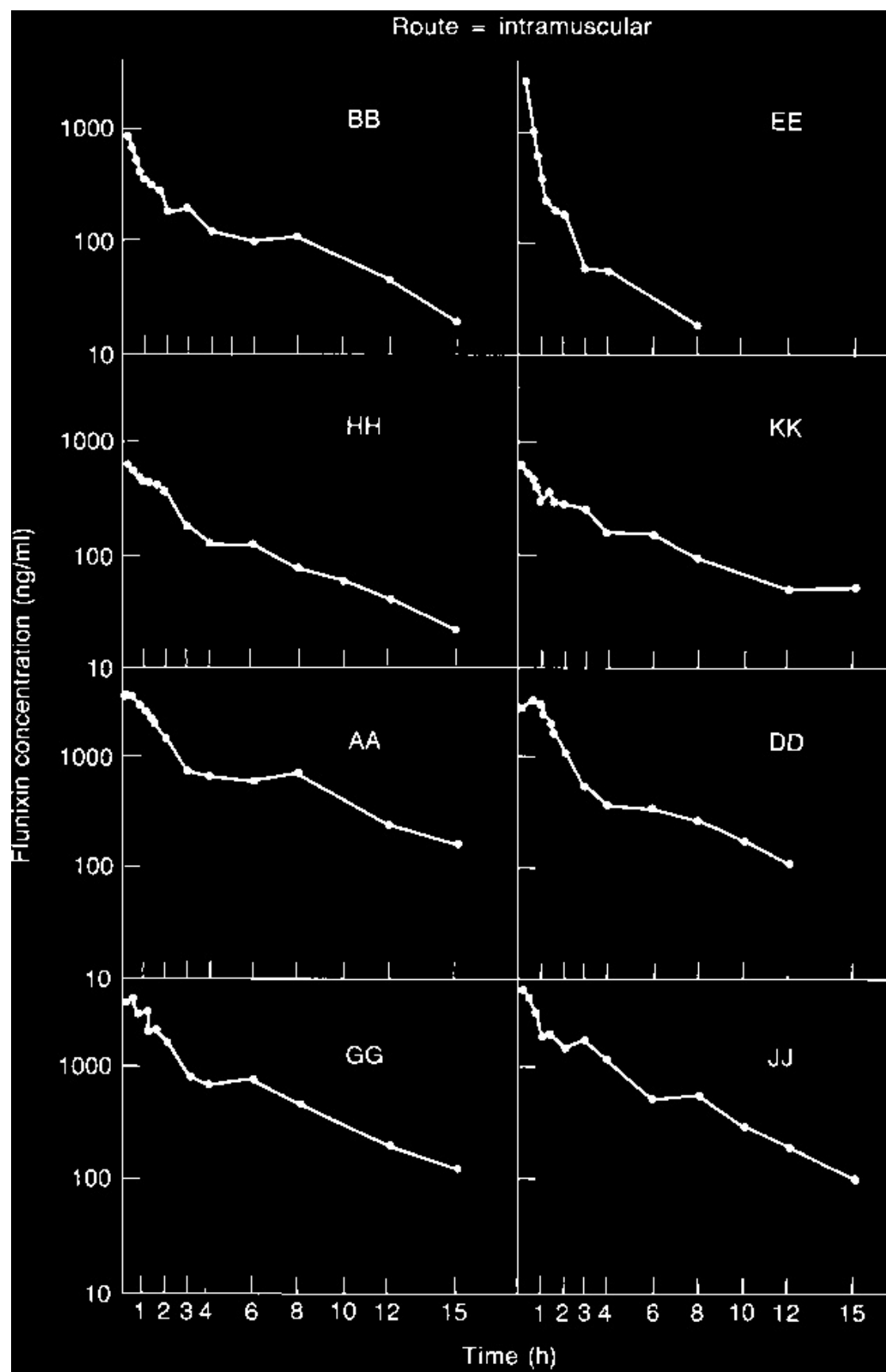
attributed to an initial rapid absorption phase from the

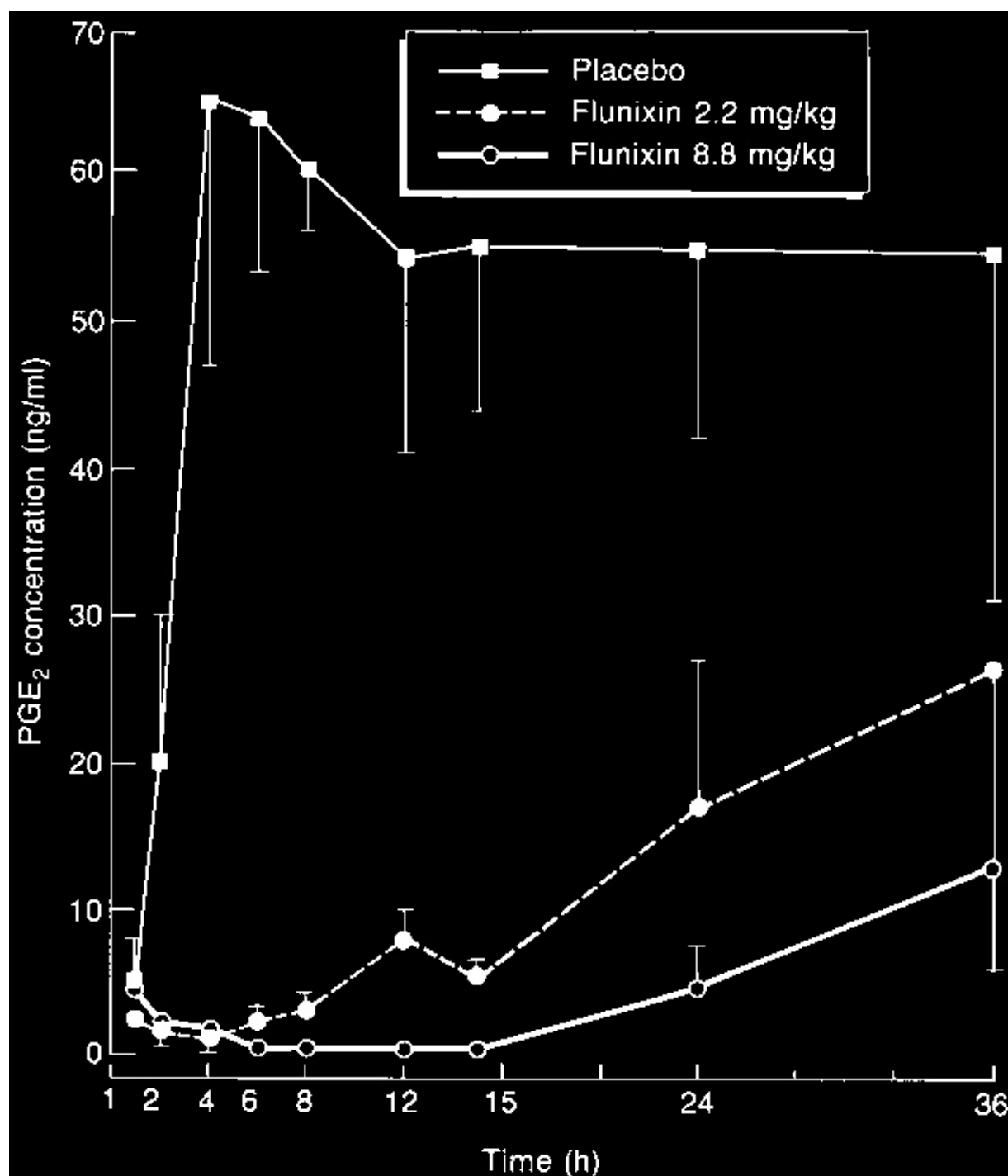
cance than any decreased renal excretory capacity in

abomasum and slower subsequent absorption from the the case of NSAIDs, since virtually all are highly bound rumen-reticulum of that fraction of the administered (usually greater than 98 per cent) to plasma protein. dose which enters the rumen (Marriner & Bogan, Consequently, less than 2 per cent of the drug in plasma 1979). A similar mechanism of oesophageal closure is available for excretion in urine by glomerular filtration. This small fraction is readily excreted in the urine adult cattle (Lees et al. , 1988). Further studies are of herbivores, since it is 'trapped' in ionized form in the required to determine to what extent species, drug sub-alkaline urine, i.e. tubular reabsorption is limited or stance, drug formulation and age determine operation absent. Nevertheless, only a very small proportion of of the oesophageal groove reflex. NSAIDs are usually the administered dose will be removed from the body administered parenterally. Some NSAIDs are irritant by urinary excretion of parent drug. Reduced dosage or to tissues when injected perivascularly but flunixin,

longer intervals between doses is probably required in meloxicam and carprofen can be given intramuscularly both young and old animals.

or subcutaneously when absorption is rapid and tissue Limited and indirect evidence indicates that at least irritation is minimal. Species differences in pharmacokinetic parameters, particularly clearance and elimination in cattle, possibly with subsequent reabsorption to create an entero-hepatic shunt. This is suggested, for example, by the 'kink' in the plasma concentration–time curve that occurs consistently following the administration of flunixin intravenously and intramuscularly to calves (Fig. 62.3). Studies of bile : plasma concentration ratio are required to investigate this possibility further.





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Fig. 62.4

Influence of two dose rates of flunixin (2.2 and 8.8

mg/kg body weight) administered intramuscularly to calves on the generation of the pro-inflammatory mediator, PGE₂, in carageenan-induced inflammatory exudates. Each point is the mean \pm SEM for six animals.

Fig. 62.3

Plasma concentration–time curves for eight calves

clearance of carprofen was slower in animals aged 8

receiving flunixin meglumine by intramuscular injection at two weeks than in 16 week old animals (Lees et al. , 1986; dose rates: 2.2 mg/kg body weight (BB, EE, HH, KK) and 8.8

Delatour et al. , 1996). Profound differences in ketopro-mg/kg body weight (AA, DD, GG, JJ).

fen pharmacokinetics were demonstrated in young

goats compared to adult animals (Eltom et al. , 1993).

Second, the pharmacological actions may be longer for

Knowledge of the pharmacokinetics of particular

NSAIDs than might be predicted from plasma clear-

NSAIDs cannot be transposed between species since

ance data. Studies have shown that relatively prolonged

interspecies differences are marked. Pharmacokinetic

inhibition of production of the inflammatory and hyper-

data in cattle are available for flunixin, tolfenamic acid,

algesic mediator, PGE₂, is produced by therapeutic ketoprofen, carprofen and meloxicam and these drugs doses of flunixin, tolafenamic acid and ketoprofen, have licensed products in the UK for use in food-administered to calves (Landoni et al. , 1995a, 1996; Lees producing species (NOAH Compendium of Data Sheets et al. , 1998) (Fig. 62.4) and, moreover, there are likely for Veterinary Products 2000–2001). Pharmacokinetic to be species differences in IC₅₀ values for inhibition of data are available for other drugs including aspirin and COX isoenzymes. This has been shown for example in phenylbutazone, neither of which are licensed currently studies comparing COX isoenzymes from the horse and for use in food producing animals. Phenylbutazone has the dog (Brideau et al. , 2001; Armstrong & Lees, 2002). a long half life (40–60 hours) in both calves and adult In cattle, tolafenamic acid was shown to significantly cattle (Arifah & Lees, 2002). Even when basic pharmacokinetic data are available, it is not possible to define measured as indicators of blockade of the isoforms dosage schedules with such data alone for two reasons.

COX-1 and COX-2, respectively (Lees et al. , 1998).

First, different plasma and tissue concentrations of

Whilst penetration of these drugs into tissue cage fluid

NSAIDs are likely to be required for the various

was poor, passage into acute inflammatory exudate

therapeutic effects of analgesic, anti-inflammatory,

was good and clearance from the latter fluid was slow,

antipyretic and antithrombotic actions. In calves,

i.e. there was a strong tendency for them to accumulate.

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It is likely that three distribution compartments

melaena. For example, high doses of phenylbutazone

relating to the different sites of action of the NSAIDs

administered to the horse can quickly lead to significant

exist: a central compartment and shallow and deep

plasma loss, haemoconcentration and death from hypo-

peripheral compartments (Landoni et al. , 1995b, 1996).

volaemic shock only a few days after the commence-

The very high degree of binding to plasma protein

ment of therapy. The horse may be particularly

probably explains both the relatively high concentra-

susceptible to this effect of phenylbutazone (Lees & Higgins, 1985).

tion in inflammatory exudate, into which plasma
Until recently,

it was considered that the
interstitial fluid into which it does not. Similar con-
prostaglandins that mediate inflammation, fever and
siderations apply to most NSAIDs, since almost all
pain were produced by COX-2,

while the
are highly bound to plasma protein (Galbraith &
prostaglandins that are important in gastrointestinal
McKellar, 1996).

and renal function are produced via COX-1. This led to
the belief that the NSAIDs exerted their therapeuti-
cally beneficial effects primarily by inhibiting COX-2,

Side-effects and toxicity of NSAIDs

while inhibition of COX-1 was considered to be respon-
At high dose rates NSAIDs may affect a number of bio-
sible for most or all of the toxic side-effects associated

chemical pathways. However, with clinical doses the with these drugs, e.g. gastric ulceration and renal papillary necrosis. Recent work has indicated that the side-effects are probably caused primarily by inhibition of COX. The distinction between these two roles is not as clear as was previously thought. There are exceptions, however. Carprofen is a relatively weak inhibitor of COX, except in sheep where it produces marked inhibition (Cheng et al. , 2002), but it is, gastro-protective and does not induce gastric ulceration. Nonetheless, an effective anti-inflammatory agent. However, COX-2, while expressed in low amounts in a healthy stomach, appears to play an important role in irritation leading potentially to ulceration of the gastrointestinal mucosa. The NSAID side-effect reported most commonly is promoting ulcer healing in the stomach. Consequently, where gastric ulceration is present, COX-2 inhibitors at high dose rates and with most drugs some gastric irri-

may delay healing as, indeed, may non-selective COX tation is associated with oral dosing with therapeutic inhibitors.

dose levels. The mechanism of action may involve Useful data on the ulcerogenic actions of particular back-diffusion of acid, although ulceration is not NSAIDs in cattle and any relationship to age, dose restricted to the stomach; it may occur throughout the administered and route of administration are generally gastrointestinal tract and it may accompany intra-lacking. However, there is no reason to suppose that venous as well as oral dosing. The vasodilator ruminant species are not susceptible to the ulcerogenic eicosanoid PGI₂, generated in the COX pathway, may action of these drugs. Attempts have been made to be a local hormone controlling blood flow to the gas-reduce the degree of gastrointestinal tract irritation by trointestinal mucosa. By inhibiting the synthesis of the use of slow-release formulations, enteric coating PGI₂, NSAIDs may produce ischaemia and hence and water-soluble salts. The use of such approaches in

hypoxia of the mucosal surface. This may be the cattle does not seem to have been described in the literature. In ruminants, in general, NSAIDs are administered parenterally.

The ratio of doses of NSAIDs producing ulcerogenicity relative to anti-inflammatory and analgesic activities seems to be low for some drugs, e.g. aspirin, tubular nephritis. Renal papillary necrosis has also been intermediate for others such as phenylbutazone and reported in man. In horses papillary necrosis has been high for some selective agents, e.g. COX 2 inhibitors. It is, however, not possible to draw general conclusions concerning particular NSAIDs since many factors have been claimed to influence their ulcerogenicity

Retention of fluid leading to oedema is a commonly (Rainsford, 1977); these include species, age, sex and the reported side-effect of NSAIDs in humans. Oedema animal's nutritional status. Moreover, high drug concentrations in bile for some NSAIDs may explain has been described in horses receiving phenylbutazone lesions occurring in distal regions of the gut. When (Lees et al. , 1983). Cholestatic and parenchymal hepa-severe, the gastrointestinal ulceration induced by totoxicity have also been reported in animals. Since NSAIDs can lead to plasma protein-losing enteropathy most NSAIDs are metabolized in the liver, any toxicity or to blood losses into the gastrointestinal tract and to parenchymal cells might, potentially, lead to a cycle

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of drug cumulation and toxicity. Again, evidence for in lactating dairy cattle, including flunixin, ketoprofen, drug-induced hepatotoxicity has been described for tolfenamic acid and meloxicam.

horses receiving a high dose rate of phenylbutazone,

but whether similar effects occur in cattle has not been

Steroids

reported.

A number of blood dyscrasias have been reported in

Classification and general properties

human subjects treated with NSAIDs, generally over

long periods. Similar blood cell effects in dogs, cats and

Three classes of steroid hormone are secreted by the

horses have been described. However, the data avail-

adrenal cortex into the peripheral circulation: sex

able are limited and it has not been established whether

steroids (generally in small quantities only), mineralo-

particular drugs or certain species are especially impli-

corticoids and glucocorticoids. The latter two groups

cated. With the use of clinical dose rates of NSAIDs,

together comprise the corticosteroid hormones. The

the incidence of blood dyscrasias in animals seems to

mineralocorticoids, secreted by the zona glomerulosa

be low.

and zona fasciculata, include aldosterone; their princi-

At high dose rates some NSAIDs, notably aspirin and

pal endocrinological actions are exerted on electrolyte particularly when administered for long periods, impair and water balance. They promote sodium and chloride platelet aggregation and thereby affect blood clotting. retention and potassium loss by their actions on the Potentially, this may lead to internal or external bleed-renal distal tubule and on parts of the gastrointestinal ing. Another problem of impaired clotting, through an tract, notably the large intestine. Sodium retention is indirect mechanism, may occur when NSAIDs are used accompanied by osmotically obliged water. When together with anticlotting drugs of the coumarin group. administered as drugs or secreted in excessive amounts For example, the combination of phenylbutazone and in some disease states, aldosterone and related com-warfarin has been used in the therapy of navicular pounds cause oedema. Glucocorticoids influence lipid, disease in the horse. Phenylbutazone-induced suppress-carbohydrate and protein metabolism and some exert sion of warfarin metabolism may lead to warfarin tox-weak mineralocorticoid effects also. The principal glu-

icity. Both cardiovascular and respiratory effects result from the glucocorticoid hormone secreted by the zona fasciculata when very high doses of NSAIDs are administered, but the zona reticularis in most species is cortisol. Acute cardiovascular/respiratory effects do not generally occur with normal dose rates.

The main veterinary use of glucocorticoids derives from the fact that they are potent anti-inflammatory agents. Cortisol itself is used as an anti-inflammatory agent, being administered systemically or applied locally.

Residues

Published data on NSAIDs and their metabolite

locally. In addition, a number of synthetic steroids with residues in cattle are sparse. Tissue concentrations of

much greater glucocorticoid potency than cortisol and

meclofenamate and phenylbutazone are lower than

with the advantage of reduced mineralocorticoid

corresponding concentrations in plasma and, for the

potency are available for clinical use. For example,

latter, drug plasma and tissue concentrations decrease

prednisone and prednisolone are some four to five in parallel (Toutain et al. , 1982). Pharmaceutical manu-facturers have submitted residue data to registration they possess slightly weaker mineralocorticoid activ-ities. Unwanted side-effects on water and electrolyte balance are therefore less likely when prednisone and meloxicam in cattle. Theoretically, the passage of prednisolone are used at equi-effective dose rates to NSAIDs into milk in healthy cattle will be severely limited by the more acid pH relative to plasma and by corticosteroids in producing glucocorticoid and miner-the high degree of drug binding to plasma protein. In practice, this has been found to be the case for one drug, been reported by Keen (1987) and are presented in Table 62.4. It will be seen that methylprednisolone and

of plasma concentration), and will almost certainly triamcinolone are about five times as potent as cortisol apply to others. In mastitis, however, the pH of milk as glucocorticoids and both have little or no mineralocorticoid activity. For dexamethasone and betamethasone, effects on electrolyte balance are also slight, but milk may occur and it is necessary therefore that milk these drugs are 25–30 times as potent as cortisol as glucocorticoids. In contrast, the synthetic corticosteroid adhered to by farmers and veterinary practitioners. deoxycorticosterone acetate (DOCA) is a potent mineralocorticoid with negligible glucocorticoid activity.

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Table 62.4

Relative potencies and biological half-lives of some corticosteroids used in veterinary practice (from Keen, 1987).

Relative potency (hydrocortisone = 1.0)

Biological half-life (h)

Glucocorticoida

Sodium retaining

Hydrocortisone (cortisol)

1

1

8–12

Cortisoneb

0.8

0.8

Prednisolone

5

0.8

12–36

Prednisoneb

4

0.8

Methylprednisolone

5

Minimal

Triamcinolone

5

None

24–48

Dexamethasone

30

Minimal

36–54

Betamethasone

25

Negligible

Flumethasone

100

Negligible

a Closely paralleled by anti-inflammatory and HPA-suppressant activity.

b Inactive until converted to active drug in the body.

*Anti-inflammatory potency of corticosteroids closely
tissue. For example, in some species (rabbit, mouse)
parallels glucocorticoid activity. Clinically, this is most
high doses reduce thymus weight and reduction in the
important since it means that whilst mineralocorticoid
size of the spleen and lymph nodes occurs. Immuno-*

activity has been divorced from anti-inflammatory suppression by glucocorticoids is reflected in a number of ways, principally by a reduction in cell-mediated immunity and, usually at higher dose rates, by suppression of the general actions of glucocorticoids on antibody production. The secretion of mineralocorticoid hormones is regulated in a number of ways, side-effects, which are described in a subsequent section but the principal mechanism involves the renal release of this chapter. Glucocorticoids are gluconeogenic, promote the release of renin from the juxtaglomerular apparatus and sub-sequent conversion of angiotensin I to angiotensin II, moting the release of amino acids from skin, muscle and connective tissue, and they subsequently increase the secretion of angiotensin II, which acts on the adrenal cortex zona glomerulosa cells. Glucocorticoids also promote the conversion of amino acids to glucose. Glucocorticoids produce hyperglycaemia in many species, especially in

the other hand, is increased in response to stress, but ruminants, and they promote the storage of glucose as there is also a basal level of secretion that is subject to liver glycogen, although both uptake and utilization of a diurnal rhythm. In the dog, cortisol secretion peaks glucose by other tissues are reduced. In general, rates during the day, usually late morning, whilst in nocturnal of lipolysis are increased, leading to fatty acid and glyc-species, like the cat, peak levels occur at night. The erol release from adipose tissue. Subsequently, the glyc-regulatory control involves secretion of adrenocortierol may be converted to glucose. Calcium metabolism cotrophic hormone (ACTH) from the anterior pituitary, which in turn is regulated by the release of calcium absorption from the intestines is decreased and corticotrophin releasing factor from the hypothalamus renal excretion is increased. Second, there is increased (Fig. 62.5). There is a negative feedback of cortisol on parathormone secretion and a resulting osteoclast this system and high levels of glucocorticoid drugs

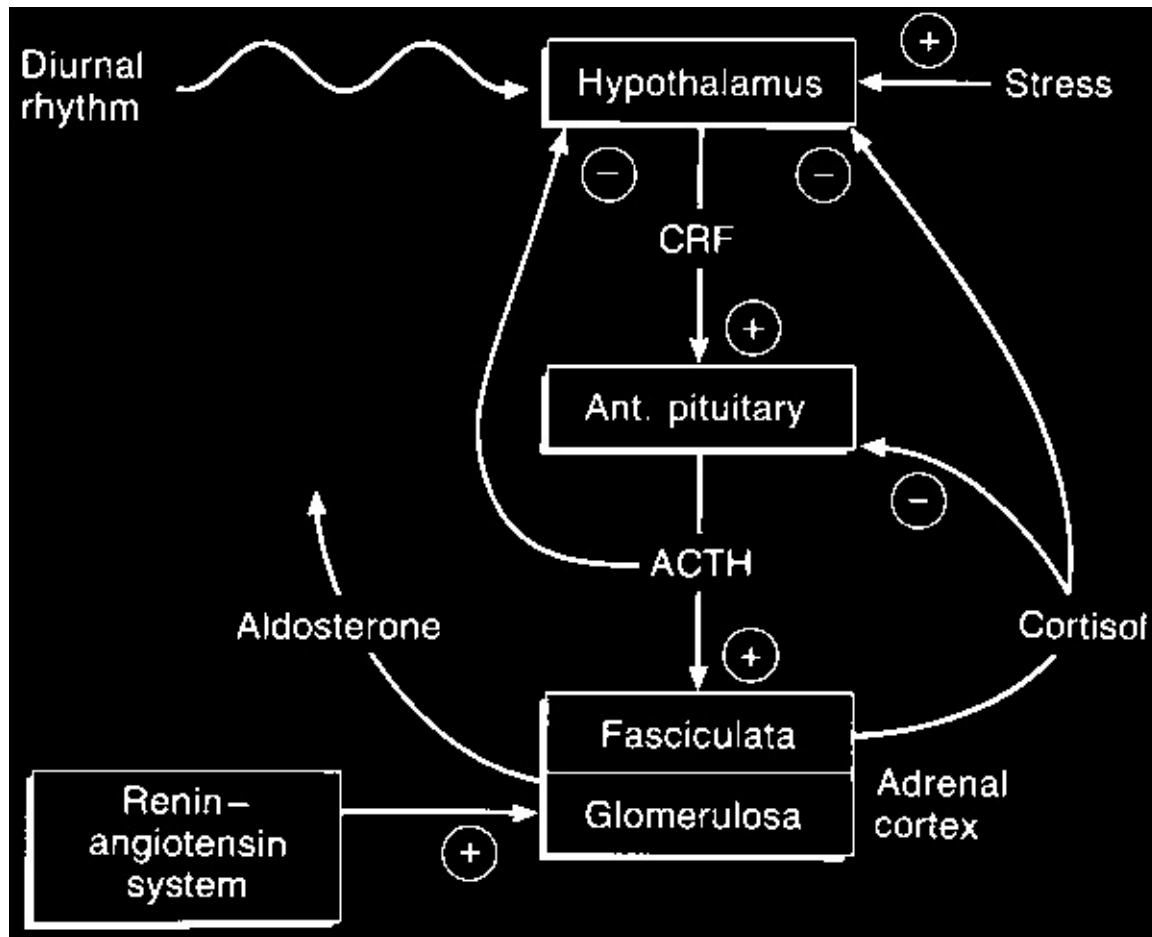
stimulation whilst osteoblast activity is inhibited. These in the circulation similarly suppress ACTH secretion. actions together promote calcium mobilization from bone, and bone strength is reduced. Long-term suppression induced by steroid drugs leads to bone, and bone strength is reduced. Glucocorticoids not only to inhibition of cortisol secretion, but also to cause diuresis by dilating glomerular afferent arterioles reduced cell function to the point of adrenal atrophy. and thus increasing glomerular filtration rate and also by depressing tubular water reabsorption in the distal

Mechanism of action

nephron.

The main action of glucocorticoids on blood is to The mode of action of steroids is multifactorial. Unlike promote leucophilia, principally reflecting an increased NSAIDs, they generally suppress all components of number of circulating neutrophils. However, circulating acute and chronic inflammation. Indeed, steroids are lymphocyte and eosinophil numbers are reduced. In the anti-inflammatory drugs 'par excellence'. Steroids addition, glucocorticoids are depressant to lymphoid

inhibit the vascular dilatation in the microcirculation



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response. Because of this delay, it follows that any relationship between effect achieved and plasma or tissue drug concentration will be complex. Drug effects will generally outlast the period for which they can be detected in biological fluids. This may explain why dexamethasone, which has an elimination half-life of approximately 1 hour in horses and 2.5 hours in cattle

(Toutain et al. , 1982), possesses a 'biological' half-life of 36–54 hours (Table 62.4).

As discussed earlier, arachidonic acid may act as a substrate for two groups of enzymes, COX and LO.

Through the production of lipocortins and consequential blockade of phospholipase A2 steroids can be expected to block the synthesis of COX-derived inflammatory mediators, including PGE2 and PGI2, and LO-derived HETE and leukotriene compounds. One of

Fig. 62.5

Pathways of mechanisms of control of secretion of adrenal steroids.

these, LTB₄, is a potent chemotactic agent for neutrophils and the peptidoleukotrienes LTC₄ and LTD₄ are believed to be important mediators of allergic and the increased capillary and small vein permeability airway diseases. They may be involved in such conditions and they thereby lessen oedema. Steroids also inhibit conditions as fog fever in cattle and chronic obstructive hyperalgesia and fibrin deposition. The extravascular pulmonary disease in horses. The evidence that anti-

migration of polymorphonuclear leucocytes initially inflammatory steroids act through the production of and subsequent movement of monocytes are reduced. lipocortins and blockade of eicosanoid synthetic path-

In addition, phagocytic activity of leucocytes is

ways is, however, controversial (Lane et al. , 1990). High depressed. In the repair stage of the inflammatory

doses certainly do affect eicosanoid synthesis, but response and in chronic inflammation, proliferation and whether doses used in veterinary therapy effectively do the deposition of collagen are suppressed. In consequence, wound healing and cicatrization are delayed.

steroids at the molecular level is inhibition of induction Glucocorticoids also decrease the production of inflammatory complement components, reduce lymphocyte accumulation and decrease production of interleukin-2

of COX-2 in response to inflammatory stimuli.

Administration and pharmacokinetics

(IL-2) and other lymphokines from lymphocytes.

of steroids

Glucocorticoids exert a stabilizing action on all biological membranes. Cell, mitochondrial and lysosomal membranes are affected and this suppresses the synthesis or release of acute inflammatory mediators such as PGE₂ and histamine, and subsequently the tissue breakdown arising from the effects of released lysosomal and non-lysosomal enzymes is suppressed. The application of steroids topically to skin and mucous membranes, e.g. the eye, is commonly used in veterinary practice. It provides high drug concentrations at required sites of action and at the same time minimizes the risks of inducing systemic side-effects, although some systemic absorption is likely. In addition, most However, these actions, although relevant to the anti-steroids are readily absorbed from the gastrointestinal tract, so that licking sites of drug application can lead to significant absorption from the gut. In some topical Many studies have shown that one biochemical products local activity is high, but systemic absorption

pathway inhibited by glucocorticoids is the arachidonic acid cascade. Arachidonic acid is released from its esterified form from the phospholipid component of cell membranes by the action of phospholipase A₂ (Fig. 62.2). Anti-inflammatory steroids inhibit this enzyme. Oral corticosteroid administration is a frequently used route for medium or long-term therapy. There are endogenous polypeptides, the lipocortins, which possess unavoidable potential risks of significant suppression of antiphospholipase activity. This indirect mechanism of action may account for the lag period of 1–5 hours between steroid administration and the biological ways of reducing this to a minimum have been sought.

esters with activity restricted to local sites.

62.2). Anti-inflammatory steroids inhibit this enzyme

Oral corticosteroid administration is a frequently indirectly by promoting the synthesis of a family of

used route for medium or long-term therapy. There are endogenous polypeptides, the lipocortins, which possess unavoidable potential risks of significant suppression of antiphospholipase activity. This indirect mechanism of the hypothalamic–pituitary–adrenal axis (HPA) (see Chapter 67) when long-term treatment is employed and

between steroid administration and the biological ways of reducing this to a minimum have been sought.

A short-acting preparation administered at twice the achieve benefits without the undesirable side-effects of recommended dose rate on alternate days instead of high doses.

normal dosing once per day has been proposed on Some steroids are prodrugs, requiring metabolic conversion in the body. This is true of cortisone and prednisone which are converted, respectively, to cortisol and on the other hand, may persist since steroid biological half-life prednisolone. Such drugs may be used for systemic therapy, but they are not applied locally.

life (Table 62.4). Another approach has been to recommend drug dosing in the morning in the dog and in the

Side-effects of steroids

evening in the cat. Then, maximum circulating concentrations of drug can be expected to coincide with peak The side-effects of those steroids with mineralocorticoid circulating concentrations of endogenous cortisol. This,

coid activity, e.g. prednisolone and prednisone, include in theory, will produce HPA suppression for the sodium and water retention and resulting oedema, and minimum time over each period of 24 hours.

hydrogen and potassium loss leading to hypokalaemia

Corticosteroids are frequently administered pa-

and metabolic alkalosis. Effects arising from glucocor-
renterally and generally this will give less variable

ticoid action on the kidney include a transient polyuria

plasma concentrations than oral dosing for short- or

and a compensatory polydipsia. Hyperglycaemia, pos-

medium-term therapy. Water-soluble formulations

sibly leading to glucosuria, also occurs. Protein catabo-

include succinates, phosphates and m-sulphobenzoates.

lism with long-term therapy causes muscle wasting and

They may be administered intravenously or intramus-

in the short term may delay wound healing. The lipoly-

cularly. Pharmacokinetic properties have been reported

tic action of glucocorticoids is associated with redistri-

for commonly used corticosteroids. Thus, with intra-

bution of body fat with the characteristic 'moonface'

venous dosing the half-life of dexamethasone ranges
appearance in man and pendulous abdomen in animals.

from 290 to 335 minutes in the cow, 53 minutes in the

With prolonged treatment, changes in calcium metabo-

horse to 110 to 130 minutes in the dog (Toutain et al. , lism can lead to
osteoporosis and bone fractures.

1982).

Gastrointestinal side-effects include reduced motility,

The water-soluble short-acting esters are rapidly
thinning of the gastric mucosa and reduced mucus pro-
absorbed from intramuscular injection sites to give an
duction. However, unlike NSAIDs, glucocorticoids are
onset and duration of action almost as quick as and of
not normally regarded as ulcerogenic. Nevertheless,
fairly similar duration to intravenous administration. In
colonic perforation in corticosteroid treated dogs has
addition, water-insoluble esters providing longer dura-
been described (Toombs et al. , 1986). The immunosup-
tion of action, from 2 to 14 days, are available. These
pressant actions involve a reduction in cell-mediated
suspensions, providing a depot at the injection site,

immunity, suppression of lymphoid tissue and lymphocyte movement and activity and, possibly, reduced anti-propionate esters. Some very insoluble esters, e.g. body production.

acetone and adamantate, provide effective concentrations over several weeks, and the use of these esters terminated, the HPA axis suppression present during involves undesirable, but unavoidable, HPA axis suppression. therapy is restored only over several weeks and animals

are susceptible to stress during this period. Stepwise Intra-articular injection, for the treatment of joint reduction in steroid dose rate and increasing the inter-diseases, is another route employed. Intra-articular dose interval may be helpful in assisting restoration of dosing reduces but does not prevent the risks of systemic HPA axis function.

temic side-effects, since some formulations are rapidly absorbed. Long-acting formulations maintaining local

General uses of steroids

concentrations for several days are sometimes used.

A careful aseptic technique is required with all intra-

Anti-inflammatory steroids are widely used for the articular injections. Steroids are used in this way to suppress soft tissue inflammation, reduce stiffness and pain conditions. Allergic and non-allergic eczemas respond to and allow more joint movement. Disadvantages are steroid therapy and steroids are used in the treatment that they may suppress natural repair processes and of inflammatory conditions of the eye and the ear. In dogs and cats, steroids have been used to terminate the Steroids are chondrodepressant and can lead to 'steroid itch-scratch cycle in non-specific dermatoses. Steroids arthropathy'. However, in recent years it has been increasingly recognized that low dose steroids can have been used extensively in the therapy of anaphylactic, endotoxic and haemorrhagic shock, but there are

several disadvantages. The slow onset of action of mended and a number of contraindications may be steroids has been discussed earlier and very large doses stated.

are required, making therapy in cattle and other large animal subjects impractical and/or prohibitively expensive. Administration to patients in shock should be by Guidelines

intravenous injection since absorption from intramus-
(1)

As animals vary in their response, depending on ular and subcutaneous sites in shocked patients would disease severity, dosing schedules should be estab-
be slow.

lished for individual subjects.

In inflammatory conditions associated with bacterial
(2)

Single large doses of steroids or short-
infections, steroids are commonly administered with
term therapy are unlikely to produce serious
appropriate antibacterial agents. However, the im-

side-effects.

munosuppressant actions are undesirable and some

(3)

The incidence and severity of side-effects gener-

authorities believe that such combinations are not jus-

ally increase with the duration of treatment.

tified. Where antimicrobial cover cannot be provided,

(4)

Corticosteroid treatment is usually symptomatic

as in some viral infections, corticosteroid therapy is gen-

rather than curative.

erally totally contraindicated.

(5)

Cessation of therapy abruptly after a pro-

Corticosteroids have been used successfully in treat-

longed course of treatment may reveal adrenal

ment of bovine iritis, or 'silage eye', a condition that is

insufficiency.

*thought to be associated with infection with *Listeria**

monocytogenes (see p. 923) and involves the patho-

physiological change iridocyclitis. Corticosteroids are

Contraindications

most frequently administered by subconjunctival

(1)

Glucocorticoids are insulin antagonists and may administration, either alone or in conjunction with therefore exacerbate or precipitate diabetes mellitus (see p. 930). They should not generally be antibiotic injections.

There are a number of musculoskeletal conditions for

used in diabetic animals.

which steroids may be indicated, including traumatic

(2)

Steroids should be used with extreme caution in

arthritis, osteoarthritis (see p. 453), myositis (see p. 451), acute infections because of their immunosuppressive-eosinophilic myositis, tendonitis (see p. 452) and bursitis-

sant actions and, if used, this should be in combination with bactericidal antibiotics.

through their analgesic and anti-inflammatory actions.

(3)

Steroids are generally contraindicated in viral

However, they may suppress natural repair mechanisms infections, in late pregnancy due to the risk that and repeated injections or long-acting formulations they may induce parturition and in subjects with may produce degenerative changes in cartilage (steroid corneal ulcers.

arthropathy). For this reason, glucocorticoid use in all (4)

Administration by intra-articular injection should forms of arthritis is increasingly controversial, and they be avoided when there is sepsis in or close to the are definitely contraindicated in septic arthritis. Simi-joint, in the presence of intra-articular fracture, larly, in patients with laminitis steroids have fewer and when the articular cartilage is damaged, when fewer advocates. If they are used, treatment should be extensive degenerative bony lesions are present, if limited to the initial 24-hour period after the appear-previous injections were ineffective and when ance of signs. Otherwise steroids may exacerbate the there is any likelihood that the treated joint will

condition.

be over-exercised.

A number of respiratory conditions (see Chapters 17, 20, 49), both allergic and non-allergic, have been treated with systemic steroids. They include acute respiratory

Therapy with anti-inflammatory drugs

distress syndrome in cattle and chronic obstructive pulmonary disease in horses. In these conditions steroids

Respiratory diseases (see Chapters 17, 20, 49)

are most useful when disease signs are severe; high doses of water-soluble formulations are administered. Acute and sometimes overwhelming life-threatening intravenously. Depot preparations of steroids have inflammatory conditions occur in cattle and it is possible that the use of anti-inflammatory drugs, especially disease 'sweet itch'. Other indications for steroid non-immunosuppressant agents, will reduce morbidity therapy are ulcerative colitis, cerebral oedema and and mortality in such cases. Acute calf pneumonias of autoimmune haemolytic anaemia.

viral or bacterial origin and those of mixed aetiology,

In view of the potentially serious side-effects of

for example, have been shown to respond to NSAID

steroids, guidelines for their use have been recom-

treatment. Although such treatment is symptomatic it

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can be effective in life-threatening pneumonias. Selman

A study comparing the effectiveness of carprofen to

and coworkers showed that flunixin reduced signi-

flunixin for respiratory disease showed that a single

ficantly lung consolidation scores in calves infected

dose of the former drug had similar beneficial clinical

experimentally with PI3 virus (Selman et al. , 1984).

effects to three doses of the latter drug when used as an

Flunixin also reduced lung lesions, post-mortem lung

adjunctive therapy to antimicrobial treatment (Balmer

weights (reflecting an anti-oedematous action) and

*et al. , 1997). Meloxicam has been licensed for use in clinical signs of the
disease in an experimental bovine*

dogs for medium to long term therapy of muscu-

pasteurellosis model based upon challenge with

loskeletal pain and inflammation and is available as a Mannheimia haemolytica A1. However, the benefits of suspension and in injectable form. It is available as flunixin therapy were not apparent when the drug was an anti-inflammatory drug for use in the therapy of calf used alone but when used in combination with oxytetracycline pneumonia. Further evaluation of NSAIDs in acute racycline (Selman, 1988). The additional benefits pro-life-threatening respiratory infections is needed, since vided by the combination over oxytetracycline by itself some of the reports have reached conflicting conclusions. Nevertheless, carprofen, flunixin, ketoprofen, ratory rate and improved food intake.

tolfenamic acid and meloxicam all possess marketing

Anderson (1989) confirmed the benefits of a flunixin/oxytetracycline product in comparison with

oxytetracycline alone in field cases of pneumonia, with

Mastitis and endotoxaemia (see Chapters

treatment daily for three days. Reduction in coughing,

22, 23, 24, 30)

return to normal food intake and weight gain were

improved more with the combination product and there

Mastitis in cattle also benefits from NSAID treatment.

were fewer relapses. Since these early studies, other

The main indication is in acute and peracute cases

NSAIDs have been used in combination with antibi-

in which *Escherichia coli* or other endotoxin-releasing otics in calf pneumonia; they include ketoprofen,

bacteria are involved. Thus, within the udder, the endo-

meloxicam, carprofen and tolfenamic acid (Delaforge

toxin-induced increases in PGE₂ and TXA₂ concentra-

et al. , 1994). The precise way in which NSAIDs act in tions were significantly reduced by systemic flunixin

these subjects is unclear, but it seems likely that the

therapy (1.1 mg/kg at 8-hour intervals for seven doses)

anti-inflammatory action (reduced pulmonary oedema)

(Anderson et al. , 1986a) and clinical signs of both udder improves pulmonary function and respiratory gas

inflammation and depression as well as pyrexia were

exchange, while the antipyretic action may improve

suppressed (Anderson et al. , 1986b). The enhanced

the clinical status to the point where animals wish to eat phagocytosis of *Staphylococcus* spp., which occurs in and drink. In this way nutritional and fluid requirements will be maintained. Species such as cattle, which quarters, was not affected by flunixin treatment. rely principally on respiration for temperature regulation. However, flunixin did suppress significantly the tion, may be under particular stress from infections increased whey IgG1 and IgM concentrations produced causing pyrexia, thus the antipyretic actions of NSAIDs by endotoxin inoculation (Anderson et al. , 1986c). The may be particularly useful in such species. NSAIDs are local effects of systemically administered flunixin may also potent analgesics (against inflammatory pain) and seem surprising when the high degree of binding to when pain is a significant element in cattle pneumonias, plasma protein and its acidic nature are considered; this analgesic action of flunixin is likely to contribute to these properties should severely limit penetration the improved clinical status.

across the blood–milk barrier. However, in the acutely inflamed udder blood flow is increased and milk pH will be much closer to plasma pH than in non-mastitic milk and pulmonary lesions (congestion, oedema, interstitial emphysema) that are indistinguishable from naturally occurring acute bovine pulmonary emphysema (fog fever). Intoxication with 3MI is accepted as a valid model of the natural disease. Selman and co-workers have shown that parenteral flunixin (2.2 mg/kg) cascade with release of PGE₂ and TXA₂. Clinically, markedly reduces respiratory rate and the extent of lung lesions assessed by lung weights and both patho- Anderson et al. (1986b) also demonstrated improvement, including reduction in rectal temperature, in mas-

*logical and histopathological examinations. Flunixin
titic cows with coliform mastitis.*

*resulted in a prompt return to normal demeanour and
Flunixin in combination with oxytetracycline has also
respiratory rate and it reduced the degree and severity
been evaluated in cases of peracute mastitis in com-
of alveolar epithelial hyperplasia.*

parison with the antibiotic alone and clinical appraisal

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*suggested that the combination product gave better
provide analgesia in inflammatory conditions in cattle,
results (Christie, 1988). Similarly, ketoprofen was
although further work is required to establish optimal
shown to significantly improve recovery in naturally-
and economical treatment regimens.*

occurring clinical mastitis in dairy cows when used in

There have been few attempts to administer NSAIDs

combination with sulphadiazine and trimethoprim

locally by intramammary infusion to establish whether

*(Shpigel et al. , 1994). Carprofen has also been evaluated their analgesic and
anti-inflammatory effects are of*

in cows with endotoxin-induced mastitis, at a dose rate value in providing symptomatic relief in any form of of 0.7 mg/kg. Clearance of the drug was slower, elimination half-life longer and penetration into milk greater than the irritant properties of many NSAIDs. One NSAID, ibuprofen, has been administered locally into udders of mastitic animals carprofen reduced heart rate, rectal temperature and quarter swelling significantly (Lohuis et al. , 1991), although at the time of writing no licensed control cows and steroid-treated cows (DeGraves & product containing carprofen is available for use in Anderson 1991), but no licensed product is currently lactating cows in the UK. Similarly, flunixin showed available in the UK. A specially prepared, non-antipyretic, anti-inflammatory effects and reduced clinical depression following experimental intramammary

*via the intramammary route in the study by Fitzpatrick
infusion with endotoxin.*

*et al. (1998), described above. As expected, the somatic Mastitis is an
inflammatory disease associated with*

cell count increased on infusion of the compound, but

pain (Chapter 23). Indeed bradykinin, a hyperalgesic

there was no evidence of an excessive induced inflam-

mediator, has been detected in milk from cases of clin-

matory response from observation of clinical signs or

*ical and subclinical mastitis in cows (Eshraghi et al., from milk yield subsequent
to the clinical episode.*

1999). Kinin peptides are released during inflammation

Interestingly, abrogation of the allodynia resulting from

and are among the most potent mediators of vasodila-

the use of intravenous flunixin was not seen when the

tion, oedema and pain. Bradykinin was detected in milk

intramammary route was employed, indicating the pos-

from cows with clinical and subclinical disease and the

sibility that the drug had a central effect on pain pro-

concentrations of bradykinin were shown to correlate

cessing, probably at the level of the spinal cord.

with the severity of the disease. Bradykinin concentra-

Steroids with anti-inflammatory actions, such as prednisolone, have been used extensively for the therapy of somatic cell counts and from which Staphylococcus aureus was isolated compared to high cell count quarters in a number of products containing also one or more antibiotics and used to treat clinical cases during systemically administered NSAIDs are beneficial in lactation. The popularity of such products is indicated by volume usage; in the UK at the present time the be classified as acute or peracute has received little three most widely used lactating-cow intramammary attention to date. However, Pyorala and co-workers products all contain an anti-inflammatory steroid. The (1988) examined the influence of phenylbutazone value of steroids in such products is unclear and in view (10 mg/kg) and flunixin (2.2 mg/kg) administered of their potential immunosuppressant actions such

intravenously in cases of chronic subclinical mastitis. usage has been questioned. One suggestion has been Neither drug influenced bacterial growth, somatic cell that the main value of steroids is not to influence the count or the levels of inflammatory markers (N-acetyl-disease, but merely to suppress the irritancy that b-d-glucosaminidase activity and trypsin-inhibitory inevitably accompanies intramammary infusions and capacity). Eckersall et al. (2001) showed that dairy cows which varies in degree with the product formulation/ with naturally-occurring clinical mastitis of mild or moderate grades, assessed by farmer observation, had significantly different concentrations of the acute phase proteins serum amyloid A and haptoglobin in serum by E. coli endotoxin, Streptococcus uberis and Staphylococcus aureus (Bywater et al. , 1988). Signs of inflammation (swelling, milk consistency) were reduced, but

5 days and 40 days in the case of mild and moderate leucocyte infiltration was not impaired. Further studies cases of mastitis, respectively (Fitzpatrick et al., 1998). in this field, for example to establish that the phagocytic The allodynia was shown to be abrogated by a single capacity of infiltrating neutrophils and immune protective mechanisms are not impaired, would provide confirmed for 24 hours, after which the allodynic state resumed. This indicates the potential use of NSAIDs to mastitis. In acute and peracute mastitis the slow onset

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of action of steroids is likely to limit their value, but in aspirin and the actions of these drugs in acute interstitial pneumonia has been described above.

benefit was obtained when treatment was administered 16–18 hours after challenge (Bywater, pers. comm.).

Arthritis and joint diseases (p. 453)

In summary, it seems at least possible that the anti-

endotoxaemic effects of systemically administered

In contrast to the horse and dog, long-term therapy of

NSAIDs may be of value (together with other sup-

joint diseases in cattle is not commonly undertaken.

portive therapy, including fluids and systemic antibiotic

This in part relates to economic factors, in part to drug

administration) in cases of acute and peracute mastitis,

and drug metabolite residue problems in milk and

whereas reduced swelling and pain provided by intra-

edible tissues and in part to the lack of knowledge of

mammary infusion of steroid/antibiotic products may

drug pharmacokinetics linked to dosage regimens that

assist recovery in less severe clinical mastitis. The latter

are known to be effective and safe. However, some joint

proposal is compatible with the suggestion of Sandholm

diseases of cattle have been treated with phenylbuta-

and co-workers (1990) that inflammatory changes in the

zone, corticosteroids or NSAIDs but there is, as stated,

mastitic gland seem to improve bacterial nutrition and

an urgent need to establish rational dosage schedules.

growth, with potential adverse effects on the host.

Whether the chondroprotective class of agents, which

However, further studies are needed to examine these

includes polysulphatedglycosaminoglycans and pen-

proposals concerning both steroids and NSAIDs.

tosan sulphate, is likely to be used in the future is not

Steroids have also been used, usually systemically, to

clear. Some increase in therapy in this field can be antic-

counteract the life-threatening symptoms of endotoxic

ipated, should the economic situation in the farming

shock in cattle, commonly together with large volumes

industry improve, as joint disease is relatively common

of fluids. In addition to their anti-endotoxaemic effects,

and can affect individual valuable animals, where treat-

steroids promote gluconeogenesis and this may con-

ment may be justified.

tribute to the therapeutic response. The disadvantages

of steroids are the large doses required and conse-

Foot-and-mouth disease (Chapter 43(c))

quential high cost of treatment and the latent period

prior to onset of action. This can be minimized by the

This contagious viral disease is characterized by fever

use of water-soluble steroids.

and vesicular eruptions in the mouth and in the inter-digital area of the feet. The clinical presentation varies but a proportion of animals suffer severe ulceration

Metritis (pp. 520, 521)

with loss of large areas of oral epithelial tissue and

While some studies showed no beneficial effect of under-running of hoof structures. The use of NSAIDs

NSAIDs in the treatment of reproductive disorders

might be of value as adjunctive therapy and one agent,

such as retained placenta and post-partal endometritis

flunixin, has been studied. Artificially infected cattle

(Konigsson et al. , 2001), flunixin was shown to reduce treated with flunixin showed clinical improvement, indi-the incidence of pyrexia in cows with either acute and

cated by lowered body temperature and increased

subacute metritis compared to non-treated controls

weight gain, whilst antibody titres were not affected. In

(Amiridis et al. , 2001). Uterine involution and onset light of the recent epidemic of foot-and-mouth disease

of oestrus occurred earlier in cows that received

(FMD) in the UK (2001) and the considerable concerns

flunixin.

for the welfare of affected animals prior to slaughter, the role of NSAIDs in palliative therapy could be evaluated and implemented, to allow improved pain

Calf scours (Chapter 14)

management, should future outbreaks of FMD occur.

Early studies by Jones et al. (1977) indicated a possible benefit of flunixin in suppressing inflammation, reduc-

Pain

ing fluid losses in faeces and suppressing morbidity and mortality in calf scours. Until recently, no drugs were

Calves that received ketoprofen before and after available with licensed indications for such use.

dehorning following a sedative and a local anaesthetic

However, meloxicam is now licensed for use in calf

showed reduced behavioural signs thought to be associated with pain, such as head shaking and ear flicking, compared to untreated controls (Faulkner & Weary, 2000). Different methods of castration such as use of

scours in combination with anti-microbial drugs.

ciated with pain, such as head shaking and ear flicking,

compared to untreated controls (Faulkner & Weary, 2000). Different methods of castration such as use of

Hypersensitivity reactions (p. 927)

rubber rings, bloodless castrators or surgical methods

Anaphylaxis can be inhibited both in vivo and in vitro were shown to result in different levels of pain

in cattle by the NSAIDs sodium meclofenamate and

response, demonstrated by altered behaviour (Robert-

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son et al. , 1994). This indicates that there may be the with amoxycillin and clavulanic acid. In Proceedings of the potential to use NSAIDs either pre- or post-procedure

15th World Buiatric Congress, Spain (abstract).

to reduce or abrogate pain caused by castration.

Cheng, Z., McKellar, Q.A. & Nolan, A. (1998) Pharmacokinetic studies of flunixin meglumine and phenylbutazone in plasma, exudate and transudate in sheep. Journal of Veteri-

Conclusions

nary Pharmacology and Therapeutics, 21, 315–21.

Cheng, Z., Nolan, A. & McKellar, Q. (2002) Anti-inflamma-

It seems likely that both steroids and, in particular,

tory effects of carprofen, carprofen enantiomers and Ng-

NSAIDs, judiciously used, may be of value in a number

nitro-L-arginine methyl ester in vivo using sheep as a

of cattle diseases in which inflammation, endotoxaemia

model. *American Journal of Veterinary Research*, **63**, 782–8.

and/or pain are major components. Further work is

Christie, H. (1988) The veterinary uses of a non-steroidal anti-needed if such prospects are to be realized and espe-

inflammatory agent: flunixin meglumine. *British Veterinary cially if licensed pharmacological products that will*

Journal, Suppl. No. 1, **8**.

improve animal health and welfare are to result from

Coderre, T.J. & Melzack, R. (1987). Cutaneous hyperalgesia: these studies.

contributions of the peripheral and central nervous systems

to the increase in pain sensitivity after injury. *Brain*

Research, **404**, 95–106.

Davies, P., Bailey, P.J. & Goldenberg, M. (1984) The role of **References**

arachidonic acid oxygenation products in pain and inflam-

mation. *Annual Review Immunological*, **2**, 235–57.

Amiridis, G.S., Leontides, L., Tassos, E., Kostoulas, P. & DeGraves, F.J. & Anderson, K.L. (1991) Ibuprofen treatment Fthenakis, G.C. (2001) Flunixin meglumine accelerates

of endotoxin-induced coliform mastitis. In *Proceedings of*

uterine involution and shortens the calving-to-first-oestrus

the International Congress of Mastitis, Ghent, Belgium.

interval in cows with puerperal metritis. *Journal of Veteri-Delaforge, J., Thomas, E., Davot, J.L. & Boisrame, B. (1994) A nary Pharmacology and Therapeutics*, **24**, 365–7.

field evaluation of the efficacy of tolfenamic acid and oxy-

Anderson, D.B. (1989) Utilisation de la flunixin dans les mal-tetracycline in the treatment of bovine respiratory disease.

adies respiratoires. L'Action Veterinaire. (Suppl.), 1092, Journal of Veterinary Pharmacology and Therapeutics, 17, 22–7.

43–7.

Anderson, K.L., Kindal, H., Smith, A.R., Davis, L.E. &

Delatour, P., Foot, R., Foster, A.P., Baggot, D. & Lees, P. (1996) Gustafsson, B.K. (1986a) Endotoxin-induced bovine masti-Pharmacodynamics and chiral pharmacokinetics of carpro-

tis: arachidonic acid metabolites in milk and plasma and

fen in calves. British Veterinary Journal, 152, 183–98.

effect of flunixin meglumine. American Journal of Veteri-

Dolan, S. & Nolan, A.M. (1999) N-methyl d-aspartate induced nary Research, 47, 1373–7.

mechanical allodynia is blocked by nitric oxide synthase and

Anderson, K.L., Smith, A.R., Shanks, R.D., Davis, L.E. &

cyclooxygenase-2 inhibitors. Neuro Report, 10, 449–52.

Gustafsson, B.K. (1986b) Efficacy of flunixin meglumine for

Dolan, S. & Nolan, A.M. (2000) Behavioural evidence sup-

the treatment of endotoxin-induced bovine mastitis. Amer-

porting a differential role for group I and II metabotropic

ican Journal of Veterinary Research, 47, 1366–72.

glutamate receptors in spinal nociceptive transmission.

Anderson, K.L., Smith, A.R., Shanks, R.D., Whitmore, H.L.,

Neuropharmacology, 39, 1132–8.

Davis, L.E. & Gustafsson, B.K. (1986c) Endotoxin-induced

Eckersall, P.D., Young, F.J., McComb, C., Hogarth, C., Safi, S., bovine mastitis: immunoglobulins, phagocytosis and effect

Weber, A., McDonald, T., Nolan, A.M. & Fitzpatrick, J.L.

of flunixin meglumine. American Journal of Veterinary

(2001) Acute phase protein in serum and milk from dairy

Research, 47, 2405–10.

cows with clinical mastitis. Veterinary Record, 148, 35–41.

Arifah, A.K. & Lees, P. (2002) Pharmacodynamics and phar-

Eltom, S.E., Guard, C.L. & Schwark, W.S. (1993) The effect of pharmacokinetics of phenylbutazone in calves. Journal of

age on phenylbutazone pharmacokinetics, metabolism and

Veterinary Pharmacology and Therapeutics, 25, 299–309.

plasma protein binding in goats. Journal of Veterinary Pharmacology and Therapeutics, 25, 299–309.

(2002) Effects of carprofen (R and S enantiomers) on the production of IL-1, IL-6 and

tiomers and racemate) on the production of IL-1, IL-6 and

Eshraghi, H.R., Zeitlin, I.J., Fitzpatrick, J.L., Ternent, H. & TNF-alpha by equine chondrocytes and synoviocytes.

Logue, D. (1999) The release of bradykinin in bovine

Journal of Veterinary Pharmacology and Therapeutics, **25**, mastitis. *Life Science*, **64**, 1675–87.

145–53

Faulkner, P.M. & Weary, D.M. (2000) Reducing pain after

Balmer, T.V., Williams, P. & Selman, I.E. (1997) Comparison dehorning in dairy calves. *Journal of Dairy Science*, **83**, of carprofen and flunixin meglumine as adjunctive therapy

2037–41.

in bovine respiratory disease. *The Veterinary Journal*, **154**, Ferreira, S.H. (1983) Prostaglandins: peripheral and central

233–41.

analgesia. In *Advances in Pain Research and Therapy* (ed.

Brideau, C., Van Staden, C. & Chan, C.C. (2001) *In vitro*

by J.L. Bonica). Raven Press, New York.

effects of cyclooxygenase inhibitors in whole blood of

Fitzpatrick, J.L., Young, F.J., Eckersall, D., Logue, D.N., Knight, horses, dogs, and cats. *American Journal of Veterinary*

C.J. & Nolan, A.M. (1998) Recognising and controlling pain *Research*, **62**, 1755–60.

and inflammation in mastitis. In *Proceedings of the 11th*

Bywater, R.J., Godinho, K. & Buswell, J.F. (1988) *Effects of British Mastitis Conference*, pp. 36–44. Stoneleigh.

prednisolone on experimentally induced mastitis treated

Galbraith, E.A. & McKellar, Q.A. (1996) Protein binding and

Inflammation and Pain • 1065

in vitro serum thromboxane B₂ inhibition by flunixin meglumine

Biochemical and haematological effects of recommended

dosage with phenylbutazone in horses. *Equine Veterinary*

Research Veterinary Science, **61**, 78–81.

Journal, **15**, 158–67.

Gerring, E.L., Lees, P. & Taylor, J.B. (1981) Pharmacokinetics of phenylbutazone and its metabolites in the horse. *Equine Evaluation of carprofen in calves using a tissue cage model*

Veterinary Journal, **73**, 152–7.

of inflammation. *British Veterinary Journal*, **152**, 199–

Guerrini, L., Blasi, F. & Denisdonini, S. (1995) Synaptic activation of NF- κ B by glutamate in cerebellar granule

Lees, P., Ewins, C.P., Taylor, J.B.O. & Sedgwick, A.D. (1987) *neurons in vitro. Proceedings of the National Academy of Sciences USA* Serum thromboxane in the horse and its inhibition by

Science USA, **92**, 9077–81.

aspirin, phenylbutazone and flunixin. *British Veterinary*

Higgs, G.A., Flower, R.J. & Vane, J.R. (1979) A new approach *Journal*, **143**, 462–76.

to anti-inflammatory drugs. *Biochemical Pharmacology*, **28**, Lees, P. & Higgins, A.J. (1985) *Clinical pharmacology and therapeutics* 1959–61.

apeutic uses of non-steroidal anti-inflammatory drugs in the

Higgs, G.A., Moncada, S. & Vane, J.R. (1980) The mode of

horse. Equine Veterinary Journal, 17, 83–96.

action of anti-inflammatory drugs which prevent the perox-

*Lees, P., McKellar, Q.A., Foot, R. & Gettinby, G. (1998) Phar-ication of
arachidonic acid. Clinics in Rheumatic Diseases, macodynamics and
pharmacokinetics of tolfenamic acid in*

6, 675–93.

ruminating calves: evaluation in models of acute inflamma-

Higgs, G.A., Palmer, R.M.J., Eakins, K.E. & Moncada, S.

tion. The Veterinary Journal, 155, 275–88.

(1981) Arachidonic acid metabolism as a source of inflam-

*Lees, P., Maitho, T.E. & Taylor, J.B. (1985) Pharmacokinetics matory mediators
and its inhibition as a mechanism of*

of phenylbutazone in young and old ponies. Veterinary

action of anti-inflammatory drugs. Molecular Aspects of

Record, 116, 229–32.

Medicine, 4, 275–301.

*Lees, P., May, S.A. & McKellar, Q.A. (1991) Pharmacology and Jones, E.W.,
Hamm, D., Cooley, L. & Bush, L. (1977) Diar-therapeutics of non-steroidal anti-
inflammatory drugs in the*

rhoeal diseases of the calf: observations on treatment and

dog and cat: 1 General pharmacology. Journal of Small

prevention. *New Zealand Veterinary Journal*, **25**, 312–16.

Animal Practice, **32**, 183–93.

Keen, P.M. (1987) Uses and abuses of corticosteroids. In The Ley, S.J., Livingston, A. & Waterman, A.E. (1995) A field study *Veterinary Annual*, 27th issue (ed. by C.S.G. Grunsell, of the effects of lameness on mechanical nociceptive

F.W.G. Hill & M.E. Raw). pp. 45–62. Sciotechnica, Bristol.

thresholds in sheep. *Veterinary Record*, **137**, 85–7.

Konigsson, K., Gustafsson, H., Gunnarsson, A. & Kindahl, H.

Lohuis, J.A., van Werven, T., Brand, A., van Miert, A.S.,

(2001) Clinical and bacteriological aspects on the use of

Rohde, E., Ludwig, B., Heizmann, P. & Rehm, W.F. (1991)

oxytetracycline and flunixin in primiparous cows with

Pharmacodynamics and pharmacokinetics of carprofen, a

induced retained placenta and post-partum endometritis.

non-steroidal anti-inflammatory drug, in healthy cows and

Reproduction in Domestic Animals, **36**, 247–56.

cows with *Escherichia coli* endotoxin-induced mastitis.

Kopp, E. & Ghosh, S. (1994) Inhibition of NF-kappa B by

Journal of Veterinary Pharmacology and Therapeutics, **14**, sodium salicylate and aspirin. *Science*, **265**, 956–59.

219–29.

Landoni, M.F., Cunningham, F.M. & Lees, P. (1995a) Com-

McKellar, Q.A., May, S.A. & Lees, P. (1991) Pharmacology and parative pharmacodynamics of flunixin, ketoprofen and

therapeutics of non-steroidal anti-inflammatory drugs in the

tolfenamic acid in calves. *Veterinary Record*, **137**, 428–31.

dog and cat: 2 Individual agents. *Journal of Small Animal*

Landoni, M.F., Cunningham, F.M. & Lees, P. (1995b) Deter-

Practice, **32**, 225–35.

mination of pharmacokinetics and pharmacodynamics of

Maitho, T.E., Lees, P. & Taylor, J.B. (1986) Absorption and flunixin in calves by use of pharmacokinetic/pharmacody-pharmacokinetics of phenylbutazone in Welsh Mountain

namic modeling. *American Journal of Veterinary Research*, ponies. *Journal of Veterinary Pharmacology and Therapeu-*56, 786–94.

tics, **9**, 26–39.

Landoni, M.F., Cunningham, F.M. & Lees, P. (1996) Pharma-

Marriner, S. & Bogan, J.A. (1979) The influence of the rumen cokinetics and pharmacodynamics of tolfenamic acid in

on the absorption of drugs; study using meclofenamic acid

calves. *Research in Veterinary Science*, **61**, 26–32.

administered by various routes to sheep and cattle. *Journal Lane, P., Lees, P. & Fink, J. (1990) Action of dexamethasone of Veterinary of Pharmacology and Therapeutics*, **2**, 109–

in an equine model of acute non-immune inflammation.

15.

Research in Veterinary Science, **48**, 87–95.

Nolan, A.M. (2000) *Patterns and its management of pain in*

Lascelles, B.D.X., Cripps, P.J., Jones, A. & Waterman, A.E.

animals. In Pain: Its Nature and Management in Man and

(1997) Post-operative central hypersensitivity and pain: the

Animals. International Congress and Symposium Series, (ed.

pre-emptive value of pethidine for ovariohysterectomy.

by Lord Soulsby & D. Morton), vol. 246, pp. 93–100. Royal

Pain, **73**, 461–71.

Society of Medicine Press.

Lees, P. Ayliffe, T., Maitho, T.E. & Taylor J.B.O. (1988)

*Pyorala, S., Patila, J. & Sandholm, M. (1988) Phenylbutazone
Pharmacokinetics, metabolism and excretion of phenylbu-and flunixin
meglumine fail to show beneficial effects on*

tazone in cattle following intravenous, intramuscular and

bovine subclinical mastitis. Acta Veterinaria Scandinavica, oral administration.

Research in Veterinary Science, **44**, **29**, 501–503.

57–67.

Rainsford, K.D. (1977) The comparative gastric ulcerogenic

*Lees, P., Creed, R.F.S., Gerring, E.L., Gould, P.W., Humphreys, activities of non-
steroid anti-inflammatory drugs. Agents*

D.J., Maitho, T.E., Michell, A.R. & Taylor, J.B. (1983)

and Actions, **7**, 573–7.

Robertson, I.S., Kent, J.E. & Molony, V. (1994) Effects of treated dogs. *Journal American Veterinary Medical Association*-different methods of castration on behaviour and plasma

tion, **188**, 145–50.

cortisol in calves of three ages. *Research in Veterinary*

Toutain, P.L., Brandon, R.A., Alvinerie, M., Garcia-Villar, R.

Science, **56**, 8–17.

& Ruckebusch, Y. (1982) Dexamethasone in cattle: phar-

Sandholm, M., Kaartinen, L. & Pyorala, S. (1990) Bovine

macokinetics and action on the adrenal gland. *Journal of*

mastitis – why does antibiotic therapy not always work? *An*

Veterinary Pharmacology and Therapeutics, **5**, 33–43.

overview. *Journal of Veterinary Pharmacology and Thera-*

Vane, J.R. (1971) Inhibition of prostaglandin synthesis as a

peutics, **13**, 248–60.

mechanism of action for aspirin-like drugs. *Nature*, **231**, Scheuren, N., Bang, H., Munster, T., Brune, K. & Pahl, A.

271–3.

(1998) Modulation of transcription factor NF-kappa B by

Wallace, J.L. (1999) Clinical significance and potential of selec-enantiomers of the nonsteroidal anti-inflammatory drug

tive COX-2 inhibitors. *Trends in Pharmacological Sciences*, ibuprofen. *British*

Journal of Pharmacology, **123**, 645–52.

20, 389–90.

Selman, I.E. (1988) *The veterinary uses of a non-steroidal*

Welsh, E.M. & Nolan, A.M. (1994) *Repeated intradermal*

anti-inflammatory agent: flunixin meglumine. The British

injection of low dose carrageenan induces tachyphylaxis to

Veterinary Journal, Suppl. No. 1, 4–6.

evoked hyperalgesia. Pain, **59**, 415–21.

Selman, I.E., Allan, E.M., Gibbs, M.A., Wiseman, A. & Young, Whay, H.R.,
Waterman, A.E., Webster, A.J.F. & O'Brien, J.K.

W.B. (1984) *Effect of anti-prostaglandin therapy in experi-*

(1998) *The influence of lesion type on the duration of hyper-*

mental parainfluenza type 3 pneumonia in weaned conven-

algesia associated with hindlimb lameness in dairy cattle.

tional calves. Veterinary Record, **115**, 101–105.

The Veterinary Journal, **156**, 23–9.

Shpigel, N.Y., Chen, R., Winkler, M., Saran, A., Ziv, G. & Williams, T.J. (1983)
Prostaglandin E2, prostaglandin I and the Longo, F. (1994) Anti-inflammatory
ketoprofen in the treat-vascular changes of inflammation. British Journal
Pharma-

ment of field cases of bovine mastitis. Research in Veterinary cology, **65**, 577–
24.

Science, **56**, 62–8.

Willoughby, D.A. (1968) *Effects of prostaglandins F2a and Toombs, J.P., Collins, L.G., Graves, G.M., Crowe, D.T. & PGE1 on vascular permeability. Journal of Pathological*
Caywood, D.D. (1986) *Colonic perforation in corticosteroid Bacteriology*, **96**, 387–87.

Chapter 63

Growth Promoters in Cattle

K. Lawrence

Introduction

1067

use in human medicine, nor do they share a mode

In-feed additives

1067

of action with any compound in human medicine.

Rumen

1067

Interestingly, in addition, the ionophores do not have

Intestine

1069

antibiotic resistance encoded by transferable genes.

Hormonal implants

1071

Monensin acts on bacteria by facilitating the carriage of

Growth hormone (BST)

1073

sodium ions in to the cell. This causes the cell to speed

Repartitioning agents

1073

up the sodium/potassium pump in the cell membrane

to redress the ion balance. As the transport mechanism

requires energy in the form of ATP, continuous expo-

Introduction

sure to monensin could lead the cell to exhaust energy

supplies, resulting in death by osmotic disruption of

Bovine growth promoters are divided into four groups:

the cell, but more usually it prevents the bacteria from

- *In-feed additives*

competing in a mixed population and numbers decline

- *Hormonal implants*

(Pinkerton & Steinrauf, 1970; Pressman, 1976).

- *Growth hormone (somatotropins)*

The mode of action of monensin in the rumen has

- *Repartitioning agents (b-agonists)*

been elucidated and it can act as model for other products in this class, such as lasalocid (Bovatec, Alpharma

In the European Union (EU) the use of hormonal Animal Health Division) and laidlomycin (Cattlyst, implants and b-agonists has been banned since 1988, Alpharma Animal Health Division).

followed in 1990 with a moratorium on bovine soma-

The rumen is a fermentation vat that converts totropin (BST) that became a ban in 2000. Meanwhile carbohydrates into volatile fatty acids (VFAs) such the use of in-feed additives is under close scrutiny with as acetic, butyric and propionic acids that drive the a proposed 'phase out' by 2006. The EU position on animal's metabolism (see Fig. 63.1). While all three growth promoters has been taken in spite of the VFAs can be used in the nutrition of the bovine, the increased production costs to farmers and the enormous knock-on costs of increased feed usage and slurry leading to an improved efficiency of usage of both com-

production leading to environmental pollution (Verbeke & Viaene, 1996). The key to the efficiency of production of the VFAs is the preservation of the six carbons from the final fermentation substrate, usually a hexose sugar such as glucose. With acetic acid only four of the carbon atoms are retained in the rumen, with two being lost as the gases carbon dioxide and methane;

In-feed additives

butyric acid is similar although only carbon dioxide is produced. Only with propionic acid is there a complete retention of all six carbon molecules in the fermentation products.

In-feed additives are divided into two types by the site of primary activity: rumen and intestine.

Richardson et al. (1976) provided the first evidence of the effects of monensin on VFA production using anaerobic batch fermentation. In this study the propionic acid

Rumen

The most widely researched of the rumen active growth levels rose from 7.4 mM/ml to 12.5 mM/ml as the concentration of monensin was increased (Table 63.1).

Animal Health). Monensin is an 'ionophore', a group

Similar results were found in vivo by Van Maanen et al. (1978) in steers being fed both roughage and grain based rations – there

al. (1978) in steers being fed both roughage and grain based rations – there was a significant increase in the

based rations – there was a significant increase in the

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GLUCOSE

Table 63.2

The effect of monensin on rumen propionate pool

size and production rate in steers consuming roughage or grain based diets.

Treatment

Propionate Propionate

pool size (g)

production rate

ACETATE

PROPIONATE

BUTYRATE

(g/day)

Roughage

32.0 ± 1.3

441 ± 24

CO₂

CO₂

Roughage + monensin

56.5 ± 2.1

659 ± 22

Grain

36.5 ± 2.0

510 ± 10

CO

Grain + monensin

66.3 ± 4.1

899 ± 45

2

CH₃

Fig. 63.1

Rumen fermentation pattern for a simple carbohy-

drate, showing the preservation of carbon molecules.

Table 63.3

The sensitivity of rumen bacteria to monensin and

*their related products of fermentation. (After Chen & Russell **Table 63.1***

The shift of VFA production ratios with increasing

*(1989); Chow & Russell (1990); Dawson & Boling (1983); Dennis levels of
monensin, during anaerobic batch fermentation.*

et al. (1981).)

Monensin

Acetic

Propionic

Butyric

Products of fermentation

(mcg/ml)

(mM/ml)

(mM/ml)

(mM/ml)

Monensin-sensitive organisms

0.0

19.6

7.4

7.1

Ruminococcus

Acetic acid

0.10

20.8

8.6

7.2

Methanobacterium

Acetic acid, methane

0.25

20.2

9.4

6.9

Lactobacillus

Lactic acid

0.50

19.6

10.6

6.5

Butyrivibrio

Acetic acid, butyric acid

1.00

19.1

11.1

6.3

Streptococcus

Lactic acid

5.00

17.5

11.2

5.8

Methanosarcina

Methane

25.00

16.7

12.5

5.6

Monensin-insensitive organisms

Selenomonas

Propionic acid

Bacteroides

Acetic acid, propionic acid

size of the propionate pool and the production rate. The

Veillonella

Propionic acid

results are summarized in Table 63.2.

The effect of monensin seems to be mediated by

changes in the balance of the bacterial population in the

Table 63.4

The effect of monensin on rumen protozoa and pro-

rumen. The sensitivity of a range of rumen bacteria to

propionic acid production in fistulated sheep.

monensin is reviewed in Table 63.3, along with their

fermentation output. Monensin clearly inhibits the

Monensin feed

Total protozoa

Molar % propionic

major acetate- and butyrate-producing bacteria, allow-

inclusion (mg/kg)

(¥105/ml)

acid concentration

ing a proliferation of the primary propionate producers.

Monensin has also been observed to reduce the con-

0

10.1

20.2

centration of rumen protozoa in many experiments.

11

9.0

23.6

This has also been associated with an increase in the

22

6.2

24.4

proportion of propionic acid production, as is shown in

33

7.1

25.8

the sheep study presented in Table 63.4.

44

5.4

28.7

Goodrich et al. (1984) and Potter et al. (1986) have reviewed the expected performance benefits from the

use of monensin and highlighted the different nature but the ADF is markedly reduced. This contrasts with of the response in grain-based and roughage-based the roughage diets where the ADG is increased while systems. In all performance trials two measurements the ADF is unchanged. In balanced rations composing are made to derive a third. The feed intake (ADF, both grain and forage the effects on ADF and ADG are average daily feed in kg) and the growth rate (ADG, less clear, but there is still an 8 to 10 per cent improve-average daily gain in kg) are measured and the feed ment in FCR.

conversion ratio (FCR) is calculated by dividing There are additional benefits from the rumen active ADF/ADG. While the FCR is improved in all the trials, growth promoters in the control of acidosis (Nagaraja in grain-based diets the ADG often remains unchanged et al. 1985, 1987), bloat control (Sakauchi & Hoshino,

Growth Promoters in Cattle • 1069

1981; Bartley et al., 1983; Lowe et al., 1991) and the pre-animals matched that of the germ-free group; there

vention of coccidiosis (Watkins et al., 1986).

was no effect of the antibiotic on the germ-free group.

Antibiotics within the digestive tract of animals inhibit the growth and modify the metabolism of susceptible

Intestine

populations of bacterial flora. Since most of the anti-

The growth promoters with a more pronounced effect

biotics used for growth promotion have a Gram-

in the intestine are the bambermycins (Flavomycin,

positive spectrum of activity, such bacteria as Clostridia, Intervet) and virginiamycin (Stafac, Phibro Animal

Enterococci and Bacilli seem likely candidates. The Health). This does not imply that these products have

inclusion of growth promoters in cattle feeds serves to

no effect in the rumen and indeed with virginiamycin

allow a conventionally raised animal to grow at the

there have been reports of significant effects on VFA

same rate as a germ-free animal.

production and the control of acidosis (Hedde et al.,

Studies on the physiological, nutritional, metabolic 1980).

and immunological effects associated with the use of

Likewise monensin is described as a rumen active antibiotic feed additives demonstrate the complexity of growth promoter, but this does not imply it has no effect their mode of action (Vissek, 1979; Hays, 1979; Coates, 1980; Anderson et al., 2000). The reported effects be an effective growth promoter in pigs it must also include:

have a more conventional mode of action in this species

- *Inhibition of the growth or modifying the metabolism of harmful gut bacteria;*

(Mackinnon, 1987).

The mode of action of in-feed growth promoters in

- *Decreased elaboration of toxic substances, including bacterial toxins;*

is complex and still open to debate. The comparison of

- *Reduced bacterial destruction of essential nutrients;*
 - *Increased synthesis of vitamins and other growth*
- healthy animals with a normal digestive tract bacterial flora to germ-free animals has produced an insight into*

factors;

the role of the gut flora in inhibiting nutrient absorption.

- Improved efficiency of nutrient absorption by

modification of the gut wall;

response of each group to antibiotic incorporated into

- Reduced intestinal mucosal epithelial cell turnover;

their diet (Vissek, 1979). Conventional animals grow

- Reduced intestinal motility.

more slowly and use their feed less efficiently than gnotobiotic or 'germ-free' animals because of the presence

The net effect of the changes in the intestine listed in

of the intestinal flora. When antibiotic was administered,

Table 63.5 is an increased availability and absorption of

nutrients and a reduced cost/kg gain.

tered, the growth response of the conventionally grown

nutrients and a reduced cost/kg gain.

Table 63.5

Some physiological, nutritional and metabolic effects ascribed to in-feed growth promoters (after Ewing & Cole, 1994).

Physiological effectsa

Nutritional effects

Metabolic effects

Gut food transit time

Energy retention

—

Ammonia production

、

Gut wall diameter

、

Gut energy loss

、

Toxic amine production

、

Gut wall length

、

Nitrogen retention

—

Alpha-toxin production

、

Gut wall weight

、

Limiting amino acid supply

—

Fatty acid oxidation

、

Gut absorptive capacity

—

Vitamin absorption

—

Faecal fat excretion

、

Faecal moisture

Vitamin synthesis

、

Liver protein synthesis

—

Mucosal cell turnover

、

Trace element absorption

—

Gut alkaline phosphatase

—

Stress

、
Fatty acid absorption

—

Gut ureasè

Feed intake

_, ` , 6

Glucose absorption

—

Calcium absorption

—

Plasma nutrients

—

à denotes a reduction; _ denotes an increase; 6 denotes no change.

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In spite of the different emphasis of the mode of

(1)

Selection of antibiotic resistance in the gut flora. In action of the rumen and gut-active in-feed growth pro-any population of bacteria, there is a range of antibiotic

motors, their effects on the efficiency of performance is

susceptibility from fully sensitive to solidly resistant –

very similar with a 6 to 8 percent improvement in FCR.

even in the absence of antibiotics (Linton, 1982).

Consumer concerns have been raised about the use

Usually, the bulk of the bacteria remain sensitive to

of in-feed growth promoters in all species. The spectrum

an antibiotic, unless a therapeutic dose is administered

of data on this aspect of these products submitted for

which kills the sensitive and allows the few resistant

registration was summarized by Lawrence (1998).

bacteria to proliferate; thus a resistant population

As with therapeutic veterinary medicines, stringent

develops (Evangelisti et al., 1975).

safety studies are necessary for growth promoters. The

It is a truism that the use of a therapeutic dose of an

safety data requirements for registration in the EU are

antibiotic selects for the survival of the few resistant

listed below (source: Official Journal of European Com-

organisms in the population; it does not cause indi-

munities L208-21 T025):

vidual sensitive bacteria to become resistant. As the

growth promoters are included at levels approximately

1.

Studies on target species

10 to 100 times lower than therapeutic doses (Corpet,

1.1

Safety studies

– Biological

1996), there is little, if any, selection pressure on the gut

– Toxicological

flora. A selection of studies on growth promoters that

– Macroscopic

have demonstrated their low resistance selection capac-

– Histological effects

ity at authorised use levels are listed below:

1.2

Microbiological studies of the additive

• Flavomycin

(Dealy & Moeller, 1977; Devriese,

1.2.1

Antibacterial spectrum of activity

1980; Mee, 1982; Corpet, 1984; Dutta & Devriese,

1.2.2

Cross-resistance to therapeutic

1984)

antibiotics

- *Monensin*

(Dutta & Devriese, 1984; Newbold

1.2.3

Tests to find out whether the additive is

et al., 1993)

capable of selecting resistance factors

- *Virginiamycin (Gaines et al., 1980; Devriese, 1980;*

1.2.4

Tests to determine the effect of the

Mee, 1982; Dutta & Devriese, 1984)

additive:

—

on the microflora of the digestive

It is usual for the antibiotic growth promoters to have

tract

a Gram-positive spectrum of activity; therefore they

—

on the colonisation of the digestive

cannot have a selection pressure on the Enterobacteri-

tract

aceae and do not contribute to antibiotic resistance in

–

on the shedding or excretion of

Salmonella spp. or E. coli.

pathogenic micro-organisms

1.2.5

Field studies to monitor bacterial

(2)

Effect on the excretion of enteric pathogens by

resistance

animals. An increased number of organisms or an

1.3

Studies of the metabolism and residues

extended shedding time in the faeces could lead to an

2.

Studies on excreted residues – ‘ecotoxicity’

increased risk of contamination of human food. All

3.

Studies on laboratory animals

current growth promoters have been examined for their

3.1

Acute toxicity

effect on Salmonella and, in many cases, other bacteria

Chronic toxicity

including E. coli. All modern studies, undertaken with

Mutagenicity

authorized inclusion levels, have given satisfactory

3.4

Carcinogenicity

results (Nurmi & Rantala, 1974; Hinton, 1988).

3.5

Reproductive toxicity

Ungemach (1996) considered that the use of growth

3.6

Toxicity of metabolites

promoters in animal feeds posed no risk to the indigenous gut flora of human consumers.

Microbiological safety of the use of growth promot-

(3)

Residues in the meat and the effect on human gut

ers has been included in the registration dossiers from

flora. Growth promoters administered in animal feed the earliest legislation. All the potential hazards of their do not leave detectable residues in the meat. Indeed, use have been well recognized from the very beginning. they can be used from birth to slaughter, i.e. they have The microbiological safety is assessed in three areas: a zero withdrawal. As there is no detectable residue in the meat, there is unlikely to be a selection pressure on

(1)

Selection of antibiotic resistance in the gut flora. human gut flora (Ungemach, 1996).

(2)

Effect on the excretion of enteric pathogens by animals.

The public health implications of the use of antibi-

(3)

Residues in the meat and the effect on human gut

otics in animal feeds have been examined on many

flora.

occasions:

Growth Promoters in Cattle • 1071

Netherthorpe Committee

1962

The mode of action of the hormone implants has not

Swann Committee

1969

been fully elucidated although some work has indicated

FDA Task Force

1972

that changes in serum concentrations of IGF-1 occur in

Kennedy Report

1977

implanted steers, while serum IGF-1 concentration

Office of Technology Assessment (OTA) Report 1979

in the control steers remains or changed or, indeed,

AVI Conference – “Ten Years after Swann”

1979

decreases across time. This suggests that IGF-1, the

National Academy of Sciences Review (NAS)

1980

essential growth hormone, is related to the additional

Council for Agricultural Science & Technology

gain of the implanted steers. However, the reason for

Report

1981

this linear decrease in serum IGF-1 concentration

American Society of Animal Science

1986

sometimes reported in control cattle is unclear. For

Office of Technology Assessment (OTA) Report 1995

example, it does not appear that the effect can be attri-

Scientific Conference on Growth Promotion in

buted to the control animals approaching their physio-

Meat Production

1995

logical maturity, because the control and implant

Antimicrobial Feed Additives

1997

animals usually exhibited similar degrees of fatness. The

Office International des Epizooties. The Use of

net effect could be an increase in protein synthesis and

Antibiotics in Animals. Ensuring the

protein gain, which translates into greater, more effi-

Protection of Public Health

1999

cient liveweight gain and profit for the cattle producer

Advisory Committee on the Microbiological

(Simpkins et al., 2000).

Safety of Food Report on Antimicrobial

The usual explanation is more mundane as bulls grow

Antibiotic Resistance in Relation to

faster and produce a leaner carcass than steers; steers

Food Safety

1999

grow faster and produce less fatty meat than heifers

All have discussed the hazards but none have proved

and cows. The differences in growth rate are usually

any risk to human health.

ascribed to hormonal status and the levels of androgen

in particular. The hormone implants are used to replace

or adjust the circulating hormone levels to achieve

Hormonal implants

optimal growth.

The efficacy of hormonal implants is well accepted,

Hormonal implants are not currently licensed for use in both alone and in combination with in-feed growth promoters in the European Union. Those listed in Table 63.6 are in use in the rest of the world.

Table 63.6

*Hormonal implants licensed for use as growth promoters in non-EU countries (modified from table in the report of the fifty-second meeting of Joint FAO/WHO Expert Committee on Food Additives (JECFA, 1999) **Product names***

Comparison of the composition (mg/implant) of hormonal implants used for growth promotion Oestradiol

Testosterone

Testosterone

Progesterone

Trenbolone

Target

propionate

acetate

cattle

Compudose 200

24

Cattle

Compudose 400

45

Cattle

Synovex S

20

200

Steers

Synovex H

20

200

Heifers

Synovex C

10

100

Calves

Steroid

20

200

Steers

Heiferoid

20

200

Heifers

Implix BM

20

200

Steers

Implix BF

20

200

Heifers

Torelor

40

200

Steers

Revalor

20

140

Calves

Revalor G

8

40

Steers

Revalor S

24

120

Steers

Revalor H

14

140

Heifers

Finaplix-S

140

Steers

Ralgro

Active ingredient is Zeranol 3 ¥ 12 mg pellets/dose

Cattle

a Product names are registered trade marks®.

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Table 63.7

The efficacy of an implant in grazing steers used either alone or in combination with supplementary feed and the in-feed growth promoter monensin. (Source: Elanco Animal Health in-house data.) **Treatment**

ADG (kg)

Increased ADG (kg)

Total gain in 150 days (kg)

Control

Compudose®

No supplement

0.554

0.631

0.077

11.55

Compound feed (1 kg)

0.613

0.709

0.096

14.4

Compound feed (1 kg) + monensin

0.659

0.782

0.123

18.45

®, registered trade mark (oestradiol 17b).

evidence that the response to an implant varies with

1.00

40

the start weight, with the greatest response in finishing

0.90

0.84

0.87

35

0.76

cattle being reported from the heaviest cattle (see Fig.

0.80

0.71

30

e

0.70

0.60

0.65

g

63.2).

25

0.60

Some carcass traits can be changed by the use of

0.50

20

implants, with lower dressing percentages (Kuhl et al., ADG (kg) 0.40

Percenta

15

1989), reduced marbling (Eversole et al., 1989) and a

0.30

10

reduction in top grade carcasses (Foutz et al., 1989)

0.20

5

0.10

being reported. These changes vary between implants,

0.00

0

but have not been associated with any significant

<300

300 to 400

>400

changes in the eating quality of the meat.

Initial weight (kg)

While the efficacy of hormonal implants is accepted,

Control

Implant

Percentage increase over control

the consumer safety of implanted meat has been a focus of conflict between the USA and the EU, leading to

Fig. 63.2

The effect of the initial weight on the percentage a complaint to the World Trade Organization (WTO). response from an implant in finishing cattle.

The USA consider the hormone ban in the EU to be an unjustified barrier to trade and have effectively won as well as the recent assessment of the SCVPH report this case at arbitration through the WTO Appellate by a subgroup of the Veterinary Products Committee Body. The findings of the case centred round the lack (VPC) of the Veterinary Medicines Directorate of a proper risk assessment prior to the imposition of (VMD) in the UK on the potential risks to human the ban on imports of meat from animals treated with health from hormone residues in bovine meat and

hormones.

meat products

This whole debate is best followed by reading the full

(<http://www.vmd.gov.uk/>) see under Publications.

Joint FAO/WHO Expert Committee on Food Additives

(JECFA) assessments:

The main areas of concern have been best summarized

in the VPC subgroup report and they are reviewed

Natural hormones (WHO Feed Additive Series 43–52nd

below:

Meeting JECFA)

(<http://www.inchem.org/documents/jecfa/jecmono/>

(1)

Direct consumer exposure to hormone residues in

v43jec05.htm)

meat. The residues are all very low compared with

Zeranol (WHO Feed Additive Series 44–53rd Meeting

the acceptable daily intakes (ADIs) identified by

JECFA)

JECFA. Therefore consumer exposure to oestra-

(<http://www.inchem.org/documents/jecfa/jecmono/>

diol, progesterone and testosterone will be very
v44jec14.htm)

low compared with those naturally produced in
Trenbolone (26th meeting JECFA)

the body (see Fig. 63.3). The ADI is an amount of
([http://www.inchem.org/documents/jecfa/jecmono/](http://www.inchem.org/documents/jecfa/jecmono/medicine/hormone%20that%20can%20be%20eaten%20every%20day)
medicine/hormone that can be eaten every day
v23je03.htm)

over a lifetime in the practical certainty that, based
and the Opinion of the Standing Committee on
on current knowledge, no harm will result.

Veterinary Measures related to Public Health
(2)

Hormonal residues can adversely affect the
(SCVPH) dated 30 April 1999

immune system. References used to support this
([http://europa.eu.int/comm/food/fs/sc/scv/out21_en.](http://europa.eu.int/comm/food/fs/sc/scv/out21_en.html)
argument in the SCVPH report did not reflect
html)

modern physiological understanding of the
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3.3

duction. This does not mean that the effects of BST on

3.5

3

the growth performance and carcass characteristics in

ADI

2.5

beef animals have been neglected. The earliest trial in

2

1.7

1959 (Brumby, 1959) and those in the late 1980s pro-

1.5

duced consistent effects leading to increased growth

1

Percent of

0.6

rates, improved feed conversion and carcass lean, while

0.5

0.01

0.06

0.05

decreasing carcass fat. The effects on eating quality of

0

the meat associated with reduced carcass fat were a

Oestradiol

Progesterone

Testosterone

reduced acceptability because of lower scores on ten-

derness. The available data in cattle on growth per-

Maximum exposure

formance were summarized by Enright (1989) and on

Minimum exposure

meat quality by Allen and Enright (1989). As nearly all

Fig. 63.3

The maximum and minimum consumer exposure to

the data summarized were from trials involving the

natural hormone residues from implanted meat as a percentage

daily injection of BST, and even sustained-release

of the JECFA ADI. (JECFA Joint FAO/WHO Expert Committee

injectable formulations only last 14 days, there does not

on Food Additives. ADI (acceptable daily intake) in the amount currently seem to be a place for its use in the beef industry of a medicine or hormone that can be taken every day during a

try as a growth promoter.

lifetime in the practical certainty no harm will result.)

A direct comparison has been made between ana-

bolic steroids and BST. Untreated controls (UC) were

compared with steroid implants (STI) and 160 mg BST

immune system. With reference back to the first

injected weekly (Preston et al., 1995). Daily gain was area of concern, the residues are such a small frac-increased by both treatments (UC 1.30 kg/day; STI

tion of daily endogenous hormone production as

1.66 kg/day; BST 1.51 kg/day) and feed conversion was

to be without effect.

improved (UC 5.92; STI 4.88; BST 5.13). The anabolic

(3)

Hormonal residues can lead to an increase in

effects of STI and BST were considered to be poten-

certain common cancers. However, epidemiologi-

tially additive.

cal evidence only shows a direct link between

meat and fat consumption; to relate that to

hormone residues is a leap in logic. The leap is

Repartitioning agents

difficult to support as these tumours are found equally widely in both men and women in countries where hormonal growth promoters are not permitted. *b*-Adrenergic receptor agonists (*b*-agonists) increase muscle mass and decrease fat mass when fed to cattle.

A wide range of compounds has been investigated (4)

Hormone residues in meat could affect many including cimaterol, clenbuterol, fenoterol, isoprenaline, mabuterol, ractopamine, salbutamol, terbutaline. The only evidence of such an effect comes from and zilpaterol (see Fig. 63.4). The most extensive review of the use of *b*-agonists in meat animals was published by Anderson et al. (1991), in which nearly 400 papers these suspicions in respect of residues, even with were reviewed. There are currently no *b*-agonists registered. No current data show any ground for the intensive research in this area.

tered in the EU for use in cattle as growth promoters,
The 'hormone' debate is about politics and the role
indeed in the EU this class of compound is specifically
of science to support obviously conflicting views. Even
prohibited under EU Directive 96/22/EU. There is one
if the science supporting the safety of hormones wins
product registered as a growth promoter in cattle in
the politicians can still ensure that there will be no con-
South Africa and Mexico – zilpaterol (Zilmax, Hoechst
sumer acceptance for this technology in the EU. It is
Roussel Vet) – and one in pigs in the USA, Mexico,
not anticipated that there will be a return of the implant
Brazil, Philippines and Korea – ractopamine (Paylean,
in beef production in the EU, but this should not
Elanco Animal Health).

jeopardize its use in other countries with less political
The mode of action is complex, with direct effects on
interference in product registration.

skeletal muscle cells, leading to hypertrophy, and on
adipocytes, causing lipolysis, as well as secondary mech-
anisms mediated by other hormones and changes in

Growth hormone (BST)

blood flow (Mersmann, 1998). Studies attempting to relate the relative density of β_2 -adrenoreceptors on The use of bovine somatotropin (BST) in cattle has muscle and the local response to β -agonists have failed primarily been concentrated on increasing milk pro- to find a useful tool to predict efficacy of compounds

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OH

Table 63.8

The effect of ractopamine on growth performance

H

in finishing cattle. A summary of six trials.

N

Dose (ppm)

ADG (kg)

ADF (kg)

FCR

H₂N

0

1.26a

9.40^a

7.46^a

10

1.34^b

9.31^a

6.95^b

Cimaterol

20

1.39^b

9.40^a

6.76^b

N

30

1.48^c

9.31^a

6.29^c

ppm, parts per million; ADG, average daily gain; ADF, average daily OH feed; FCR, feed conversion ratio; kg, kilogram. Means with different H superscripts are significantly different.

Cl

N

Table 63.9

The effect of ractopamine on carcass yield in

H2N

finishing cattle. A summary of six trials.

Cl

Clenbuterol

Dose (ppm)

Dressing (%)

Protein (%)

Fat (%)

OH

0

60.71a

15.16a

30.31

OH

H

10

60.76a

15.16a

30.24

N

20

61.02^b

15.36^a

29.70

30

61.03^b

15.54^b

28.81

HO

ppm, parts per million. Means with different superscripts are significantly different.

Ractopamine

and a ‘rebound’ when the product is removed, leading

OH

to an increase in fat deposition and a reduction in

H

*muscle mass (Elliot et al., 1993). The most effective use *N**

of a repartitioning agent is therefore in the finishing

period in the one to two months prior to slaughter. The

*results from the use of *b*-agonists in cattle are very con-*

HO

sistent and are summarized in Table 63.8, which shows

Salbutamol

HO

significant increases in growth rates and feed utilization, and Table 63.9, which shows significant increases in dressing percentage, increased carcass lean.

OH

b-agonists are usually divided into two classes (Smith,

H

1998):

N

(1)

Halogenated aromatic ring. This class tends to have a long serum half-life and when coupled with high oral absorption and potency of action in humans there

HN

N

can be a real safety concern from residues in meat. An

Zilpaterol

example of such a compound is clenbuterol (pharma-

O

codynamic dose in man 10 mg/day – Meyer & Rinker, 1991); levels found in contaminated meat in the EU

Fig. 63.4

Structural details of a range of b-agonists that have affected humans who consumed as little as 100 g of the been proposed as growth promoters in cattle product (Kuiper et al., 1998). See Table 63.10 for a summary of some poisoning incidents.

(Hoey et al., 1995). Moody et al. (2000) have undertaken (2)

Hydroxylated aromatic ring. The metabolism is a detailed review of the mode of action of the b-agonists much simpler with the hydroxylated b-agonists and in all species. When b-agonists are used as growth they tend to have relatively short plasma half-lives and promoters, two major problems arise during chronic when coupled with a low potency in humans they may exposure. Firstly, receptor downregulation leads to a pose little risk from residues. An example of this type fall off in effect over time (Peters, 1989; Re et al., 1994) of compound would be ractopamine, where the phar-

Table 63.10

A summary of some documented cases of β -agonist poisoning in Europe (after Witkamp, 1995).

Year

Source

Compound

No. of people

Symptoms

Country

1990

Bovine liver

Clenbuterol

135

Tremor, palpitations, tachycardia,

Spain

(0.16–0.30 mg/kg)

nervousness

1990

Calf liver

Clenbuterol

22

Tremor, headaches, dizziness,

France

(0.38–0.50 mg/kg

palpitations, malaise

1994

Calf liver

Clenbuterol (?)

127

Not specified

Spain

1995

Fillet, rump steak

Clenbuterol

16

Tachycardia, tremors, nervousness

Italy

(>0.5 mg/kg)

Class 1 – Halogenated aromatic ring

References and further reading

OH

Slow

H

Advisory Committee on the Microbiological Safety of Food

Cl

metabolic

N

(1999) Report on Antimicrobial Antibiotic Resistance in

inactivation

Relation to Food Safety. The Stationery Office, London.

H2N

Clenbuterol

Allen, P. & Enright, W.J. (1989) Effects of administration of Cl

*somatotropin on meat quality in ruminants: a review. In Use Half life in man T1/
= 30 hr*

2

*of Somatotropin in Livestock Production (ed. by K. Sejrsen, Pharmacodynamic
dose*

M. Vestergaard & A. Neimann-Sorensen), pp. 201–9.

in man 10 mg/day

Elsevier Applied Science, London and New York.

American Society of Animal Science (1986) Public health

implications of the use of antibiotics in agriculture. 77th

Class 2 – Hydroxylated aromatic ring

Annual Meeting, Suppl. 3. Journal of Animal Science, 62, OH

OH

Rapid

H

1–106.

metabolic

N

Anderson, D.B., McCracken, V.J., Aminov, R.I., Simpson, J.M.,

inactivation

Mackie, R.I., Verstegen, M.W.A. & Gaskins, H.R. (2000)

HO

Gut microbiology and growth-promoting antibiotics in

Ractopamine

*swine. Nutrition Abstracts and Reviews. Series B, Livestock Half life in man T1/
= 4 hr*

2

Feeds and Feeding, 70, 101–8.

Pharmacodynamic dose

Anderson, D.B., Veehuisen, E.L., Schroeder, A.L., Jones, D.J.

in man 25 000 mg/day

& Hancock, D.L. (1991) The use of phenethanolamines to reduce fat and increase leanness in meat animals. In

Fig. 63.5

A comparison with the metabolic deactivation rate

Proceedings of Symposium on Fat and Cholesterol Reduced

and plasma half-life of halogenated and hydroxylated aromatic

Foods – Advances in Applied Biotechnology Series (ed. by ring b-agonists.

C. Haberstroh & C.E. Morris), pp. 43–73. Portfolio

Publishing, New Orleans.

Anderson, D.B., Veehuisen, E.L., Smith, C.K., Dalidowicz, J.E., macodynamic dose in man exceeds 25 000 mg/day

Donoho, A.L., Williams, G.D., Jones, D., Schroeder, A.L.,

(Paylean Freedom of Information Summary).

Turbert, M.P. & Guneratne, R.J. (1993) Ractopamine

hydrochloride: a unique PLE (phenethanolamine lean

Effective doses of ractopamine and zilpaterol are

enhancer) for swine. In Proceedings of the 11th World

reported to have negligible to undetectable residue

Association of Veterinary Food Hygienists (WAVFH) Sym-

levels in the target species (Anderson et al., 1993;

posium (ed. by P. Ingkaninum & P. Poomvises). pp. 96–102.

Zilmax Technical Manual, 1998). These products have

The Thai Veterinary Medical Association, Bangkok.

only been cleared for use in food animals after exten-

Anon (1996) Working Group II – Assessment of health risk.

sive reviews of the safety of residues and it is perhaps

Scientific Conference on Growth Promotion in Meat

unreasonable for a class ban to have been introduced

Production, pp. 19–25. EU Commission DG VI Agriculture.

in the EU based on the misuse of clenbuterol when safe

*Association of Veterinarians in Industry (1979) In Proceedings of the Conference
‘Ten Years after Swann’. AVI, London.*

alternatives were in development.

Bartley, E.E., Nagaraja, T.G., Pressman, E.S., Dayton, A.D.,

The classes of β -agonist are contrasted in Fig. 63.5.

*Katz, M.P. & Fina, L.R. (1983) Effects of lasalocid or monThe use of highly
active β -agonists as growth pro-*

*ensin on legume or grain (feedlot) bloat. Journal of Animal moters should not be
pursued because of the potential*

Science, 56, 1400–406.

hazards for human and animal health (Anon, 1996),

Brumby, P.J. (1959) The influence of growth hormone on

however this should not preclude the development of

growth in young cattle. *New Zealand Journal of Agricultural safe effective products* (Smith, 1998).

Research, 2, 683–9.

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Chen, G. & Russell, J.B. (1989) More monensin-sensitive,

oxytetracycline on *Salmonella typhimurium* in swine, calves ammonia producing bacteria from the rumen. *Applied*

and chickens. *Antimicrobial Agents and Chemotherapy*, 8, *Environmental Microbiology*, 55, 1052–5.

664–72.

Chow, J.M. & Russell, J.B. (1990) Effect of ionophores and pH

Eversole, D.E., Fontenot, J.P. & Kirk, D.J. (1989) Implanting on growth of *Streptococcus bovis* in batch and continuous trembalone acetate and estrodiol in finishing beef steers.

culture.

Applied Environmental Microbiology,

56,

Nutritional Reports International, 39, 995–8.

1588–1561.

Ewing, W.N. & Cole, D.J.A. (1994) *The Living Gut*, pp. 67–74.

Coates, M.E. (1980) The gut micro-flora and growth. In

Context, Dungannon.

Growth in Animals (ed. by T.L.J. Lawrence), pp. 175–88.

Food and Drug Administration (FDA) (1972), Report of the Butterworths, London.

FDA task force on the use of antibiotics in animal foods. US Commission on Antimicrobial Feed Additives (1997) Report.

Department of Health, Education and Welfare.

Stockholm, Sweden.

Foutz, C.D., Dolezal, H.G., Gill, D.R., Strasia, C.A., Gardner, Corpet, D.E. (1984) The effect of bambermycin, carbadox,

T. & Ray, F.K. (1989) Trembalone acetate effects on carcass chlortetracycline and olaquinox on antibiotic resistance

traits of yearling feedlot steers. Journal of Animal Science, in intestinal coliforms: a new animal model. Annals of

67, (Suppl. 1), 434.

Microbiology (Pasteur Institute) 135A, 329–39.

Gaines, S.A., Rollins, L.D., Williams, R.D. & Selwyn, M. (1980) Corpet, D.E. (1996) Microbiological risks for the consumer of

Effect of penicillin and virginiamycin on drug resistance in

antimicrobial growth promoters used in meat production.

lactose-fermenting enteric flora. Antimicrobial Agents and In Proceedings of the Scientific Conference on Growth

Chemotherapy, 17, 428–33.

Promotion in Meat Production, pp. 401–29. European

Goodrich, R.D., Garrett, J.E., Gast, D.R., Kirick, M.A., Larson, Commission DGVI, Brussels.

D.A. & Meiske, J.C. (1984) Influence of monensin on the

Council for Agricultural Science and Technology (1981)

performance of cattle. Journal of Animal Science, 58, Antibiotics in animal feeds. Report No. 88. Ames, Iowa.

1484–98.

Craene, A. de & Viaene, J. (1992) Economic effects of tech-Hays, V.W. (1979) Benefits: antibacterials. In Drugs in

nology in agriculture. Do performance enhancers for

Livestock Feeds. Vol. 1. Technical Report, pp. 29–36. Office animals benefit consumers? Department of Agro-marketing, of Technology Assessment, Congress of the United States,

Faculty of Agricultural Science, University of Ghent,

Washington, DC.

Belgium.

Hedde, R.D., Armstrong, D.G., Parish, R.C. & Quach, R.

Dawson, K.L. & Boling, J.A. (1983). Monensin-resistant bac-

(1980) Virginiamycin effect on rumen fermentation in

teria in the rumen of calves on monensin-containing and

cattle. Journal of Animal Science, 51, (Suppl. 1), 366.

unmedicated diets. Applied Environmental Microbiology, Hinton, M. (1988) Salmonella colonization in young chickens

46, 160–2.

given feed supplemented with the growth promoting antibi-

Dealy, J. & Moeller, M.W. (1977). Influence of bambarmycins otic avilamycin.
Journal of Veterinary Pharmacology and

on E. coli and antibiotic resistance in calves. Journal of Therapeutics, 11, 269–75.

Animal Science, 45, 1239–42.

Hoey, A.J., Reich, M.M., Davis, G., Shorthose, R. & Sillence, Dennis, S.M.,
Naharaja, T.G. & Bartley, F.E. (1981). Effects of M.N. (1995) Beta 2-
adrenoceptor densities do not correlate

lasalocid or monensin on lactate-producing or -using rumen

with growth, carcass quality, or meat quality in cattle.

bacteria. Journal of Animal Science, 52: 418–22.

Journal of Animal Science, 73, 3281–6.

Devriese, L.A. (1980) Sensitivity of Staphylococci from farm Kennedy, D.F.
(1977) Antibiotics used in animal feeds. HEW

animals to antimicrobials used for growth promotion and

News. US Department of Health, Education and Welfare,

therapy, a ten year study. Annals of Veterinary Research, 11, Rockville.

399–408.

Kuhl, G., Simms, D.D. & Hartman, P.D. (1989) Sequential

Dutta, G.N. & Devriese, L.A. (1984) Observations on the in implanting with
Synovex-S or Synovex-S + Finaplix-S on

vitro sensitivity and resistance of Gram positive intestinal

steer performance and carcass characteristics. Journal of

bacteria of farm animals to growth promoting antimicrobial

Animal Science, **67**, (Suppl. 1), 434.

agents. *Journal of Applied Bacteriology*, **56**, 117–23.

Kuiper, H.A., Noordam, M.Y., van Dooren-Flipsen, M.M.H.,

Elliot, C.T., Crooks, S.R.H., McEvoy, J.G.D., McGaughey, W.J.,

Schilt, R. & Roos, A.H. (1998) Illegal use of β -adrenergic Hewitt, S.A.,

Patterson, D. & Kilpatrick, D. (1993)

agonists: European Community. *Journal of Animal Science*, Observations on the effects of long-term withdrawal on

76, 195–207.

carcass composition and residue concentrations in

Lawrence, K. (1998) Growth promoters in swine. In *Proceed-*

clenbuterol-medicated cattle. Veterinary Research, **132**, 459–

ings of the 15th IPVS Congress, Birmingham, England, pp 68.

337–43.

Enright, W.J. (1989) Effects of administration of somatotropin Linton, A.H.

(1982) Host–parasite interactions in health. In

on growth, feed efficacy and carcass composition of rumi-

Microbes, Man and Animals (ed. by A.H. Linton), p. 42.

nants: a review. In *Use of Somatotropin in Livestock*

John Wiley & Sons, Chichester.

Production (ed. by K. Sejrsen, M. Vestergaard & A.

Lowe, L.B., Ball, G.J., Carruthers, V.R., Dobos, R.C., Lynch,

Neimann-Sorensen), pp. 201–9. Elsevier Applied Science,

G.A., Moate, P.J., Poole, P.R. & Valemtine, S.C. (1991)

London and New York.

Monensin controlled-release intraruminal capsule for

Evangelisti, D.G., English, A.R., Girard, A.E., Lynch, J.E. & control of bloat in
pastured dairy cows. Australian Veteri-Solomons, I.A. (1975) Influence of
subtherapeutic levels of

nary Journal, **68**, 17–20.

Growth Promoters in Cattle • 1077

Mackinnon, J.D. (1987) The role of growth promoters in pig

Pressman, B.C. (1976) Biological applications of ionophores.

production. The Pig Veterinary Society Proceedings, **17**, Annual Revue of
Biochemistry, **45**, 501–30.

69–100.

Preston, R.L., Bartle, S.J., Kasser, T.R., Day, J.W., Veenhuizen, Mee, B.J. (1982)
The selective capacity of pig feed additives

J.J. & Baile, C.A. (1995) Comparative effectiveness of soma-and growth
promotants for coliform resistance. In Proceed-

totropin and anabolic steroids in feedlot steers. Journal of ings of the 4th
International Symposium. Antibiotics in

Animal Science, **73**, 1038–47.

Agriculture: Benefits and Malefits, pp. 349–58.

Re, G., Badino, P., Novelli, A. & Girardi, G. (1994) Homolo-Mersmann, H.J.
(1998) Overview of the effects of b-adrenergic

gous b-adrenoreceptor downregulation and heterologous agonists on animal growth including mechanisms of action.

estrogen and progesterone upregulation induced by

Journal of Animal Science, **76**, 160–72.

repeated b2-adrenergic stimulation in the female reproduc-

Meyer, H.H. & Rinke, L.M. (1991) The pharmacokinetics and

tive tract of veal calves. In Proceedings of the 6th Congress residues of clenbuterol in veal calves. Journal of Animal

of the European Association of Veterinary Pharmacology

Science, **69**, 4538–44.

and Toxicology, Edinburgh, pp. 7–11.

Moody, D.E., Hancock, D.L. & Anderson, D.B. (2000)

Richardson, L.F., Raun, A.P., Potter, E.L., Cooley, C.L. & Phenethanolamine repartitioning agents. In Farm Animal

Rathmacher, R.P. (1976) Effect of monensin on rumen fer-

Metabolism and Nutrition, (ed. by J.P.F. D'Mello), pp. 65–96.

mentation in vitro and in vivo. Journal of Animal Science, CABI Publishing, Wallingford.

43, 657–62.

Nagaraja, T.G., Avery, T.B., Galitzer, S.J. & Harmon, D.L.

Sakauchi, R. & Hoshino, S. (1981). Effects of monensin on

(1985) Effect of ionophore antibiotics on experimentally

ruminal fluid viscosity, pH, volatile fatty acids and ammonia induced lactic acidosis in cattle. *American Journal of levels, and microbial activity and population in healthy and Veterinary Research*, **46**, 2444–52.

bloated steers. *Zeitschrift für Tierphysiologie, Tierernährung* Nagaraja, T.G., Taylor, M.B., Harmon, D.L. & Boyer, J.E.

und *Futtermittelkunde*, **46**, 21–33.

(1987) *In vitro* lactic acid production and alterations in Scientific Conference on Growth Promotion in Meat Production volatile fatty acid production by antimicrobial feed addition (1995) *Proceedings European Commission DGVI, tives. Journal of Animal Science*, **65**, 1064–76.

Brussels.

National Academy of Sciences, National Research Council Simpkins, S., McElhenny, W., Rahe, H., Wolfe, D., Mulvaney, D.

(1980) *The effects on human health of subtherapeutic use of*

& Lino, S. (2000) *Weighing the advantages. Do growth-*

antimicrobials in animal feeds. National Academy Press, promoting implants impact quality beef? Highlights of

Washington.

Agricultural Research, Auburn Veterinary College, **47**, 1–4.

Netherthorpe (1962) *Report of the joint committee on anti-*Smith, D.J. (1998)

*The pharmacokinetics, metabolism, and
otics in animal feeding.*

Agricultural Research

*tissue residues of b-adrenergic agonists in livestock. Journal Council/Medical
Research Council, London.*

of Animal Science, 76, 173–94.

*Newbold, C.J., Wallace, R.J. & Walker, N.D. (1993) The effects Swann, M.
(1969) Report: Joint Committee on the Use of*

of tetracycline and monensin on fermentation, microbial

*Antibiotics in animal Husbandry and Veterinary Medicine, numbers and the
development of ionophore-resistant bac-HMSO, London.*

*teria in the rumen. Journal of Applied Bacteriology, 75, Ungemach, F.R. (1996)
Safety aspects of growth promoters*

129–34.

used as feed additives. In Proceedings of the Scientific

Nurmi, E. & Rantala, M. (1974) The influence of zinc baci-

Conference on growth promotion in meat production, pp.

*tracin on the colonization of Salmonella infantis in the intes-333–45. European
Commission DGVI, Brussels.*

*tine of broiler chickens. Research in Veterinary Science, 17, Van Maanen, R.W.,
Herbein, J.H., McGillard, A.D. & Young,*

24–7.

*J.W. (1978) Effects of monensin on in vitro rumen produc-Office International
des Epizooties (1999) The use of antibi-*

tion and blood glucose kinetics in cattle. *Journal of Nutrients in animals. Ensuring the protection of public health,*

tion, **108**, 1002–1008.

In Proceedings of the Conference, 24–26 March 1999, Paris, Verbeke, R. & Viaene, J. (1996) Environmental impact of using France.

feed additives. Economic implications and legal environment Office of Technology Assessment (1979) Drugs in livestock

in Benelux, Department of Agro-marketing, Faculty of

feed. Vol. I. Technical Report. Congress of the United States, Agricultural Science, University of Ghent.

Washington, DC.

Visek, W.J. (1979) The mode of action of growth promotion by

Office of Technology Assessment (1995) Impact of antibiotic-antibiotics. Journal of Animal Science, 46, 1447–69.

resistant bacteria.

Congress of the United States,

Watkins, L.E., Wray, M.I., Basson, R.P., Feller, D.L., Olson,

Washington, DC.

R.D., Fitzgerald, P.R., Stromberg, B.E. & David, G.W. (1986) Peters, A.R. (1989) Beta-agonists as repartitioning agents: a

The prophylactic effects of monensin fed to cattle inoculated with coccidia oocysts. Veterinary Record, 124, 417–20.

lated with coccidia oocysts. Agri-Practice, 7, 18–20.

Pinkerton, M. & Steinrauf, K.L. (1970) Molecular structure of Witkamp, R.F.

(1995) *The use of β_2 -agonists as growth promoters in food species. Toxicological aspects and possible health risks to consumers.* In *Proceedings of the Scientific Conference on Growth Promotion in Meat Production*, pp.

of Molecular Biology, **49**, 533–46.

of Molecular Biology, **49**, 533–46.

health risks to consumers. In *Proceedings of the Scientific Conference on Growth Promotion in Meat Production*, pp.

R.M. (1986) *Effects of monensin on the performance of*

297–332. EU Commission DG VI Agriculture.

cattle on pasture or fed harvested forages in confinement.

Zilmax© Technical Manual (1998) Hoechst Roussel Vet,

Journal of Animal Science, **62**, 583–92.

GmbH, Rheingaustrasse 190, D-65203 Wiesbaden.

Chapter 64

Injection Damage

J.H. Pratt

Introduction

1078

would constitute a major contribution to future meat

Damage produced by injection

1078

quality standards and may now prove possible through-

Intramuscular injection sites

1079

out Western Europe where individual cattle identifica-

Methods of introducing contamination

1079

tion is obligatory under European Community law.

*Where the farm of origin can be determined, advice
on proper injection techniques can be given thereby*

Introduction

*improving the welfare interests of the animal and
enhancing the future profitability of the producer.*

*The faulty administration of veterinary medicines by
Losses incurred by the abattoir through the downg-
injection can give rise to complaints from meat whole-
rading of affected cuts of beef from incorrect injection
salers and retailers and on occasions from consumers.
procedures would also be reduced.*

The lesion resulting from the injection of a drug may

*It is to be regretted that in Britain the use of data
be aesthetically repugnant to the consumer and com-
collected in the abattoir has not been exploited to its*

mercially unacceptable in the case of abscessation or full potential. Such information can play a significant fibrosis in muscle tissue to the wholesaler, the retailing role in future preventive veterinary medicine strategies butcher and the supermarket outlet.

and in the depiction of regional and national animal disease patterns.

The losses incurred by the meat industry through

Damage produced by injection

poor injection technique are spasmodic but nevertheless can be substantial. There are, unfortunately, no Abscesses deep in the gluteal region of cattle are reliable figures in UK meat inspection records for reported since the site is frequently used for intramuscular injection of drugs. Damage in this area reduces injection procedures alone. However, on occasions a the value of a relatively expensive part of the carcass. number of animals within one consignment of cattle Abscessation may render the entire limb unsuitable for exhibit deep muscle fibrosis consistent with damage

human consumption. Intravenous injections can result from injected material, necessitating the rejection of in haematoma formation and abscess production. In 1.5–2 kg of a high-priced cut of meat. These lesions are some cases this is due to irritant substances invading the formed in the same anatomical area in a number of perivascular tissue rather than being delivered properly animals within a group from the same unit. The similarity in age, character and distribution of these lesions into the bloodstream. In addition, the deep injection of an irritant substance causing a localized reaction within suggests a common cause, the introduction of foreign muscle tissue may elude detection at meat inspection material by injection. Both injectable nemicides and and even later in the cutting premises. The damage may substances used in the regulation of bovine reproduction, e.g. some prostaglandins, may have been responsible for such damage encountered in recent years in this shop by which time it may prove difficult to trace the

country.

animal back to the farm of origin. Indeed, in countries
The leather industry also suffers financial loss, as
where complex marketing arrangements exist, the
injection damage due to scarring and abscessation can
tracing of animals back through marketing chains may
reduce the value of the hide, particularly if the lesion is
prove impossible until unique individual identification
in a position that prevents the optimal use of the com-
of the live animal and the resultant carcass and cuts
plete hide for large leather or suede items of clothing
becomes feasible in all stock. Such an achievement
and upholstery.

1078



Injection Damage • 1079

Intramuscular injection sites

Alternative siting of injections to that of the gluteal region is therefore desirable. The middle third of the neck, about one-third of the way down from the top, is

recommended (see Figs 64.1 and 64.2); this in itself does not reduce the incidence of abscessation or muscle scarring following the injection of drugs but trimming of this less expensive area reduces blemishing of the carcass. This area also tends to be cleaner than the hindquarters and so it is less likely that infection will be introduced with injection.

The factors contributing to muscle damage and abscess formation must therefore be identified to allow the formulation of advice for veterinarians and animal owners to reduce such damage.

The efficiency of the host animal's inflammatory response in removing the foreign protein and devitalized tissue together with the persistence of the stimulus determine the size and progression of the abscess formation. In mild cases no abscess may form, but scar

Fig. 64.1

A growing calf being injected into the neck muscle. tissue will mark the tissue damage. When an abscess has been formed and the inflammatory stimulus removed the abscess may persist for a prolonged period before

resolving to scar tissue.

Methods of introducing

contamination

The introduction of foreign protein may occur in a variety of ways.

Contamination of injected drugs: This is most likely to occur prior to administration as a result of poor

hygienic procedures employed. To obviate contamination of the contents of a bottle or vial a separate sterile needle to that used for injection must remain attached to the container and used only to refill the syringe.

In multiple dosing an automatic injection mechanism avoids this requirement and results in a smoother and more efficient operation. The manufacturer's storage instructions should be followed; unused vaccine should be discarded. When therapeutic agents such as antibiotics are used they should be used up as quickly

Fig. 64.2

A bull adequately restrained before introducing an as possible after opening.

injection into the neck muscles.

Contamination of needle or syringe: Ideally, a sterile needle should be used for every injection but in practice 10–15 cattle, when groups of animals are injected and

cal situations this is often not feasible; however, in any event when contamination of the equipment special circumstances this approach may be applicable occurs or is suspected or the needle is in any way on veterinary advice in certain herds to avoid transfer damaged.

of specific viral and other infections such as enzootic bovine leukosis (EBL). It is good practice, however, to Punching a fragment of host skin: The introduction of change needles and syringes regularly, i.e. after every host tissue along with the injection material is most

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likely to occur when damaged needles are used. The equipment and uncontaminated injectable materials. regular changing of needles will reduce the chances of Preparation of the injection site is an integral part of this occurring. When needle damage has obviously this hygienic approach; the avoidance of contaminated occurred a replacement is necessary in order to avoid

sites of injection is essential. A clean site should be possible injury to the animal and to maintain good meat chosen. If not available an area should be cleaned, or if hygiene and quality standards.

necessary clipped and cleaned.

There are now available multidose syringes with special sterilization caps to reduce the possibility of Irritant injections: The introduction of an irritant infections. Caps can also be placed on the tops of substance that devitalizes tissue locally can be responsive therapeutic agents.

sible for direct or indirect damage to muscle tissue.

The recommended route of administration and the Examples used in veterinary medicine are: basic and injection technique outlined in the manufacturer's acidic compounds, such as the sulphonamide drugs instructions must be followed. The incorrect route can and spectinomycin; hypertoxic preparations, e.g. long-lead to tissue damage, e.g. drugs recommended for acting oxytetracycline; and adjuvants, e.g. aluminium subcutaneous administration must not be given by the

hydroxide, saponin.

intramuscular route or intraperitoneally unless specifi-

*Vasoactive substances, e.g. local anaesthetic prepara-
cally indicated.*

tions containing adrenaline and some synthetic

*Proper restraint of the animal will lessen the likeli-
prostaglandins, lead to ischaemic necrosis.*

*hood of tissue damage and allow hygienic procedures
to be followed. The injection of a fractious or nervous*

*Contaminated skin: Pathogenic bacteria may be intro-
animal can lead to unnecessary suffering and damage
duced not only when injecting cattle with a contami-
to the animal, not to say injury to the operator also.*

nated drug or when the needle or syringe is dirty but

Furthermore, struggling while injecting an animal may

*when the skin surface is not clean; the introduction of
preclude the proper administration of the medicine*

pathogens from the site of injection will occur in a

with less than the prescribed dose deposited at the

proportion of cattle treated. It is therefore incumbent

recommended site, the remainder forming a depot with

*upon the veterinarian and the stock owner to employ
ensuing damage. Thus all animals should be adequately
hygienic injection procedures at all times using sterile
restrained (see Figs 64.1 and 64.2).*

Chapter 65

Alternative Medicine

C.E.I. Day

Introduction

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observer, but they are united in several respects, in that

Homoeopathy

1082

they are:

Principles

1082

How to cure

1082

- *Natural, relying on no man-made chemicals for*

Conversion to veterinary application

1084

their effect;

Application

1085

- *Able to work with and through the body's own*

The challenge of herd treatment

1088

mechanisms, stimulating the 'vital force' or 'life

Materia medica

1088

Prescribing guide

1090

energy' in order to achieve health;

Acupuncture

1091

- *Holistic, embracing the concept of mind and body*

Theory

1092

as a whole in harmony with the environment, and

Diagnosis

1096

recognizing the interaction of the whole body with

Treatment

1098

each part, and the interaction of the parts with the

Technique of needling and treatment

1101

whole body and with each other; and

Homoeopathy and acupuncture combined

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• *Environmentally and dietarily appropriate, since*

Herbal medicine

1102

they give rise to no tissue residues in animals so

Essential oils

1102

treated.

Other therapies

1103

Conclusion

1103

Exceptions to this last point are herbal medicine and aromatherapy, in that they can give rise to residues and taints, thus making the selection and use of herbs

Introduction

and oils, as medicines for food-producing animals, a matter for great caution (see below).

Alternative medicine, in all its many modalities, was the

Owing to practical considerations, there are two main

object of a great resurgence of interest in the latter part

systems of alternative medicine that have been devel-

of the twentieth century. This phenomenon is consistent

oped and refined in the field of bovine medicine and

with the trend towards environmental consciousness

that have become partially accepted by the conven-

(the so-called 'green' movement), which embraces an

tional 'scientific' community. These are homoeopathy

awareness of the importance and of the mechanisms of

and acupuncture. Of these, the one used to the greater health. A large body of consumers now recognizes the

extent is homoeopathy, since it lends itself more easily

vital role played by diet in the maintenance of health.

to rapid, less specialized treatments and is applicable to

They have an awareness of the effect of additives and

herd medicine. Acupuncture is more demanding of pro-

of residues upon the value of food items and an under-

professional time and is not appropriate to herd applications of the potential impact of livestock medication. Both are very well suited to use on organic farm upon the health of food animals and upon the whole-units, since neither gives rise to any medicinal residues in tissues or in milk.

As is suggested by the epithet, alternative medicine. The title 'alternative' is more appropriate for these is relevant to this trend, in that it provides an 'alternatives' to what has become the accepted system of medicine, modern conventional drug medicine. There are full understanding of its principles and practice for its many so-called systems of alternative medicine (sometimes now described as 'complementary medicine' in contentious sentiments. The following is a brief introduction to the concepts of each of these two, followed

*bodied in the word alternative). The wide range of
by more medical detail. Other therapies, being less
such systems can present a baffling array to the casual
appropriate in the context of cattle medicine, are*
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*described in very brief format, in order to act as an
(i.e. diluted 1/100, 30 times over) giving rise to a dilu-
introduction only.*

*tion of 10-60 (at 10-23 Avogadro's hypothesis suggests
that it is unlikely that a molecule could be carried over
to the next stage). What Hahnemann found, with his*

Homoeopathy

*serial dilution and succussion method (succussion is the
violent shaking of the solution at each stage of dilu-*

*Founded by a German physician, Hahnemann, in the
tion), were two phenomena. Not only was the toxic
latter part of the eighteenth century (1790), homoeopa-
power of the substance reduced at each stage (easily
thy is a system of medicine embodying the principle
understandable) but also the curative power was*

‘ similia similibus curentur’ meaning ‘let like be cured by increased (not so readily acceptable). It is a fact of life

like’. This classical slogan is often enough in itself to that this latter phenomenon exists and that science has deter the casual student, since it constitutes an entirely not yet found the way to explain it. It therefore remains, opposite stratagem to that of conventional ‘school unexplained, to puzzle the reader and sadly, in so many medicine’. It is hoped that the reader will keep an cases, to act as a complete blockade to further enquiry. open mind, forming opinions only after searching and The big advantage of Hahnemann’s discovery of the objective study into the relative merits of each strategy effects of extreme dilutions, made prior to any under- and the part each system may play in the overall standing of molecules and atoms, is that there can be no fight against disease. The provision of an objective drug residues in meat or milk. This makes homoeopa- study requires some comparisons with the ‘known’ to thy a front-runner in the choice of medicine for organic be made, so as to shed light on the ‘unknown’ which

cattle units. Whereas herbal medicine, using the primary homoeopathy represents.

effects of material doses of pharmacologically active

The principle of like curing like is elevated from the plant material, relies on achieving significant tissue constancy of an amusing idea or the product of a fertile imagination by the fact that it exploits a natural law of secondary effects (the body's reaction) for the mediation (The Law of Similars). It was not an invention, therefore its medicinal benefit. It depends not upon material doses but upon energy interactions. The result of this is hit on his discovery as a result of objective testing of that, as there has been a rapid growth in conversions of the effects of Cinchona bark (the parent material of quinine and an accepted cure for malaria even today). demand for skills in farm homoeopathy, stimulating a He dosed a healthy person (namely himself) in order to burgeoning interest among cattle veterinarians for

try to discover the mode of action of Cinchona bark, as proper training. With the holistic discipline this study he could not accept the current eighteenth-century brings, cattle welfare can only be a beneficiary. explanation for its activity. What happened must have In more modern times, and in a veterinary context, been a great shock to him, for he developed symptoms clinical trials have revealed good results in many fields. quite indistinguishable from malaria. Cause and effect Examples are Caulophyllum and its use in controlling he objectively established, by alternately withholding porcine stillbirths (Day, 1984a), bovine mastitis prevention and restarting the administration of the substance. This tion studies (Day, 1986), bovine dystokia prevention led him to formulate his hypothesis: ‘ To cure mildly, (Day, 1985), reduction of calf rearing losses (Mahé, rapidly, certainly and permanently, choose in every case 1987) and control of canine kennel cough (Day, 1987a). of disease, a medicine which can itself produce an affection similar to that sought to be cured’ (quotation from

Principles

Organon der Heilkunst by Hahnemann 1790, translation by Boericke & Dudgeon).

A more detailed approach to veterinary homoeopathy

Having carried out tests (Prüfungen in German, is provided by Day (1984b, 1987b).

which became translated as ‘provings’) on healthy volunteers, with many substances, he started to use

How to cure

this system of medicine on his patients. He soon found that his crude substances evoked quite serious ‘aggra-

Hahnemann’s four steps to cure (a word he used very vations’ in his patients, so he proceeded to dilute the

objectively, to indicate a removal of symptoms/signs

remedies. This led to the second great stumbling block

with no further need for therapy to maintain that

for the modern, conventionally-trained observer in that

healthy state) were: (i) know the remedies, (ii) know the

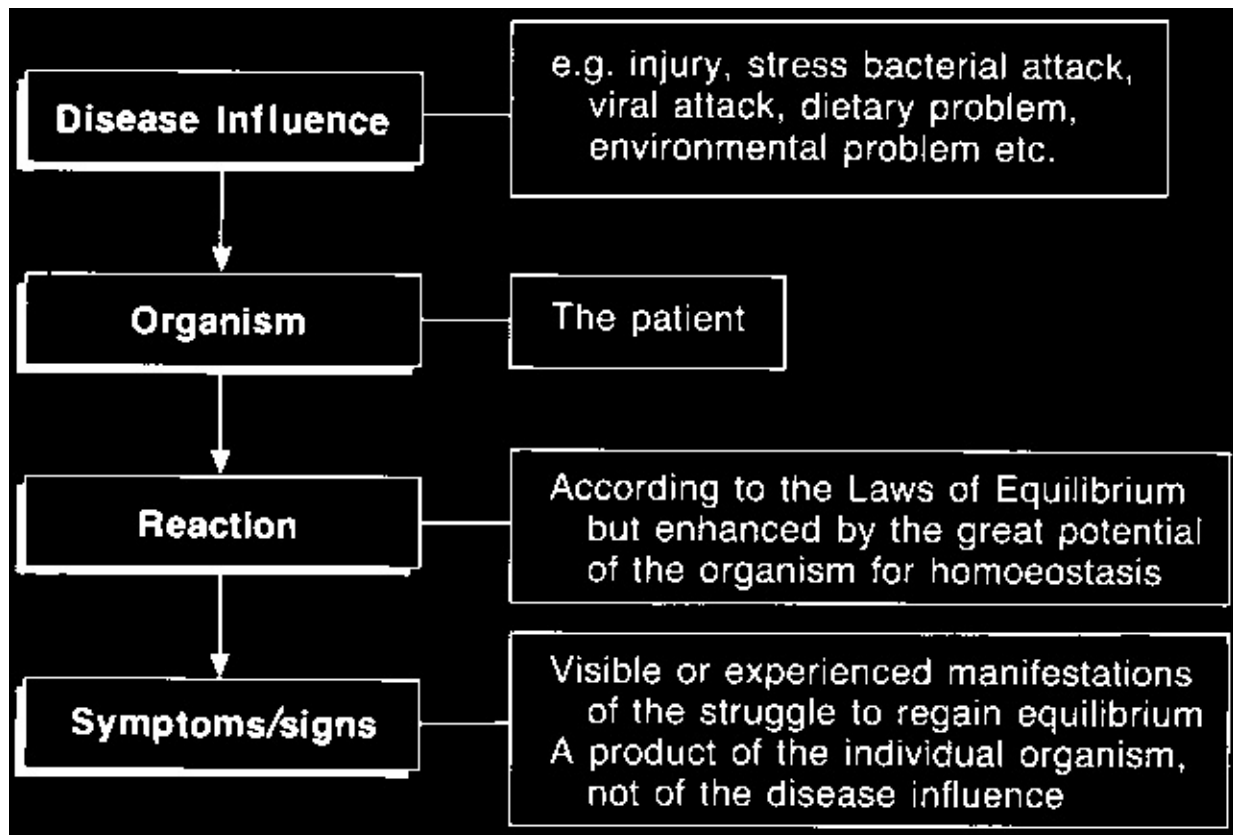
his commonly used dilutions exceeded those that could

disease, (iii) match the remedy to the disease and (iv)

reasonably be expected to contain even a single mole-

remove the obstacles to recovery. Our objective in vet-

*cule of the original substance. His dilutions, called
erinary medicine is to cure disease wherever possible,
'potencies', were often to the extent of 30c and beyond
and it is difficult to argue with the logic of Hahnemann's*



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*four steps. The fourth is, in particular, a little surprising
in a positive way, in view of our modern culture's
opinion of the soundness of eighteenth century medical
beliefs and practices.*

Know the remedies

A working knowledge of the ‘provings’ and actions of each individual ‘remedy’ or medicine is the basis of this stage. Obtaining this knowledge requires dedication.

The remedies are written up in books of Materia Medica (e.g. Boericke, 1972; Clarke, 1982; Macleod, 1983; Vermeulen, 1997), which may run into several volumes. These books give an account of the properties of the remedies, in terms of source, general properties,

Fig. 65.1

A disease event in an animal.

‘provings’ and clinical findings with applications. This represents a huge body of knowledge on each remedy’s known (‘proven’ by Hahnemannian tests) capabilities.

(1)

Aetiological or historical influences.

The information is recorded in very fine detail and

(2)

Generals:

symptoms/signs/properties of the

for each part of the body and mind, with its assumed

whole body, e.g. build, disposition, response

capabilities (from clinical data).

to environment, response to food, response to

*It is fair to say that, despite our pharmacological
climate, etc.*

knowledge of modern manufactured drugs, there is not

(3)

Mentals: symptoms/signs relating to the mind,

the equivalent amount of knowledge of their actions in

e.g. demeanour, behaviour, character, responses

the body and, for the most part, very little is known of

to various mental stimuli.

a drug's possible actions apart from the major primary

(4)

Particulars: the symptoms/signs displayed in each

and local effects. It may also be fair to say that not

part of the body, in fine detail (the gleanings of a

enough pharmacodynamic studies have been made of

very thorough clinical examination and enquiry).

homoeopathic remedy actions but, if research is never

Also any 'modalities' shown by these signs, e.g.

directed this way, that information will never accrue.

‘worse when cold and wet’, ‘worse after food’,

Not even the most learned veterinary homoeopath

‘better after exercise’, etc.

*has a full knowledge of even the major remedies (let
alone of the full range of several thousand available),*

Match the remedy to the disease

*since there is so much detail to be learnt. A basic knowl-
edge of major remedies is nonetheless essential to good*

*The findings of a detailed history-taking plus clinical
practice, as well as a superficial knowledge of minor
examination and the body of knowledge contained in
remedies. The hard work and dedication required, to
Materia Medica each constitute a picture that can be
obtain the necessary working knowledge, discourages
cross-matched, but this is often a daunting task. As each
many would-be practitioners.*

*disease incident is considered to be a unique event and
several thousand possible remedies exist, the task may
appear impossible. Once a basic knowledge of a good*

Know the disease

number of remedies is obtained, however, then the

Since each set of symptoms and signs, displayed by ability of the human brain to 'compute' at high speed each individual patient suffering disease, is a product makes the task achievable, in an acceptable number of of the individual's own reaction to a disease influence, cases, for the conscientious student, after several years then each disease occurrence encountered is a unique of practice and study. There are also books to aid the incident (Fig. 65.1).

process, which are basically computers in print. These Knowledge of each disease incident must depend, are called Repertories (Boericke, 1972; Kent, 1986; therefore, upon one's ability to discern as much as Schroyens, 1993) and are basically dictionaries of sym- possible of its aetiology in broad terms and upon toms (in human terms) listed under 'generals', 'mentals' one's ability to read the symptoms/signs shown by the and 'particulars' (for each part of the body). A dedi- organism, i.e. its reaction to the disease influence. A cated veterinary version of the latter is in preparation. detailed effort must therefore be made in history-taking

Several remedies are then suggested (with scoring or and in clinical examination. The findings can be grouped weighting) that are able to cause the precise symptoms as follows:

sought and which may therefore be applicable in the

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particular patient. If one lists enough symptoms and

Conversion to veterinary application

looks up each in a Repertory, the highest scoring

Homoeopathic medicine was developed in humans for remedy (in theoretical terms at least) is the ideal choice.

humans and therefore presents some difficulties in

Resort to the Materia Medica will then help to confirm conversion to veterinary practice. The difficulties can be or negate this choice. Obviously, this is a fairly long-summarized as:

winded process and, although helpful in difficult cases, it is not applicable to rapid, on-farm selection of

- *Lack of speech in animal patients;*

remedies. Thus, for practical clinical application, a basic

- *Interspecies differences in reaction to remedies;*

knowledge of the remedies is required, together with a

- Lack of family history; and*

practised ability to think rapidly in 'picture' format. No

- Intensivism in farm management.*

equivalent veterinary Repertory yet exists although,

in slightly differing form, help can be found in books

Hahnemann (1814) did, however, advocate its use in

by MacLeod (1981, 1983), Brock and Nielsen (1986)

animals and so veterinary homoeopathy has a long

and Day (1984b, 1987b). Each of these works gives an

tradition.

easily assimilated guide to remedy choices under given

conditions, but (therefore?) is less accurate. Computer

programs are being developed for this type of work

Lack of speech

but, again, are not applicable to practical, on-farm use,

The lack of speech in animal patients leads to the loss

with the presently available technology.

of a large range of symptoms which would be of value

to the prescriber in human medicine. The ability to

Remove the obstacles to recovery

detail mental symptoms and feelings is lost, but much of what is needed in that respect can be discerned by

Even in the early stages of the twenty-first century, extrapolation from the demeanour and behaviour of the insight of Hahnemann in the eighteenth century the patient. However, the finer details of how the mind is remarkable. The single most exceptional example of is affected by the disease influence, or why it responds futuristic perspicacity is in the concept of 'removal of in the way it does, cannot be determined. Only the end obstacles to recovery'. In trying to observe this fourth result of behaviour is seen rather than the mental provision for cure, very modern ideas must be processes behind that behaviour. Since the mental embraced. Not only must there be obvious remedial symptoms of disease are a sure guide to the unique individual response to disease, by each patient, a large proportion of diagnostic (remedy-selecting) parameters is foreign body from the bowel, etc. but also, in the herd

therefore lost to the veterinary prescriber. However, context, the myriad parameters (environmental, management, dietary, etc.) affecting herd health must be the external behavioural signs, can provide an invaluable guide to the sensitive observer. Lack of speech also been practised by forward-thinking veterinarians for deprives us of the enormous field of subjective symptoms. The adjectives used by human patients to describe clear provision for it 200 years ago. Sadly, however, the pain symptoms are legion. They are again an expression of the individuality of each disease incident and are modern cattle farms will never provide truly ideal therefore invaluable. Descriptions of pain such as environmental and nutritional conditions for the 'tearing', 'cutting', 'burning', 'aching', etc. are all lost in animals, although meticulous attention to detail can veterinary medicine. Also hidden, among these symp-

approach the ideal. Thus, however much effort is
toms, is the headache in its many forms. We can rarely
applied, one is presented, in modern dairy cattle prac-
even diagnose headache in animals, let alone describe
tice, with an imperfectly fulfilled provision for real
it. Approximately 100 pages of Kent's Repertory (1986)
cure (Hahnemann's definition). This explains the need,
are devoted to headache, showing its importance to the
sometimes experienced on intensive farms, for repeated
prescribing physician.

medication (or possibly even continuous, in some of the
worst examples), but this must not be an excuse for
lack of effort. The unattainability of perfection must

Interspecies difference

not be an excuse for failure to achieve the best
conditions possible for the cattle nor for failure of

It is not unreasonable to suppose that different species
removal, to a maximum possible extent, of the obstacles
of animals will react differently to the medicines, when
to recovery.

compared with human reactions. There will also be dif-

ferences between the individual domestic species. To

Pathological

reconstruct the painstaking ‘provings’ of Hahnemann

To prescribe at the pathological level, one needs to be

and his followers, for each remedy for each species,

satisfied that the disease influence has evoked an acute

would involve several laboratories for many genera-

and easily identifiable disease process, that could be

tions, then only with an enormous usage of laboratory

easily matched to the actions of a particular remedy.

animals. This exercise would ethically be unjustifiable.

*The effects of traumatic injury, for instance, are very Despite the obvious
existence of such a problem, as*

well covered by the properties of arnica. The adminis-

shown by the varying toxicology and pharmacology,

tration of arnica, as rapidly as possible after the trau-

known in different species, for many poisons (Garner,

matic insult, will swiftly and effectively initiate a healing

1967), drugs (Daykin, 1960) and foods, homoeopathy is

process that will reduce the pain, shock, haemorrhage,

largely transferable en bloc. This is a fortunate fact but tissue fluid accumulation and resultant tissue damage

*one that should never be taken for granted. Thus the
that normally follows trauma. The net result is a happier
astute practitioner of the art will need to be aware of
patient, less distortion at the site of injury and, more
the possibility of failure due to species difference and
importantly, less disturbance to the circulation in the
will use pharmacological and toxicological knowledge
area. Therefore there is more effective and rapid
to advantage.*

restoration of normal tissue integrity and structure.

There is also a powerful and often underestimated

Lack of family history

antiseptic effect of this remedy.

The fact that one remedy covers this process is very

Although not strictly applicable to units where pedigree

convenient for the prescriber and very helpful to the

breeding and recording has gone on down the years, the

patient. It removes the need to apply the full rigours of

lack of family history of bovine patients, particularly in

the homoeopathic method, in times of emergency.

terms of disease, will lead to loss of some accuracy in

Furthermore, this pathological level of prescribing

prescribing. Known line susceptibilities are an impor-

provides a simple proving ground for homoeopathy to

tant factor in choosing remedies in such conditions as

the sceptic and a useful starting point for learning

cancer, mastitis, lameness, etc., but the loss of some of

the first steps of the homoeopathic method. Other

this material is not likely to be as deleterious, to effec-

commonly used remedy/pathological indication pairings

tive veterinary prescribing, as the lack of speech and the

are:

potential interspecies differences.

Hypericum:

injury to areas rich in nerve endings (e.g.

in digit, tail, etc.), injury to nerves, painful

Intensive farming

grazes, photosensitization

This factor has been discussed previously (see. p. 955).

Ledum:

puncture wounds

Aconitum:

sudden shock to the mind or body,

sudden disturbances of the body's equi-

Application

librium (e.g. sudden-onset fever, sudden

haemorrhage)

Disease can affect an animal to a greater or lesser depth

Calendula:

open wounds – antiseptic and promotes

and, therefore, so can the so-called remedies of

healing

homoeopathy, since they are also ‘disease-producing

agents’ when administered to a healthy body. These

different levels of prescribing can be classified as:

Local

(1)

Pathological

One would apply this level of prescribing when the

(2)

Local

nature of the disease is acute and fairly superficial, to

(3)

Organ-specific

an otherwise healthy animal with a robust constitution.

(4)

Constitutional

Examples of this application are: acute-onset mastitis

(5)

Facilitative /regulatory

(see p. 326) resulting from a chill, where aconitum is

(6)

Detoxifying

useful, New Forest eye infection (see p. 919), where

(7)

Historical

mercurius corrosivus may be effective, and foul-in-the-

(8)

Specific and

foot infection (see p. 426), where hepar sulphuris could

(9)

Preventive

*be the correct remedy. To prescribe effectively for these conditions, one only needs to take note of the local pre-
These terms require explanation, since their usage in
senting signs and match these to a remedy. One does
this context is peculiar to homoeopathy alone.*

not need, again, to delve into the deeper homoeopathic

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method, discussed in previous pages, in order to achieve

Aurum

Corresponding to a great

a good result. However, it is vital to ensure that the

Pulsatilla

many cows that suffer

disease is not chronic in nature or an acute exacerba-

Sepia

typically female or hormonal

tion of an underlying chronic disorder. If this should

problems as a rule

prove to be the case, satisfactory results will not follow

Antimonium crudum

Fitting those cattle that suffer

from employment of this simplified technique and one

Nux vomica

digestive or liver problems

would need to apply the constitutional approach (see

Lycopodium

below).

Calcarea carbonica

Matching those cows suffer-

Calcarea phosphorica

ing from lactation-induced

Organ-specific

Phosphorus

disorders

Organ-specific remedies may be used when one organ

These generalizations are too sweeping to be strictly

is particularly embarrassed in an illness, when it is

followed but the pictures of the remedies listed above

considered important to support that organ. Some

are suitable to be grouped in the way shown. These are

remedies have a particular affinity for specific organs or

‘big’ remedies (Hahnemann’s so-called polycrests) and

tissues and the most important are:

have wide-ranging effects on all parts of the body. They

Rhus toxicodendron:

muscles

cannot, therefore, be type-cast purely in the roles shown

Ruta graveolens:

tendons,

ligaments,

joint

above, but the groupings are useful nonetheless. The

capsules,

aponeuroses,

groupings represent trends in the relevant animal's

periosteum

disease response patterns and are very useful for short-

Hypericum:

nerve fibres

listing of remedies. These and other useful remedies will

Symphytum:

bone

be discussed later (see pp. 1088–1090), in fuller detail.

Nux vomica:

liver

In the case of a patient affected by chronic disease,

Flor de piedra:

liver

no real cure can follow treatment unless this method of

Lycopodium:

liver

prescribing is followed. The organism has learnt, in the

Berberis:

liver, kidney

case of chronic disease, to live in a state of uneasy

Kali chloratum:

kidney

harmony with the disease. The animal is therefore

Digitalis:

heart

unable to regain health, often lost a long time previ-

ously, unless the deep, powerful and most appropriate

stimulus of true holistic constitutional homoeopathy is

Constitutional

prescribed.

The concept of a 'constitution' embodies everything that is unique to an individual organism. It embraces

Facilitative/regulatory

the idea of the 'programmed' nature of each body's

The facilitative level of prescribing exploits the ability

response to a disease influence. Another way to con-

of certain 'potentized' substances to facilitate metabo-

sider this is to think of predetermined tendencies in

lism with respect to those substances. Examples are the

each individual. Response to disease is determined by

use of 'potencies' of calcium phosphoricum to facilitate

a number of factors: genetic make-up inherited from

metabolism with respect to calcium and phosphorus,

the parents, in utero influences during pregnancy, the magnesium phosphoricum to help magnesium metabo-birth process, postnatal influences, influences during

lism, and cuprum metallicum to assist copper metabo-

the growing period and influences during adulthood. In

lism. It is well known that so-called mineral deficiencies

the farm context, one can see how breeding, pregnancy,

are rarely absolute deficiencies but can be more often

calving, rearing and general management or dietary a malfunction of the relevant absorptive, metabolic regimens can affect the make-up of an adult cow. By and eliminative processes for that mineral. Cases of reading the response pattern of the animal (signs and 'relative' excesses and deficiencies can both be helped symptoms) in the face of disease or situations, and by by such methods. In the case of a hormone disturbance, studying her conformation and behaviour, one can for- a 'potency' of the hormone may help regulate and mulate an idea of her 'constitutional type'. This whole balance the body with respect to that hormone.

picture can be matched to a whole remedy picture, as previously discussed. Not many remedies have a com-

Detoxifying

bination of wide enough or deep enough effects on the body to rank as constitutional remedies and, in the The detoxifying method of prescribing echoes very world of cattle, the major nine examples can be listed, closely the preceding remarks. Should a specific sub-conveniently classified into three groups:

stance be causing a toxic effect, e.g. alkaloid, metal,

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*chemical, etc., then the body's natural ability to break
cific disease, there will be no remedy specific to the
down and/or eliminate such toxins will be enhanced by
disease but a whole host of possible remedies, the most
the administration of the specific substance, or a close
suitable of which must be selected for each patient.*

relative, in 'potency' (homoeopathic dilution). Exam-

Having said this, it is possible that baptisia could be spe-

ples are the use of plumbum in cases of lead toxicity,

cific to salmonellosis (Chapter 15), mercurius cyanatus

coumarin or melilotus in cases of warfarin toxicity,

to calf diphtheria (see p. 250), mercurius corrosivus to

opium or nux vomica in cases of anaesthetic toxicity or

New Forest eye or even borax to foot-and-mouth

even of carbohydrate overdose in the ruminant. Such

disease (see Chapter 43(c)) (in those countries in which

methods have often been shown to produce unexpected

it is permitted to treat this disease).

results, even in extreme cases of toxicity.

Preventive

Historical

In herd medicine it is very important to apply preven-

The historical method of prescribing utilizes the body's

tive principles, in order to minimize or to obviate the

ability to respond to an appropriate curative homoeo-

risk of spread of a known or predicted infectious

pathic stimulus, long after the initial disease-producing

disease within the herd. This is the basis of effective

influence has subsided, should the body still be suffer-

veterinary involvement on the modern bovine farm

ing from the effects of that influence. Examples are:

unit. In an individual animal context, it is important to

using arnica long after an initial traumatic insult, should

prevent disease occurring in the face of a predictable

the body still be suffering disease from that injury; using

challenge or disease-producing event.

aconitum long after a mental or physical shock, if the

The greatest demands on the veterinarian using

body is still suffering effects or fears produced by that

homoeopathy are on a herd basis. Where infectious

shock; using a 'specific' remedy (see below) long after disease has either entered the herd, in which case the a specific infectious disease has passed its acute phase, healthy individuals must be protected, or is threatening should the body still be suffering effects from that the herd, in which case the whole herd must be protected.

tected, one would select for prevention a specific

All that is necessary, in order to utilize this method,

remedy. This would generally be a nosode (see Specific is to detect, in a chronic disease situation, a facet of the

prescribing, above) although it could be any remedy

history that leads one to believe that the current disease

from the homoeopathic Materia Medica that the pre-

has its origins in a specific historic incident. One can

scribe feels specific enough to a certain disease (see

then treat for that incident, as if it were in the here

Prescribing guide, p. 1091). In conventional medicine

and now, even though it may have occurred years

one would be reaching for a vaccine in this context, but

previously.

in the case of some infectious diseases, no effective vaccine is available or no vaccine exists at all. Nosodes may be considered as if they were vaccines, if it helps

Specific

the reader to understand the principle of their applica-

The specific method of prescribing would usually be tion in this context. However, they do not produce any used in an acute infectious disease context, where a antibody response in the treated animal, due to the known specific infectious disease agent is involved.

extreme dilutions in which they are usually used (30c).

Generally speaking, this method of treatment would

Neither do nosodes carry the potential hazards that imply use of nosodes (remedies made from disease vaccination sometimes can, e.g. strain 19 Brucella material). Examples are: mastitis nosode in cases of abortus vaccine (Day, 1986, 1987b).

mastitis (see Chapter 23), infectious bovine rhinotra-

These preventive remedies, in order to be swiftly,

cheitis (IBR) nosode in cases of IBR (see p. 286),

effectively and easily applied to the whole group at risk,

bovine virus diarrhoea (BVD) nosode in cases of BVD may be administered via the drinking water. Water (see p. 853) or salmonella nosode in cases of salmonellosis. In the acute phase of an infectious disease, it may be large enough or well enough supplied with water, so as to be dangerous to use the nosode in an attempt to not to run dry on the day of dosing. The quantity of produce a cure. For this reason, this form of therapy is remedy used does not appear to be very critical but an usually reserved for use during the recovery phase of easy guide is to use 5 ml per drinking trough, unless the the disease, in order to prevent any tendency for the trough is unusually large or small. Frequency of dosing disease to become chronic. During the acute phase it is would depend upon the estimated properties of the always better to select an appropriate homoeopathic challenge. In an enzootic disease situation within the remedy for cure, according to the Law of Similars in the herd, the frequency would be selected corresponding to usual way. This implies that, in each case of acute spe-

the estimated violence and virulence of the disease and

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according to the animals' estimated ability to withstand

(2)

To select a combination of remedies for the group.

it. At times of high risk, when the disease agent is

This will allow the incorporation of the spectrum

perhaps given the upper hand by management factors

of signs shown by the different individuals. A

or by climate or the animal is compromised by similar

maximum of three different remedies should be

factors, then one would dose more frequently. In the

incorporated.

case of assumed risk from an epizootic disease, i.e. a

(3)

To select a number of different remedies

disease not yet met by the herd, dosing could be more

(maximum of three) to stimulate the individuals

sporadic.

at different levels, as in the foregoing section. A

The second sphere of use of the preventive technique

*‘constitutional’ remedy, say, could work alongside
is in the case of the individual animal, which may be
a ‘specific’ remedy and a ‘local’ prescription.*

threatened by a predictable disease challenge. Thus
(4)

*A single constitutional prescription may be pos-
sible, especially if the group all come from one
used to help prevent problems (Day, 1985, 1987b), or
family or from one breeding line.*

for an anticipated stressful journey, aconitum could be
(5)

*A specific or nosode prescription could be
used in advance of the challenge. Incidentally, aconitum
selected, as in the above section. This can be very
can be used on a group or herd basis, in order to prevent
successful in the case of a known infective disease
the production losses consequent on herd stress, such as
and lessens the need for more detailed homoeo-
the periodic tuberculin test.
pathic knowledge or skill.*

Thus it can be seen that the application of the

(6)

The group could be ‘repertorized’, as if it were a homoeopathic method has come a long way, since it was single animal. The technique would be as outlined first devised in humans some 200 years ago. Although in the section describing how to match the remedy the rigorous and in-depth diagnostic and prescribing to the disease. This is an advanced technique and processes may daunt the novice, the classification into should not be tackled without experience.

different levels at which the method can be applied in The reference given describes how these techniques are bovine medicine makes it easier to start out. They compatible with proper homoeopathic philosophy and provide scope, for even the newly initiated and hesitant principles.

prescriber, to test the principles of homoeopathy and its efficacy at an easier level, without immediately being committed to long-term study or to large and therefore

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expensive stocks of medicines.

There follows a brief summary of the properties of some of the homoeopathic remedies most commonly

The challenge of herd treatment

used in a farm context and some of their possible clini-

Much has been made, in the foregoing pages, of the

cal applications. This is not intended to be an exhaus-

need to individualize therapy for best results. This high

tive source on the subject but an introduction to these

ideal is frankly not practical in a herd or group situa-

30 useful remedies. Fuller works on this material can be

tion. If we consider, for instance, a group of calves suf-

found in the Further reading section. Remedies are pre-

fering from pneumonia, treating each on an individual

sented in alphabetical order for simplicity, not in order

basis is not possible. What is needed is a modification

of importance nor grouped according to possible appli-

of principles, while maintaining the essence of homoeo-

cation. The way in which these remedies are presented

pathic philosophy and methodology. This short section

shows the emphasis placed upon the ‘picture’ presented

can only be a summary of the techniques involved, in by the disease rather than upon its usual name. Rem- order to show the options. Clearly, homoeopathy will edies are taken from the plant kingdom, animal not be a practical therapy on the farm if it is not adapt- kingdom and minerals and are available in tablet, able to herd applications.

pillule, crystal, powder, tincture, injection, cream, oint-

A detailed account of how this can be done is to be ment or lotion forms. The thirtieth centesimal (30c) found in the Further reading section (Day, 1999). In potency is a useful potency of most remedies, for the essence, there are six main possibilities:

purposes of beginners.

Aconitum napellus: this is a remedy suitable for use in cases (1)

To choose a single similimum (a homoeopathic

of sudden shock (physical or mental in nature), sudden-onset

medicine which is similar to the signs and symp-

fevers, disorders from chilling and cold winds, and in cases of toms displayed) for the entire group. This will only

profuse bright red haemorrhage.

be properly effective in an epizootic situation, in Affected animals tend to shudder in response to fear or which all individuals are likely to be similarly shock, display a rapid pulse, run a fever and suffer conjunctivitis with fluid lacrimation and nasal discharge. Symptoms affected.

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tend to centre on the chest, so use in 'shipping fever' is an in movement and responses, difficult to handle and more obvious application (see p. 286).

fearful.

Antimonium tartaricum: this remedy is particularly suited *Carbo vegetabilis*: abdominal distension by gas, flatus, to respiratory signs. Indications are mucoid, rattly breathing flatulent colic, weakness of circulation and musculature, poor or coughing, frothy saliva and a tendency to cyanosis. Animals resistance to infection and even collapse characterize this

display minimal thirst and a rapid, weak pulse. Cold, damp remedy. Its main application is in the treatment of the cold, weather aggravates the condition and affected animals are collapsed individual, in whom its results can be spectacular.

unwilling to lie on their sides. This remedy is one of several It has been dubbed the 'homoeopathic corpse reviver', on

useful pneumonia remedies and should be considered in cases

account of its prowess in such cases.

of fog fever (see p. 866).

Caulophyllum thalyctroides: one of the Native North

Apis mellifica: it is useful in cases characterized by oedema.

Americans' so-called 'squaw-root' remedies, it has great appli-Urine retention, pulmonary oedema, oedematous swellings of

cation in all disorders of labour, at any stage (Day, 1984a,

vulva/perineum or udder, ascites, cystic ovaries, etc. may all 1985). It also has an effect on shifting lamenesses, particularly respond if concomitant signs agree. The patient is usually

if the origin is in the small joints.

thirstless and prefers cool open air. Cold bathing of affected Colocynthis (genus: Cucumis or Citrullus): this remedy areas produces comfort.

is of particular use in colic. The abdomen is distended and the Arnica montana: this is a remedy of use in all condi-back arched. Animals will roll and kick at the belly in cramp-

tions arising from trauma. Bruising, haematoma formation,

like paroxysms.

pain, swelling, shock and even resultant local infection will

Hepar sulphuris: if any homoeopathic remedy could be said respond to treatment. Patients often display a fear of being

to fill the role of antibiotics in septic conditions, then this is touched.

the one. Suppurative processes in their early stages respond

Arsenicum album: this remedy is a polycryst and therefore well. In suitable cases, there is always great sensitivity to pain, of constitutional importance.

Restlessness, chilliness and thirst with relief from warm applications. Pain is aggravated by the

characterize the patient. The remedy can be useful, on a more

least touch. Joint-ill and navel-ill usually respond (see pp. 249, local basis, in cases of diarrhoea, dehydration and collapse, if 255).

concomitant signs agree. Diarrhoea is usually profuse, watery

Hypericum perforatum: injury to nerves or to areas rich in and offensive-smelling.

nerve endings is an indication for this remedy. It has a very

Belladonna (genus: Atropa): a 'fever' remedy, this is useful rapid healing effect, both on the pain and on the physical

in cases where there is an acute febrile or inflammatory state damage. Not surprisingly (on account of modern knowledge

characterized by heat, redness, swelling, fullness and pain. The of the physiology involved and of the symptoms) the lesions

pulse may be full and bounding. There are often delirious or

and pain of photosensitization also respond well (see p. 884).

convulsive signs too. The animal is usually thirsty and displays Lycopodium clavatum: a polycrest, this remedy has an

a dilated pupil. Symptoms tend to centre on the chest, making

application constitutionally. With an affinity for the liver, the a useful distinction from the usual pattern of aconitum.

remedy is often used in this context. Possible digestive distur-Bryonia alba: this remedy has an affinity for serous membance, dry, withered appearance to the skin, tendency to flatus, branes. All suitable conditions are worsened by movement, so

abdominal distension, liver dysfunction, dyspnoea, tachypnoea, movement of the nostrils with every breath and an

A major sign in the affected animal is unwillingness to move.

Affected animals are thirsty for long cold drinks. Guided by overall anxiety distinguish this animal type in disease. Probably these three points one may prescribe confidently in cases of pneumonia, mastitis, arthritis, peritonitis, etc. The pneumonic water to drink (if offered).

lems are often right-sided and the animal prefers warmer

calf will stand still, despite its fear, and will tend to lie on its Mercurius cyanatus: a thirsty patient with a paradoxically affected side to prevent movement. The mastitic cow would

wet mouth and possibly even drooling saliva may require a

allow herself and her hot swollen painful udder to be

mercury-type remedy. Mercurius cyanatus patients display examined, rather than move away.

offensive breath, offensive-smelling ulceration of mouth and

Calcareo carbonica:

throat and swollen glands. The picture fits that of calf

another polycryst, this remedy has con-

diphtheria.

stitutional applications. Disorders of production are an indi-

cation, e.g. fertility, lameness, mastitis. The animal has a heavy Mercurius

solubilis: as with all mercury remedies, there is a skeletal structure, large limbs, large joints and large feet. It tendency to thirst, to profuse salivation, to offensive breath, to usually has a good condition score and is generally peaceful

swollen glands in the throat and to ulceration of mouth, bowel but dominant. Movements tend to be unhurried. The appetite

or teats. Ulcers will discharge pus. If there is diarrhoea, there is very good and, if a milking cow, the milk yield is high. There will also be a degree of tenesmus. Since the remedy is a poly-animal is susceptible to chilling and usually shows catarrhal-

crest, it has constitutional applications. The preceding disease type responses from the mucous membranes.

tendencies in a dominant, usually heavy, animal are an indication for its use.

Calcarea phosphorica: a polycryst, this remedy is suited to similar conditions to *calcarea carbonica* and the typical *Nux vomica*: as a polycryst, it has wide application in a con-animal is similar, although lighter in skeletal structure, quicker constitutional context in the bovine. Patients tend to be dull and

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sluggish in movements, with paradoxically rapid or violent

teum, this remedy is a powerful aid in the treatment of sprains reactions when disturbed, bad tempered and thin. They suffer

and other damage to such tissues and regions of the body.

digestive complaints, characterized by cessation of rumen

Sabina (juniperus): sabina's main sphere of activity is the function and a tendency to constipation, having hard knotty

uterus, where bloody discharges and lack of tone may respond.

stools. Some cases of 'acid' or fermentative diarrhoea may

It is not indicated where sepsis exists. Retained fetal membranes respond and complaints following from overeating of con-

centrate will respond well. Conditions tend to be worse in the morning and worse for disturbance or chilling (Mahé,

Sepia: a polycryst, *sepia* has great value as a bovine consti-1987).

tutional remedy. It is predominantly of use in a female

context. Lack of tone and lack of tautness typify the condi-

Phosphoric acid: in common with all the 'acids', phosphoric tions and the constitutional type suited by this remedy. The

acid is indicated in cases of general weakness. More specifi-

animal takes on a worn-out, tired, sagging appearance and so,

cally there is debility, dehydration, pale flatulent painless

also, do individual body regions, e.g. uterus/perineum, limbs, diarrhoea with a dry mouth, thirst and possibly a degree of

udder, etc. Venous congestion and tendency to prolapse are jaundice.

also characteristic. Fertility problems may respond, if con-

Phosphorus: because of its extent of action, it is a polycrystitutional aspects agree.

and therefore of great value in a constitutional context. *Phos-Silica*: Better known for its ability to stimulate expulsion of phorus conditions are

characterized by sudden onset and by foreign bodies from the tissues or to awaken chronic low-their serious nature, e.g. sudden-onset pneumonia, sudden grade inflammatory lesions, silica is also a great constitutional haemorrhages, hepatitis, haematogenous jaundice, etc. The remedy. Structural weakness and distortion, whether of skeleton or feet, with a poor immune response, typify the animal averse to separation from the rest of the herd and apt to panic. able to respond to the remedy.

The type is of lean build and averse to handling. Conditions are worse for touch and change of weather.

Thuja occidentalis: The reputation of this remedy rests mostly on its action on papillomatous lesions (p. 882), espe-*Phytolacca americana*: this is the remedy above all others cially when they occur on neck or abdomen. Ill-effects of

with a reputation in mastitis. This reputation can lead to vaccination also may respond. The constitutional and other overuse in this context, as it will only produce results when effects of the remedy are not so important in cattle work.

used in the correct homoeopathic manner. Lymph glands and mammary glands become indurated swollen and painful.

Urtica urens: The lesions of nettle rash are well known to There is usually heat too. Milk becomes thickened, stringy and everyone, as is the pain that follows.

The remedy is of value in yellowish. Cold wet weather aggravates the symptoms.

such conditions. Apart from these properties, the remedy has

the ability, in high potency, to promote milk flow and, in low Pulsatilla nigricans: this is a common constitutional type in potency, to induce suppression of milk. This dual regulatory

the bovine, particularly in the case of heifers. The type is shy, power gives the remedy great value in bovine medicine.

feminine in appearance, suffers catarrhal complaints and has

Lesions respond favourably to warm applications and nega-

little thirst. Breeding problems respond well, if it is used in tively to cold applications, so too does the mastitis helped by the correct constitutional context. Catarrhal syndromes of

urtica.

respiratory tract, udder, reproductive tract, etc. are typical.

The animal prefers fresh, open air and symptoms are aggra-

It is hoped that the foregoing brief account of 30

vated by warm, stuffy atmospheres and in the evening.

remedies will allow a start to be made in homoeopathic

prescribing. It cannot be expected that, in so few pages,

Pyrogenium: fevers, particularly those of a septic nature, it will provide a comprehensive knowledge of the reme-respond well to this remedy, especially if pulse and tempera-

ture are not in agreement. In suitable cases, there is a tendency dies. The reader must also realize that these few reme-to toxaemia, offensive secretions, dark blood and offensive,

dies, although forming a good nucleus of prescribing dark-coloured, septic lesions.

material, actually represent only a fraction of what is available. For each condition or syndrome mentioned,

Rhus toxicodendron: American poison ivy (the source of a great many other remedies have a potential value,

this remedy) produces, and is therefore able to help, severe

should they prove to have properties 'similar' to the

pruritic conditions or conditions characterized by rheumatic

or arthritic stiffness and pains with great thirst and an aggra-

vation from cold, wet or damp conditions. Skin lesions are

often vesicular in nature, resembling cowpox lesions. Muscu-

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loskeletal problems are further characterized by an aggrava-

tion when moving from rest, with a subsequent 'loosening' and

There follows a small vade-mecum, showing some indi-

easing of the stiffness and pain. Suitable diarrhoea cases

cated remedies that may prove effective in the named

display dysentery and tenesmus.

syndrome. Please be reminded that true homoeopathy

Ruta graveolens: with an affinity for fibrous structural does not rely upon the name of a condition for remedy

tissues such as ligaments, tendons, joint capsules and perios-
selection but upon the similarity between disease

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picture and remedy picture, on as many counts as pos-
position of confidence. Books giving a greater depth
sible. The following list only serves as a pointer to some
of homoeopathic knowledge are listed in the Further
commonly indicated remedies. Final selection between
reading section and accredited courses in Britain, on
those remedies mentioned (and many not mentioned)
veterinary homoeopathy, are conducted under the aus-
rests with the prescriber. All the remedies mentioned
pices of the Faculty of Homoeopathy, 15 Clerkenwell
below appear in the preceding section, so that section
Close, London EC1R 0AA. The Faculty has an exami-
can act as a brief guide to help selection of the most
nation structure, for postgraduate qualifications. On
relevant remedy. (Remedies may be given orally, by
an international basis, the Faculty does help courses in
injection, topically or orally via the drinking water.)
other countries, and the International Association for

Veterinary Homoeopathy both runs courses and sets

Acetonaemia (p. 793)

Lycopodium, nux vomica

examinations for veterinary homoeopathy.

Arthritis (pp. 454–458)

Bryonia,

caulophyllum,

phytolacca, rhus tox., ruta

graveolens

Acupuncture

Bloat (p. 832)

Carbo veg., lycopodium, nux

vomica

Acupuncture is a science of energy medicine, having its

Calf diphtheria (pp. 250,

Mercurius cyanatus

origins in ancient China anything up to 4000 years ago.

822)

The oldest medical textbook known, the Huang Ti Nei

Constipation

Nux vomica, sepia

Jing Su Wen (Veith, 1972), may have originally been

Convulsions

Belladonna, nux vomica

written as long ago as 1000 bc. Its origins are confused,

Dystokia (Day, 1984a,

Caulophyllum, pulsatilla

since it has undergone many alterations and commen-

1985)

taries since it was first written. (In fact the Su Wen was Fever

Aconitum,

belladonna,

possibly a later addition to a scaled down Nei Jing.) This pyrogenium

book has formed the basis of traditional Chinese

Foul-in-the-foot (p. 426)

Hepar sulphuris

medicine theory down the centuries, but there have

Haemorrhage

Aconitum, arnica, phosphorus

been myriad adaptations and variations applied since

Infertility (Chapter 36)

Apis mell., aurum, pulsatilla,

*its inception, giving rise to traditional Chinese medicine
sepia*

*as we know it today. Acupuncture is but a component
Injury (Chapter 32)*

*Aconitum, arnica, calendula,
of this, Chinese herbalism also forming an integral part,
hypericum, ledum, rhus tox.,
along with moxibustion (the application of heat). The
ruta grav., symphytum*

*practice of traditional Chinese medicine is truly holis-
Joint ill (pp. 249, 255)*

*Hepar sulphuris
tic, in that it embraces an understanding of the part*

Ketosis (p. 793)

*See Acetonaemia
played by diet and lifestyle in the health of the individ-*

Mastitis (Day, 1986)

Aconitum,

belladonna,

*ual and it makes a positive effort to optimize these, as
(Chapter 23)*

bryonia, carbo veg., hepar

an integral part of therapy.

sulphuris,

mercurius sol.,

The Chinese have long treated animals with acupunc-

phytolacca, silica, urtica

ture, so our veterinary use of this in the West is no new

Metritis (Chapter 34)

Caulophyllum,

pulsatilla,

thing. Dogs, cats, horses, cattle and other farm animals

pyrogenium, sabina, sepia

can be treated. In countries other than Britain, working

Navel ill (p. 249)

Hepar sulphuris, silica

animals such as elephants, llamas, donkeys, camels and

Papillomatosis (p. 882)

Thuja

buffalo are patients. In the UK, acupuncture is much

Placenta (retained)

Caulophyllum, sabina, sepia

*more widely used on dogs and cats than in horses, and
(Chapter 34)*

*more in horses than in cattle. This is more a function of
Pneumonia (Chapters*

Aconitum, antimonium tart.,

*the pattern of specialization in the veterinary profes-
17, 49)*

bryonia, lycopodium, phos-

*sion and that more interest has been shown by those
phorus, pyrogenium*

veterinarians who are in small animal practice, than a

*This list is far from complete and is aimed at pro-
reflection of the efficacy and value of acupuncture in
viding an insight and a starting platform, for deeper
cattle. Cattle respond very well to the methods but
homoeopathic prescribing and learning.*

chronic disease is less a feature of bovine medicine than

*Having read this guide to the principles of
in the other species. This means that there is less call for
homoeopathy and having tried out some of the
a specialist second opinion than in equine or pet med-*

methods and suggestions contained in this chapter, it is
icine. The call for acupuncture help in cattle is reduced,
hoped that the reader will go on to further study and
compared with the other species, since the species is
will venture into more ambitious prescribing, from a
kept more on a financial (productivity) basis than for
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emotional, competitive or recreational reasons. The
of traditional acupuncture and encourage the diligent
reality is that there is less perceived value in the indi-
student to look further into the subject.

vidual as a result.

Acupuncture is a system of internal medicine

Theory

devised, in ancient times, purely from the outside of the
body. In ancient China, the human body was not to be
The theory of the science of acupuncture is based upon
violated, either in life or in death, so post-mortem
concepts of energy and forces which are rooted in Taoist
examinations and anatomical or physiological studies
philosophy. In this there exists no linear thinking, no

were not performed. The basis of the system is the cause and effect. The order and pattern of life (and relationship of points on the surface of the body to therefore disease, which is a part of life) and of the uni- the internal organs and their integrated functions. The verse are governed by principles of inevitability. Con- mechanism of the relationship is not clear to Western cepts are not placed one under the other in a deductive science and is unquestioned in the East, where such a sequence or subsumption, but side by side in a picture. different philosophy of life and living pertains as to be Events follow each other, not by cause and effect but incomparable. Clearly, as far as some of the acupunc- by a kind of inductance. Light does not cause darkness ture points are concerned, there is a cutaneous–visceral and vice versa, one becomes or leads to the other reflex at work but this does not satisfactorily explain inevitably. Similarly, the concept of light cannot exist the mechanism of acupuncture, any more than does without a concept of darkness, nor the concept of good the discovery of the release of endorphins or opioids

without that of evil.

in response to treatments.

Our Western way

It has been said of Taoism that it does not deny of looking at life leads to a linear thought process, reason, but that it remains just beyond its grasp. It is trying to establish 'cause and effect', and an attempt a concept of balance, of rhythm, of sustainability, of to rationalize, classify and compartmentalize in a way harmony. It describes a complex web of interrelation- that does not sit well with the oriental philosophy and ships, in which the whole is dependent upon and method.

affected by its parts and in which each part is depend- The Chinese discovered, empirically, a collection of ent upon and affected by the whole and each of the points (some 700 forming the basis of the system) that other parts. There is an essential understanding of flux, related to bodily function and to disease patterns or of dynamism and of interconnectedness.

pictures. In order to explain the indisputable facts and

The two forces that maintain the universe, and the life verifiable correlations so discovered, they wove a web within in it, are called Yin and Yang and represent of philosophical concepts, in harmony, of course, with philosophical polarities. They are the eternal opposites. their philosophy of life (so turning an empirical art into They are aligned to other pairings of polarities, ranging a science). In ancient China, and still today, medical lore, over the whole of our existence. Examples are, respectively, dark and light, cold and hot, female and male, low and high, sluggish and fast, lower and upper, night and day, soft and hard, earth and heaven, light and heavy, considered as separate entities. This chapter does not argue which view may be the more correct, valid or water and fire, weak and strong, under- and overactive, healthy, but merely presents comparisons. ity, humble and exalted, front and back (more logical in

The philosophical lattice work, developed so long quadruped terms than in the human biped, i.e. ventral ago, is not there to confuse the would-be student but to and dorsal, in that dorsal is higher, therefore reexplain and elucidate the empirical findings. This is not senting Yang) and so forth. It should be becoming clear apparent to the sceptical Western observer, who will that these two polarities act both simultaneously and undoubtedly find the concepts not only confusing but antagonistically within the universe (and within its amphigoric. Atavism and Taoism are indigenous to microcosm, the body). There is, however, no absolute in the oriental way of thinking and are totally foreign to life and so with Yin and Yang. There is no Yin without us. However, acupuncture was devised in the East and Yang. They are forever transforming into one another, developed in the light of oriental concepts. It must be on an almost imperceptible basis. Nothing is all Yin, taken or left at face value, at present, until some more nothing is all Yang. All systems are composed of Yin readily assimilable theory may be formulated that can

and Yang. Within Yin there is Yang and within Yang explain the observable phenomena as well as the origin there is Yin. Within any given Yin/Yang system under nal. This development is not in the offing. It is hoped discussion, there is a further yin/yang system and, within that by presenting a rapid overview of the theory and that, another. This applies over and over, ad infinitum. philosophy, it will not confuse the reader but will serve Zou Yen, the philosopher of the third century bc, to give an introduction to the principles and methods observed: ‘ Heaven is high, the earth is low, and thus they

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Table 65.1

The paired (tsang/fu) organs, relating to Yin and Yang.

Yin (tsang organ, ventrally/

Yang (fu organ, dorsally/

medially-routed meridian)

laterally-routed meridian)

Heart

Small intestine

Kidney

Bladder

Pericardium

Triple heater

Liver

Gall bladder

Lung

Large intestine

Spleen/pancreas

Stomach

Fig. 65.2

The Taoist T'ai Chi T'u symbol.

are fixed. As the high and low are thus made clear, the energy on the point of materializing. They did not honourable and humble have their place accordingly. As trouble to define, merely to observe its function. It pervades the entire universe, so is not simply life energy. and weak are thus differentiated. . . . Cold and hot The Qi of the body is called normal Qi or upright Qi. season take their turn . . . and Heaven knows the great

This is derived in equal parts from each parent, as the beginning, and Earth acts to bring things to completion.

original Qi or conceptual Qi. It is nourished and replenished—Heaven is Yang and Earth is Yin’ (Kaptchuk, 1983).

ished throughout life by grain Qi and air Qi, which are embodied in the consistencies and (apparent)

combined with each other and with normal Qi, to permeate the entire body. inconsistencies of the oriental approach and there is

no way to determine the beginning or the end. The Chinese theory also gives the ultimate power of life classic ‘chicken and egg’ situation holds, and one

to the kidneys, in that the kidneys store the Jing. This can only observe the continuum/perpetuum. The Taoist

is an undifferentiated ‘substance’, which is most closely

T’ai Chi T’u symbol (Fig. 65.2), more correctly a sphere associated with life itself. It contains the potential

rather than a two-dimensional object, is the tradi-

tion for birth, maturation, decay and death. It supplies the

tional and elegant attempt to represent this concept

potential for differentiation into Yin and Yang, in other

diagrammatically. In this yin-yang model, the dark

words, the capability of life. Since all organs require

area represents Yin and the light area, Yang. The dots
Jing, the kidney bestows the gift of life on each organ.
represent yin-within-yang and yang-within-yin. As the
Since Jing also controls conception, future life is gifted
sphere turns, so the interplay of yin and yang are
by the kidneys. The anatomical relationship of the
illustrated.

gonads to the kidney would have escaped the ancient
Yin and Yang act together and antagonistically within
Chinese. Jing gradually diminishes in quality and quan-
the body and are components of the life energy – Qi
tity with time, so ageing is controlled in this way. Death
(pronounced ‘chee’). This energy is said to circulate
is inevitable when there is not enough Jing to maintain
continuously and rhythmically (to a circadian rhythm)
life. It is said ‘The kidneys are the root of life, the resi-through twelve paired (left
and right) channels. These
dence of Yin and Yang, the channel of life and death’.
channels are often also called meridians. The correct
This makes a lot of sense in Western terms, since the
balance and harmony of Yin and Yang within the body

kidneys, once damaged, cannot regenerate.

and the even and regular circulation of energy (Qi)

The channels are related to organ function, thus the

within the channels maintain perfect health. The con-

twelve meridians, or channels, have names indicating

verse applies: an imbalance of Yin and Yang and

these relationships. The organs, to which they relate, are

an interrupted flow of energy will imply disease. Thus

each said to be either Yin or Yang in nature and, need-

disease is considered to be more Yin or more Yang,

less to say, there are six Yin and six Yang organs/chan-

relative to the state of health. This can occur as a rela-

nels (Table 65.1) in order to maintain balance. The Yin

tive deficiency of one or the other, or as a relative excess

organs are the solid ‘ Tsang’ organs, the Yang organs are of one or the other.

Each would produce an apparent

the hollow or ‘ Fu’ organs. Each Tsang organ is related excess of one over the other. There is unlikely to be an

to a Fu organ, e.g. (respectively) kidney is related to

absolute deficiency or excess, since life could not then

bladder.

be supported.

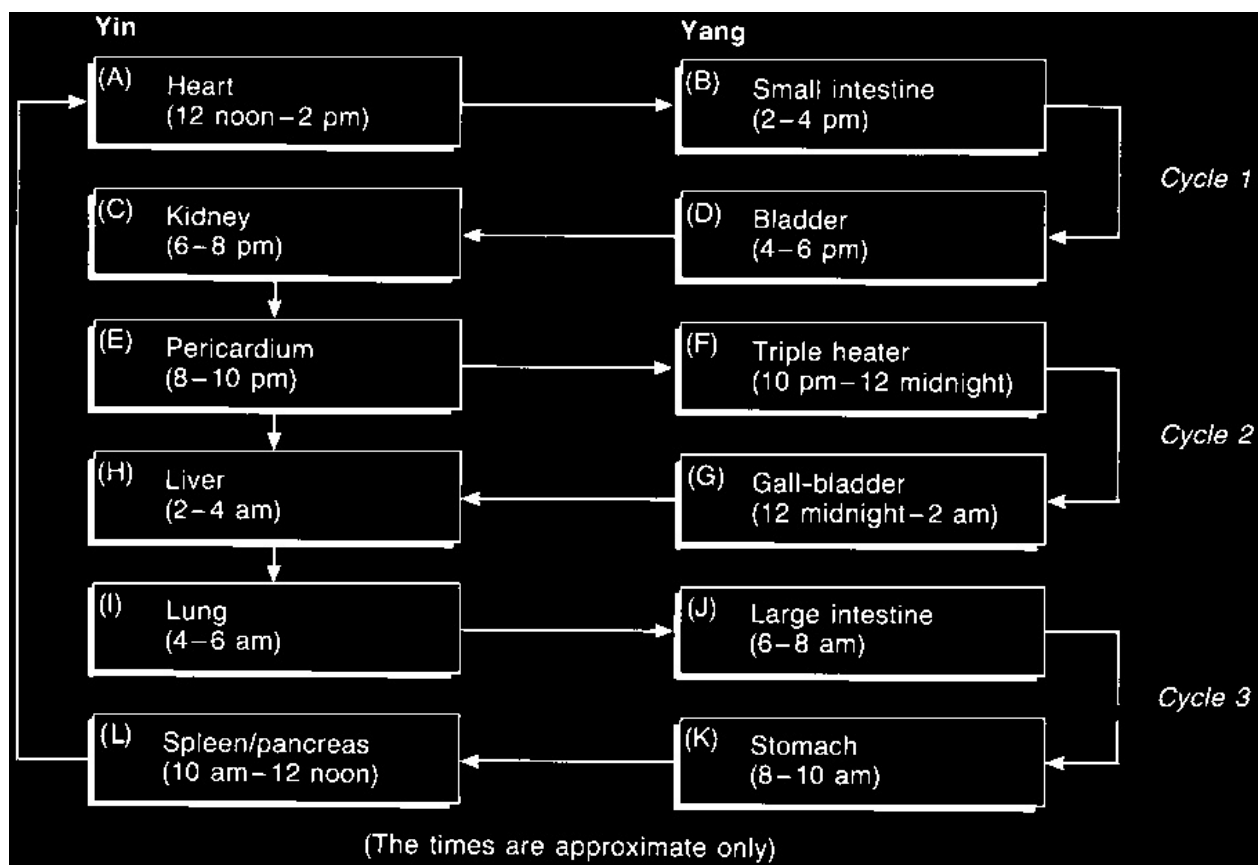
The energy (Qi) dwells in any single channel for two

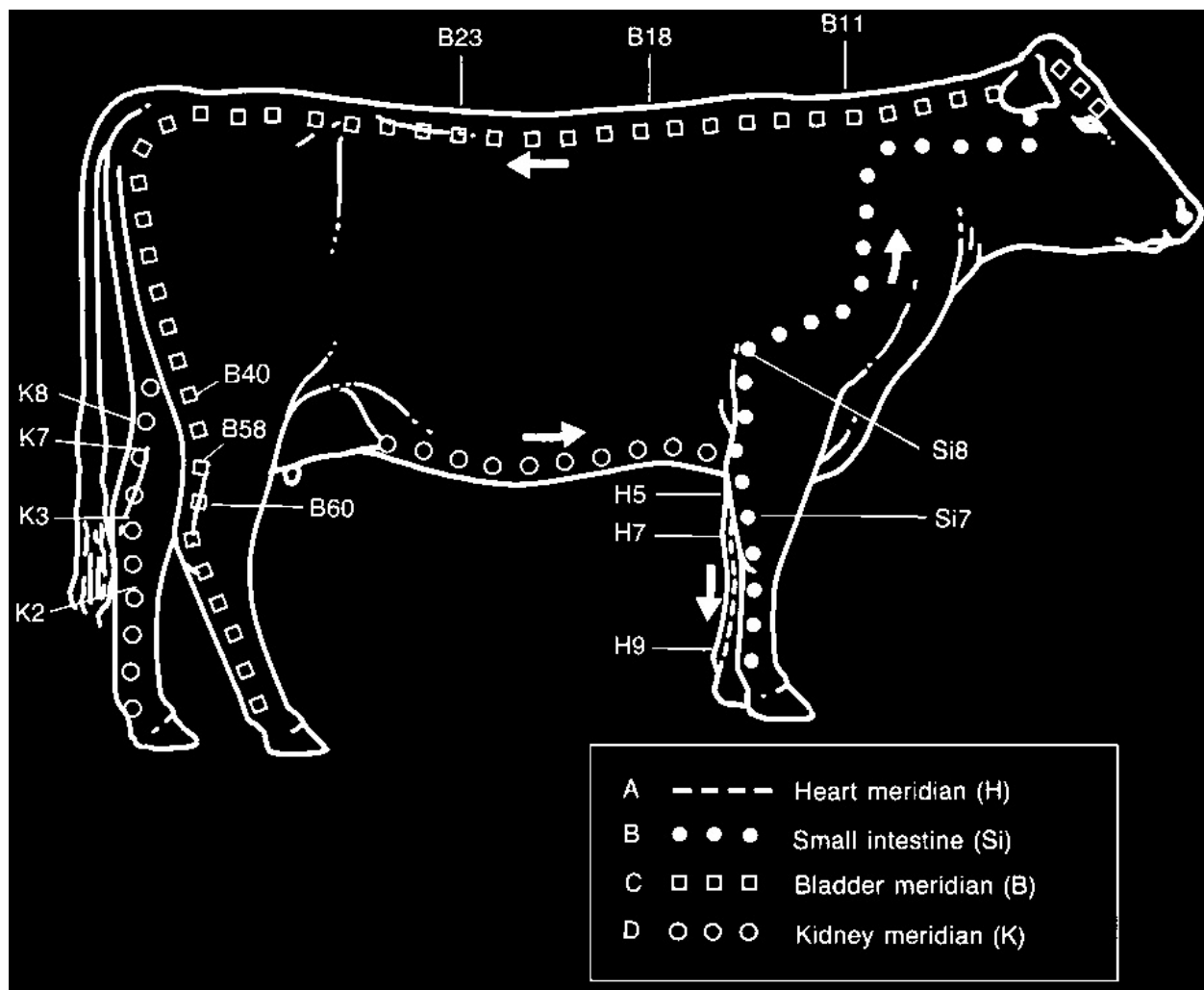
Qi itself has many facets. To the Chinese, who did

hours and the flow pattern follows a ‘-Yin-Yang-Yang-

not distinguish between matter and energy, Qi could be Yin-’ cycle, repeated three times. We are then able to

described as matter on the verge of becoming energy or draw a flow chart, built from the preceding lists





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Fig. 65.3

An energy flow chart for the
'-Yin-Yang-Yang-Yin-' cycle.

Fig. 65.4

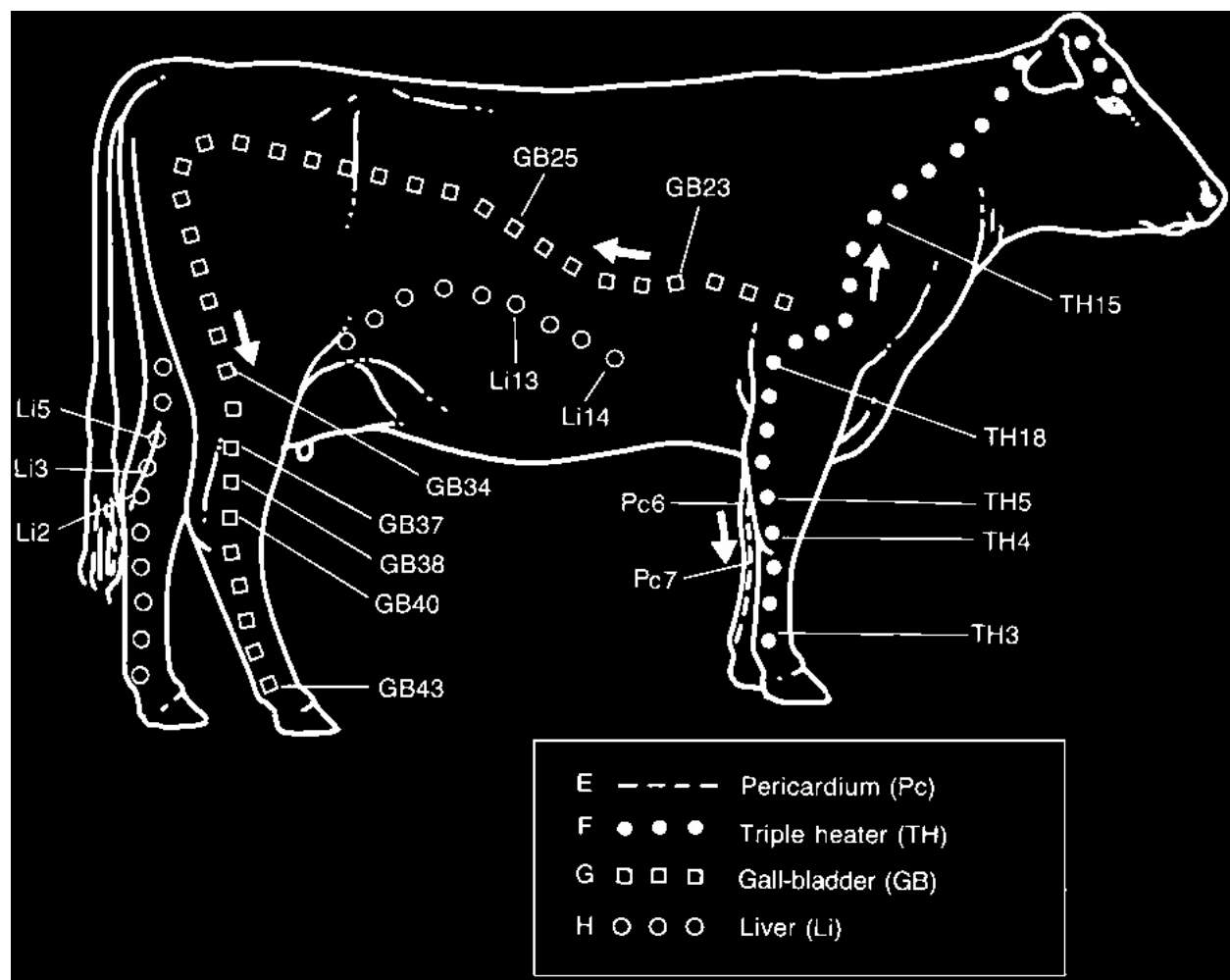
Cycle 1 of the energy flow
chart for -Yin-Yang-Yang-Yin-.

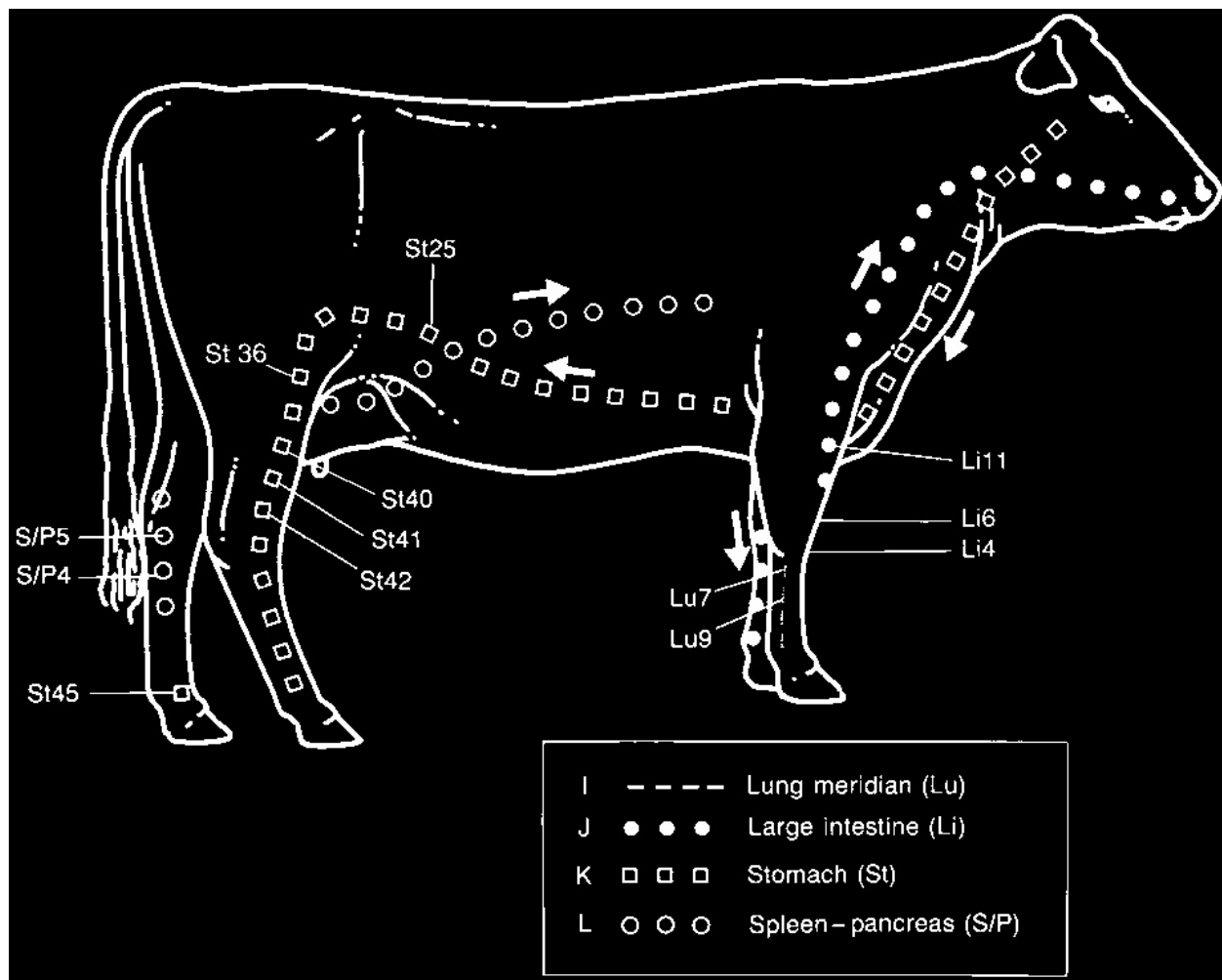
(Fig. 65.3). On the body, these are represented in

*interpretation (Fig. 65.8) and different authors disagree
diagrammatic and simplified form in Figs 65.4–65.7.*

*in detail. Experienced practitioners, nonetheless, are
Routes of meridians, and point locations thereupon, are
able to develop their own working models and to
approximate guides only. Accurate point location
achieve the desired results.*

*requires reference to more substantial works; the titles
Having read that the channels are each related to an
of important reference works can be found in the
organ, the reader is probably wondering about the
Further reading section. Owing to the loss of digits on
triple heater. This ‘organ’ represents thorax, abdomen
the bovine limb (compared with the human five per
and lower abdomen (pelvic), and therefore takes in the
limb) the distal sections of some meridians are open to
functions of respiration, digestion and the urogenital*





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Fig. 65.5

Cycle 2 of the energy flow chart for -Yin-Yang-Yang-Yin-.

Fig. 65.6

Cycle 3 of the energy flow chart for -Yin-Yang-Yang-Yin-.

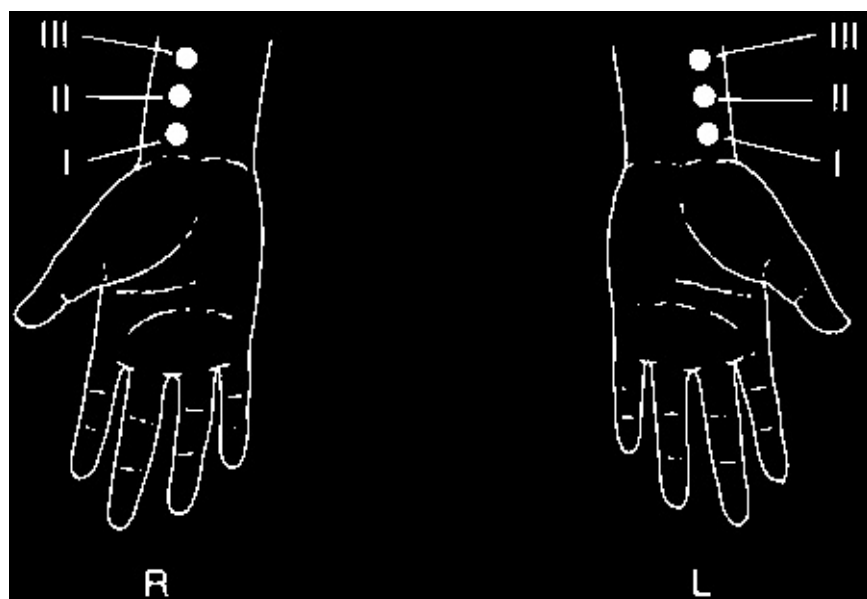
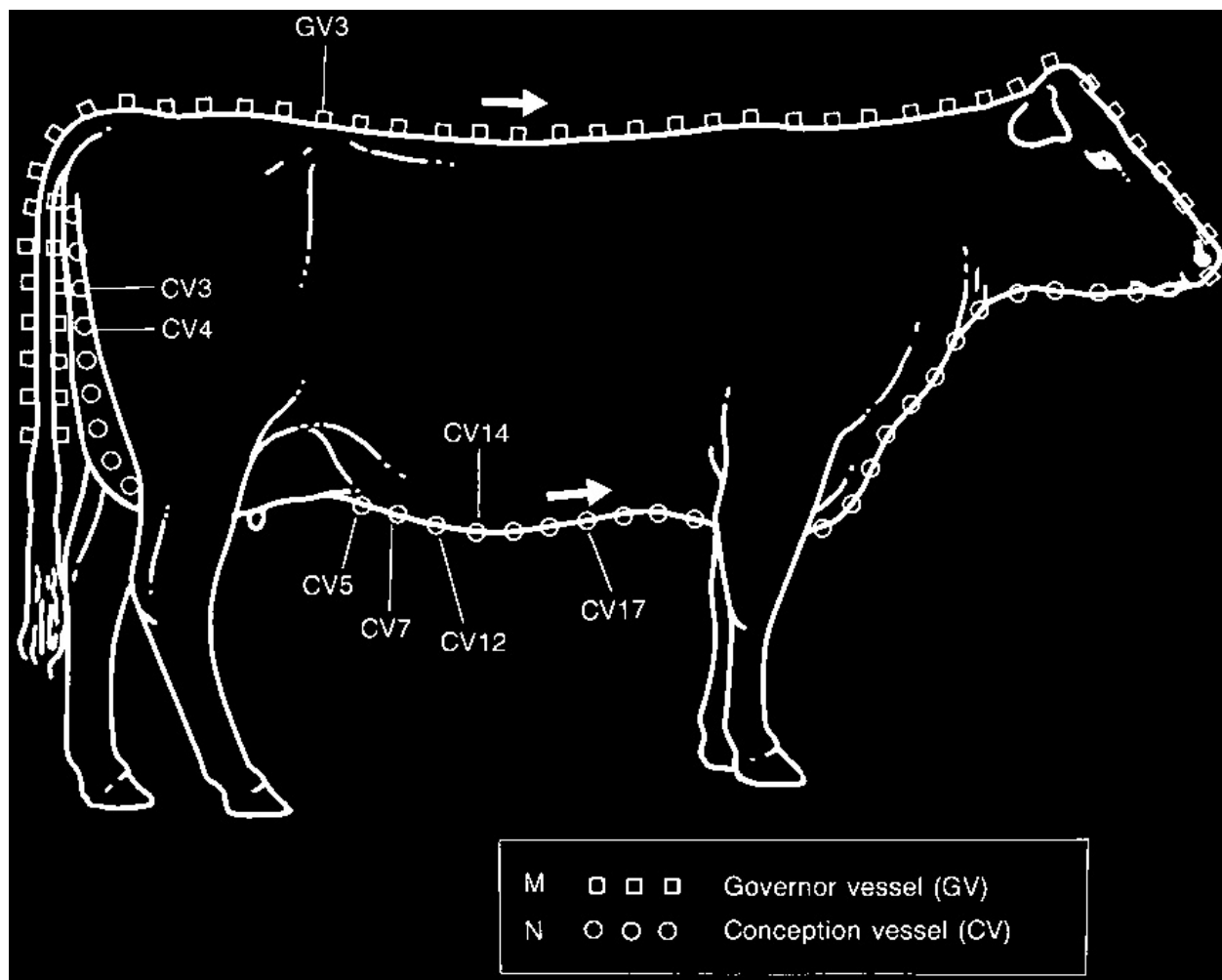


Fig. 65.7

The two extraordinary

(unpaired) meridians (Governor vessel

[yang] and Conception vessel [yin]).

deep internal connections, creating continuity proximally and of the exterior 'secondary vessels', creating continuity at the extremities.

The opposing forces of Yin and Yang are constantly at work in the body and are supposed to be in total balance, flowing evenly through the 'channels' that the meridians represent. In fact it is better to picture a constant strife between Yin and Yang with alternating conquest and defeat but, in a state of health, with a balanced result throughout the 'championship'. It is impor-

Fig. 65.8

Pulse positions on the human wrists. Each position

tant to note that the 'correct' balance for each

has a deep and superficial pulse.

individual is unique, and that a female will tend more towards Yin and a male more towards Yang.

Any stimulus that leads to an uneven 'contest', with

system. It can be considered also as the seat of humoral one or other side in the ascendancy on balance, will lead control and interconnections.

to disease. The Chinese called such adverse forces the There are two other (extraordinary) vessels or merid-
'pernicious influences'. These are wind, cold, heat/fire, ians, which are of similar importance to the six pairs and dampness, dryness and summer heat. They also believed these are the conception vessel and the governor vessel. disease could originate under the influence of emotions These follow, respectively, the ventral and dorsal mid- (joy, anger, sadness, grief, pensiveness, fear and fright), lines. The energy flows from anus to mouth in both and way of life (departure from Tao – 'the way'), diet, sexual therefore the vessels are not 'paired'. They differ from activity and physical activity. Further factors were rec- the other meridians in that they also lack special toni- ognized, that did not fit into these categories, e.g. burns, fication and sedation points, etc. as described later for bites, stings, trauma, etc.

the other meridians, and are not an integral part of

the circadian energy circulation system. The governor

Diagnosis

vessel is dorsal and therefore Yang, the conception

vessel is ventral and therefore Yin (see Fig. 65.7).

Diagnosis of the nature of disorders in the body in

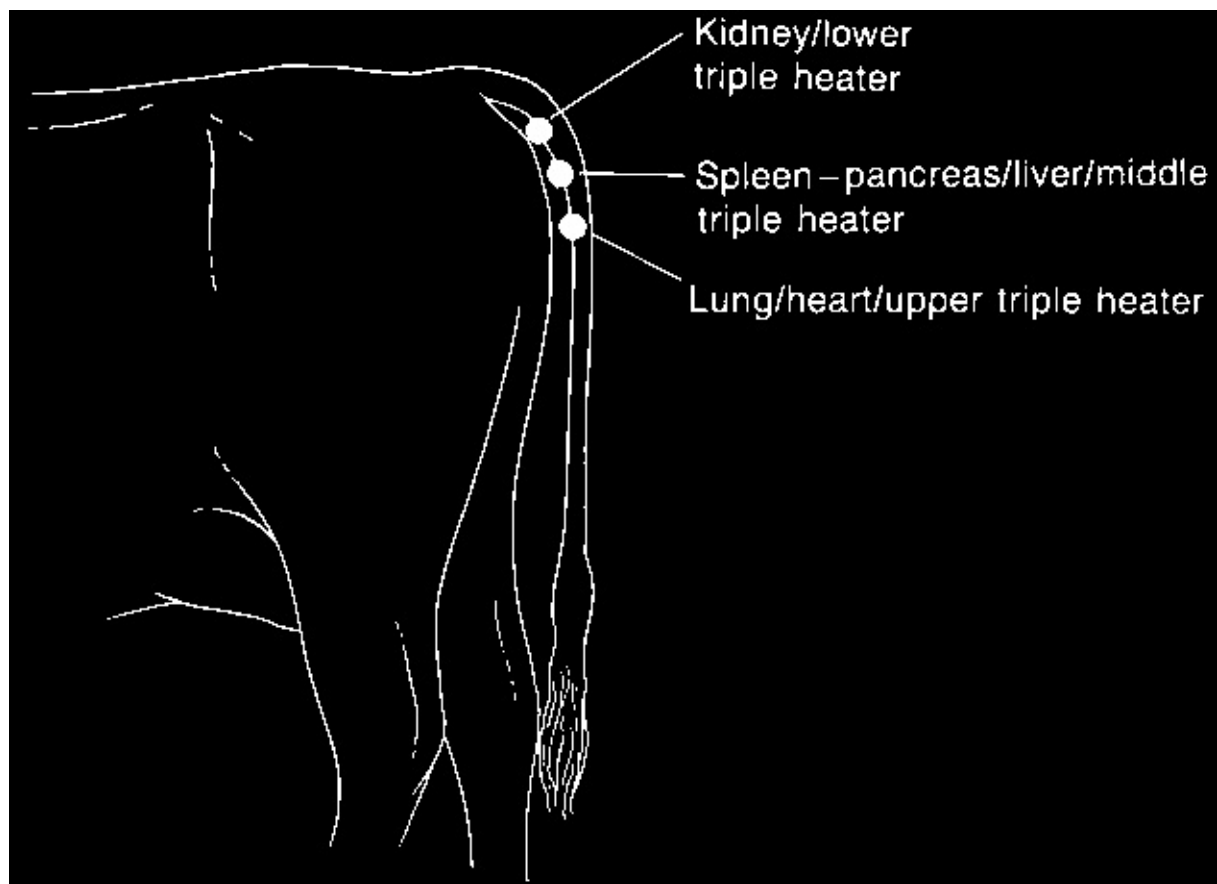
The circulation of the energy (Qi) is allowed to occur terms of the information given in the preceding pages

through the 12, seemingly-disconnected channels, with

was by pulse diagnosis in the human wrist (Fig. 65.8 and

apparent open circuits at each end, by means of the

Table 65.4). Kothbauer and Meng (1983) give a method



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using the midline caudal artery in the bovine (Fig. 65.9), patterns of pathology and in diagnosis. The five element but this is sadly a long way from the complex and theory is not respected by all practitioners of acupunc- refined method developed for the human (through no ture, but holds the key to many important deductions fault of the authors, more of anatomy and practicality). about health and disease for those who uphold its General quality of pulse can still be determined and can

tenets.

be very helpful. Overall impressions only can be gained

Water destroys Fire but creates Wood

by this method.

Wood destroys Earth but creates Fire

Inference can be made from the signs of disease, dis-

Fire destroys Metal but creates Earth

played by the body, as to the nature of pernicious influ-

Earth destroys Water but creates Metal

ence and which meridians or organs are disturbed by

Metal destroys Wood but creates Water

this influence (Kaptchuk, 1983). This requires a great

deal of further study, if the reader is to develop these

The (tsang/fu) organs and the emotions are related to

skills and it is too large a subject for this small treatise.

these elements. Thus, for example:

Help can, nonetheless, be derived from the following

Liver/gall bladder

—

Anger

—

Wood

charts of relationships, which provide information

Heart/small intestine,

on the possible route of pathology, once the disease

pericardium/triple

—

Joy

—

Fire

process has gained entrance (Table 65.2).

heater

An important and functional model for the ancient

Spleen (+pancreas)/

—

Pensiveness

—

Earth

Chinese was the concept of the ‘five elements’. In this

stomach

abstraction there are cycles of creation and destruc-

Lungs/large intestine

—

Sadness/grief

—

Metal

tion/control, which can be of great help in establishing

Kidneys/bladder

—

Fear/fright

—

Water

*In addition, the organs and functions of the body are
related to each other, for example:*

*The kidneys are connected with (control) the bones and
rule over the spleen*

*The heart is connected with (controls) the pulse and
rules over the kidneys*

*The lungs are connected with (control) the skin and rule
over the heart*

*The liver is connected with (controls) the muscles and
rules over the lungs*

The liver nourishes the muscles and the muscles

strengthen the heart

The heart nourishes the blood and the blood strengthens the spleen, etc.

Fig. 65.9

Pulse diagnosis in the bovine (according to

These connections are not so far-fetched, even in Kothbauer & Meng, 1983).

modern Western terms.

Table 65.2

Pernicious influences on disease.

Yang

Yang

Yin

Yin

Season

Spring

Summer

Late summer

Autumn

Winter

Climate

Wind

Heat

Dampness

Dryness

Cold

Direction

East

South

Centre

West

North

Organs (Tsang)

Liver

Heart/triple heater

Spleen

Lungs

Kidneys

Organs (Fu)

Gall-bladder

Small intestines/pericardium

Stomach

Large intestines

Bladder

Orifices

Eyes

Ears

Nose

Mouth

Urogenital orifices

Tissues

Ligaments

Arteries

Muscles

Skin and hair

Bones

Elements

Wood

Fire

Earth

Metal

Water

Odour

Rancid

Scorched

Fragrant

Rotten

Putrid

Emotions

Anger

Joy

Sympathy

Grief

Fear

Fluids

Tears

Perspiration

Saliva/mucus

(Mucus)

Spittle

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Wood

Water

Fire

Metal

Earth

Fig 65.10

*The creation (sheng) and
destruction or control (ko) cycles of five
element theory. The large arrows denote
creation, dashed lines denote destruction/
control.*

*What has been described, although briefly and there-
scriber has arrived by following the foregoing para-
fore not to do it full justice, is an attempt on the part of
graphs. Treatment of the body in traditional Chinese
the ancients to explain the rhythm and dynamics of life
medicine, whether by diet, herbs, moxibustion or
and disease, albeit often by metaphor, which can be off-
needles, attempts to restore the balance in the constant
putting to the Western mind. The explanation does
conflict between Yin and Yang. This treatise really only
seem to embrace the reality of the integrity of the whole
deals with treatment by insertion of needles.*

body and its interconnectedness, both in anatomy and

Moxibustion is the application of heat to specific function, and its relationship to the outside world. points of the body to supply heat energy (yang) to the Disease does not come in tidy parcels, affecting only system. Usually, artemisia vulgaris herb is used for this one part of the body in a single way, but in complex pat-purpose, as a 'wool'. Heat is applied directly to the area

terns of whole body dysfunction. The philosophy or a few millimetres above it. Needles can also be behind traditional Chinese medicine embraces this inserted at the point and heated by burning moxa. It is essential concept very satisfactorily and provides a not a practical method for the beginner, for general means of arriving at a 'diagnosis'.

farm use. It is mentioned briefly here, merely to put it From the platform of the diagnosis, in the traditional in context and to introduce the reader to the concept, Chinese sense, a treatment rationale or treatment prin- which may otherwise confuse when it is encountered in ciple can then be devised. This will be based on the need other texts.

to balance Yin and Yang, to restore harmonious flow

There are many different categories of points that of Qi, to counteract the pernicious influences and to can be utilized in acupuncture prescriptions, in an support the stressed organs. What may be surprising, to attempt to restore the desired balance. The major ones the uninitiated, is that this allegorical methodology are listed below.

can provide the clinician with a valid working model for effective diagnosis and successful therapy.

The points

Acupuncture points are not anatomically represented,

Treatment (Plates 65.1 and 65.2)

although there are histological and physiological prop-

The acupuncture prescription for the patient should be erties which are peculiar to them. A good account of

devised from the treatment principle, at which the pre-

this phenomenon is given by Kothbauer and Meng

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(1983). Their exact positions are described in relation

alone are unlikely to be far-reaching. Like master

to anatomical landmarks,

measurements being

points, the symptomatic points may also be used in expressed in terms of units which relate to the dimensions as diagnostic tools, the reliability being questions of specific parts of the body. The measurement is not reliable unless they are used as part of the whole units are:

picture.

Tsun or cun: this is the acupuncture inch. In human Pilot points are used properly to redistribute or balance acupuncture it is traditionally equal to the length of

the Yin–Yang conflict. Main points are the tonification the second phalanx of digit 3 of the human hand.

points, which increase energy in an energy deficient

Fen: One tenth of the above.

meridian (gold needles are often used) and sedation

Finger breadth, thumb width, forearm length, pubis-nates, points decrease energy in overactive meridians (silver shin length, etc. are all as they state, and apply to

needles are often used). Each meridian has one of each

human measurement, but sources vary as to their relationship of these.

tionship to the cun. It is usually accepted that a thumb width is 1 cun, two finger breadths are 1.5 cun, four The source (yuan) point on each meridian acts as an

*finger breadths are 3 cun, cubital crease to carpal
adjuvant to one or other of the two above-mentioned
crease is 12 cun, etc.*

*points. The metal used for needling is selected accord-
It may be thought strange to have this unique form of
ing to which function it is wished to augment.*

*measurement, but it allows for measurement propor-
tional to the size of each individual patient. In horses,*

*The passage or connecting (luo) points, situated on each some texts refer to the
‘inch’ as the width of the 16th*

*meridian, will help to ‘shunt’ energy from the meridian
rib, at the level of the tuber coxae. The provision for
in question to its partner. Energy usually flows ‘down-
measurement in animals is not good, so sources can
hill’ (i.e. from ‘mother’ to ‘son’ or from the meridian
refer to precise anatomical descriptions, rather than
earlier in the cycle to its paired neighbour).*

measurement units.

*The alarm (mu) points may or may not lie on the meridian to which they relate
but may be used for diagnosis*

The point types

or treatment if sensitive. A sensitive point indicates

Locus-Dolendi needling, using Trigger (ah-shi) points is stress in its related organ.

the use of points of local pain, which are directly related to the site of injury or illness. The effect is not very long-

The association (shu) points, lying on the bladder

lasting but provides relief from pain in local conditions.

meridian, are also of value diagnostically or therapeu-

It requires little knowledge on the part of the pre-

tically and relate to specific organs and meridians. For

scriber, but should probably be reserved for localised

example, the association point for the spleen/pancreas

locomotor pain or injury.

is bladder 20, for the heart it is bladder 15 and for the

stomach it is bladder 21.

Master points relate to individual organs or tissues and It is helpful to list some useful points for earlier selec-can be used regardless of the nature of the pathology

tion by the beginner; see Table 65.3 and refer to Figs

or dysfunction which that target suffers. These master

65.3–65.7.

points may be sensitive to stimulus when there is a dys-

Table 65.3 is clearly by no means comprehensive (as

function or injury in the influenced organ/tissue but are can be seen from the numeration), but represents a unreliable diagnostically, unless taken as part of the useful reference list for the main functional points. For wider picture. Effects of using these points alone, as a more comprehensive point descriptions the reader is method of prescribing, will lead to disappointment. referred to much fuller works on acupuncture therapy. There is no deep and lasting influence on disease if the Their location, as shown on Figs 65.4–65.7, is a syn-prescriber sticks to this elementary process. The master thesis of information from personal experience and point of the musculature, for instance, is gall bladder 34. the work of Klide and Kung (1977), Lewith (1982), Kothbauer and Meng (1983), Bishchko (1983), Gilchrist Symptomatic or influential points have an influence over (1984) and Janssens (1984). The nomenclature of the certain specific functions. For instance, bladder 31 influ-points is in accordance with the IVAS standard, in order ences female hormonal function very strongly and gall to prevent confusion and to enable the reader to

bladder 39 influences the function of marrow. They can compare with all IVAS-derived texts. When reading be used in a prescription when such problems predominate other texts, many different conventions of nomenclature (Plate 65.1). The action of these points is usually fairly narrow so, again, prescriptions based on these should not be confused by this, but should develop the

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Table 65.3

Useful points.

Heart (HT) meridian

34. Master point of muscles

5. Passage point

37. Passage point

7. Sedation point and Source point

38. Sedation point

(9.) Tonification point

40. Source point

43. Tonification point

Small intestine (SI) meridian

(site open to interpretation)

}

(3.) Tonification point

Liver (LIV) meridian

(4.) Source point

2. Sedation point

(medial hock)

}

7. Passage point

3. Source point

8. Sedation point

5. Passage point (postero-medial tibia)

8. Tonification point (postero-medial stifle joint)

Bladder (BL) meridian

13. Alarm point for spleen/pancreas (caudal to last rib)

11. Bone disorders

14. Alarm point of the liver

13. Association point, lung

14. Association point, pericardium

Lung (LU) meridian

15. Association point, heart

1. Alarm point for lung (between shoulder joint and third rib on 18. Association point, liver

chest wall)

19. Association point, gall-bladder

5. Sedation point (medial elbow)

20. Association point, spleen/pancreas

7. Passage point

21. Association point, stomach and master point, stomach

9. Tonification point and source point

22. Association point, triple heater

(II.) Master point for the throat (not clearly identifiable in the 23. Association point, kidney

bovine)

25. Association point, large intestine

27. Association point, small intestine

Large intestine (LI) meridian

28. Association point, bladder

(1.) Master point for teeth

(site open to interpretation)

}

31. Master point, female hormones

(2/3.) Sedation points

58

Passage point

4. *Source point*

(medial carpus)

}

60

Master point for pain

6. *Passage point*

(64) Source point

II. Tonification point

(65) Sedation point

(site open to interpretation)

}

(67) Tonification point

Stomach (ST) meridian

25. *Alarm point for large intestine*

Kidney (KI) meridian

40. *Passage point*

1/2. *Sedation point*

41. *Tonification point*

3. *Source point*

42. Source point

4. Passage point

45. Sedation point

7. Tonification point

8. Master point blood

Spleen/pancreas (SP) meridian

(II.) Alarm point pericardium (not clearly identifiable in

(2.) Tonification point

(site open to interpretation)

}

bovine) (in groin)

(1) Source point

4. Passage point

Pericardium (PC) meridian

5. Master point for connective

(anteromedial tarsus)

}

*1. Alarm point pericardium (lateral thorax in posterior
tissue and sedation point*

axilla)

6. Passage point

Conception vessel (CV) (Fig. 65.7)

7. Sedation point and source point

3. Alarm point for the bladder

(9.) Tonification point (anterolateral of second digit)

4. Alarm point for small intestine

5. Alarm point for triple heater

Triple heater (TH) meridian

7. Alarm point for lower triple heater

3. Tonification point

12. Alarm point for stomach and alarm point for middle triple

4. Source point

heater (caudal to xiphoid, cranial to navel)

5. Passage point

14. Alarm point for the heart (caudal to xiphoid)

10. Sedation point

17. Alarm point for upper triple heater and pericardium (on

15. Master point for forelimbs

sternum, rib 7)

Gall bladder (GB) meridian

Governor Vessel (GV) (Fig. 65.7)

24. Alarm point for gall bladder

No main or special points.

25. Alarm point for kidney (caudal to last rib)

20 and 26 are very important points for advanced manipulation of Qi.

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Table 65.4

The relationships based on the human pulse system.

Position

Right

Left

Superficial

Deep

Deep

Superficial

III

Large intestine

Lung

Heart

Small intestine

II

Stomach

Spleen/pancreas

Liver

Gall bladder

I

Triple heater

Pericardium

Kidney

Bladder

(Yang)

(Yin)

(Yin)

(Yang)

ability to ‘translate’ these into the ‘standard’. In texts on

the rule will operate upon stimulation of the relevant

TCM, another potential confusion arises from the use

points with appropriate needles. Using the three rules

of the various conventions of westernization or roman-

together can achieve a rapid restoration of harmony

*ization of the Chinese language (e.g. Pinyin or Wade-within the body. The system
of acupuncture thus used*

*Giles). Again, it is important not to be daunted by this, is not only complete but
also quite simple to apply, pro-but to compare texts in order to find the*

correlations.

viding infinite combinations of therapy. It is sad that pulse diagnosis is not so refined in animals, but the general quality of pulse can still be ascertained. The

Use of these points

relationships of the meridians are still valid and so too

The meridians are interconnected energetically in

is the correspondence of disease patterns with the

various 'rules' of therapy. Treatment strategies may be

effects from the pernicious influences and other origins

based on these relationships. Again the Chinese show

of disharmony. Conclusions about meridian malfunc-

their proclivity towards metaphorical description. The

tion are inferred from the disease patterns displayed by

first relationship is based upon the human system of

the patient and by the demonstrable point sensitivities.

pulse diagnosis and relates to the position of pulse-

taking in the human wrist (Table 65.4; see also Fig.

65.8).

Technique of needling and treatment

(1)

The ‘husband–wife’ rule relates meridians sharing Steel, gold or silver needles are selected according to the same positions on either wrist. Tonifying one the effect required. Alternatively, the different effects will sedate its partner and vice versa. They oppose can be achieved by varying the needling technique or each other.

by varying the duration of insertion. The needles should
(2)

The ‘mother–son’ rule relates to the sequence of be inserted confidently into the skin, as near to the the meridians in the circadian rhythm. A meridian centre of the point as possible. Depth of insertion is preceding that in question is its mother and that generally up to 1 cm, but varies from point to point. succeeding it is its son. It is logical to assume that During a treatment the body ‘reacts’ with the needles the mother is ready to give energy, the son to in a way that has not been defined physiologically or receive it. Tonifying the meridian in question and anatomically. During this time, the needles are firmly

its mother will produce extra effects. Similarly, held by the tissues. When a treatment is over, perhaps sedating both the meridian in question and its between 5 and 20 minutes later, the needles may be son will increase the effects of sedation. Passage removed with ease.

points may be used instead when the meridians in After treatment there may be a 'slump' in the question are partners.

patient's apparent energy for the next 24 hours. The (3)

The 'midday-midnight' rule relates meridians author has observed this less in cattle than in other most active in a similar time period, am and pm. species. Treatments may be repeated at intervals of One will be Yin and one Yang, e.g. lung (Yin), 4 several days to several weeks, but a useful guide for an am to 6 am., bladder (Yang), 4 pm to 6 pm. Toni- initial course of therapy is a 3 to 14-day interval for fying a meridian in its active time will sedate its three or four treatments. If no improvement is noted in

opposite number.

this period, it is possibly a sign of a poor prognosis. Most

of the author's successful cases have shown definite

These three rules should always be borne in mind

improvement within the initial three treatments. After

because, even if it is not the intention of the prescriber,

a good initial result, in chronic conditions, treatment

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may need repeating at longer intervals (say six monthly,

and using it in isolation is not at all in accordance with

but the patient's condition should determine this).

the holistic principles of herbal medicine. The obvious

Up until recently, it has been very difficult to obtain

danger of this practice, leading to potential side-effects,

meaningful training in veterinary acupuncture. At the

is that the synergistic effect of the other constituents of

time of writing, however, courses are available run by

the plant is lost.

the combined efforts of the International Veterinary

Western herbs are classified according to their per-

Acupuncture Society (IVAS), the Association of British

ceived actions, as per the following examples.

Veterinary Acupuncturists (ABVA) and Exeter University.

Alterative, anodyne, anthelmintic, antibacterial, antitarrhal,

antiemetic,

anti-inflammatory,

antifungal,

antilithic, antispasmodic, aperient/laxative, aromatic,

Homoeopathy and acupuncture

*astringent, bitter, cardiac, carminative, cathartic/purgative, **combined***

cholagogue and anticholagogue, demulcent, diaphoretic,

diuretic, ecboic, emetic, emollient, expectorant, febrifuge, Since both homoeopathy and acupuncture are address-galactagogue, hepatic, hypnotic, nervine, rubefacient,

ing the same entity or challenge, the living and inter-

sedative, sialogogue, soporific, stimulant, styptic, tonic, connected body, in disease, it stands to reason that they

vesicant and vulnerary.

must overlap if they are truly effective. The different

Chinese herbs are classified according to their ability rationales and strategies merely express the fact that

to modify the course of action of the pernicious influ-

they represent two different windows on the same
ences. The usual oriental pictorial language prevails.
room. It is possible to combine the two therapies, only
Examples, with an approximate Western equivalent
if the rationale for selection of treatments is harmo-
where appropriate, are:

nized. Failure to do this will produce disharmony and
will inevitably be counterproductive. If in doubt, the
Release externally

(diaphoretic?),

purge down

best advice is not to attempt it.

(purgative?), clear heat, transform moisture (damp),

It is beyond the scope of this text to go into detail on

facilitate urine (diuretic?), expel wind-damp, dispel this facet of therapy, but
there is a reference to further

cold, etc.

study in the Further reading section below (Day, 1993).

Because material doses of herbs are utilized, there is
the inevitability of tissue or milk residues. Some of

Herbal medicine

these will be totally harmless or even beneficial to the consumer. Some may be harmful. Because the residue

The use of plants and plant material in medicine is as side of the use of herbs is so poorly researched and so old as human existence. Those plants which form the beyond our control, it is wiser not to use herbs in food-basis of herbal medicine are called medicinal herbs, but producing animals unless absolutely sure of the consequences. A fuller account of veterinary herbal medicine could prove to be of benefit, if researched. They are is to be found in the Further reading section (Day, classified according to origin (whether ayurvedic, 1998).

traditional Chinese, Western, Australasian, etc.), or according to effect. This latter would be a different classification system, depending upon the culture which

Essential oils

devises it. Broadly speaking, in the West, we classify according to the perceived pharmacological effect.

The use of essential oils in medicine (aromatherapy) is

A large proportion of modern conventional drugs also a very ancient tradition around the world. Further owe their existence or their origins to herbal medicine. reading is recommended (Grosjean, 1994). The appli-

The rationale of the use of herbs is to exploit their cation and rationale are not dissimilar to those for primary effect, rather similarly to the rationale of herbal medicine. The active principles are distilled aro-

modern drug medicine and unlike homeopathy, which matic compounds derived from plants and represent exploits the secondary effect (the body's reaction), very powerful and effective medicines. The classifica-

although it uses many of the same plants that are used tion of the oils is as for Western herbs.

in herbal medicine. Although there are similarities

Since the same caveats apply to aromatherapy for between drug medicine and herbal medicine, the tradi-

food-producing cattle, as have been detailed for herbs, tion of herbal medicine is very holistic in the truest

with the additional worry of powerful taints of meat or sense. The more modern fashion for establishing a sup-

milk, the use of aromatherapy in production cattle is posed active ingredient of a medicinal herb, purifying it not advised.

Alternative Medicine • 1103

Other therapies

is best used on those animals with which we develop close emotional ties. This is because we are then better Two other therapies lend themselves to specialized use able to discern the necessary prescribing clues. There is in cattle and bring with them no risk of meat or milk not much application in farmed cattle, for this reason.

residues, they are Schüssler's Tissue Salts and Bach's Further reading is recommended, for a deeper under-Flower Remedies.

standing (Chancellor, 1971; Ball & Howard, 1999)

Bach's 39 remedies include agrimony, beech, cen-

Tissue Salts:

In the latter part of the nineteenth

taury, clematis, elm, holly, oak, olive, rock rose, scleran-century, Schüssler, who was a homoeopath, devised a

thus, vine and willow. The system is included in this system of medicine based upon the mineral nutrition of chapter, esoteric though it may seem, for two reasons.

cells. His 12 mineral remedies were prepared to the
It introduces the reader to the medical modality and it
sixth centesimal potency, according to homoeopathic
presents the opportunity to use of one of Bach's medi-
methodology. The 12 remedies are:

cines, which can be of great value in cattle medicine:

Rescue Remedy.

Calcarea fluorata,

calcarea phosphorica,

calcarea

Rescue Remedy is composed of Bach's cherry plum,

sulphurica, ferrum phosphoricum, kali muriaticum, kali

*clematis, impatiens, rock rose and star of bethlehem. It is phosphoricum, kali
sulphuricum, magnesia phosphorica,*

of great virtue in the treatment of stress, fear and shock

natrum muriaticum, natrum phosphoricum, natrum

situations, and can be used just as well in cattle as in

sulphuricum, silica.

other species. Situations demanding its use are post sur-

These remedies, according to the founder, are able to

gical trauma, disbudding, herding of unhandled cattle,

correct cellular chemistry in order to achieve health injuries, entrapments or 'casting', bereavement or distress on handling for procedures such as foot trimming. and the practice persists in modern times. The author It can be given by mouth or to a group via the drinking believes that these remedies act as facilitators, regulators or modulators of those cellular processes which involve those minerals. A more detailed description of

Conclusion

this action of potentized substances is found in the section on homoeopathy, presented earlier in the It is to be hoped that the reader, having studied this chapter. Knowing what we now know about enzymes chapter or one or other part of it, will have experienced and their cofactors, this is easily comprehensible in adequate stimulation to take a deeper look into alter-modern terms.

native medicine (see Further reading section). It is also In view of modern knowledge and understanding,

hoped that he or she will have gleaned sufficient information and recognizing the adverse effect of artificial nitrogenation to take the first steps in applying one or both of the main therapies described therein. What should be clear is that either of those systems of medicine is a very large and complex subject. Should the reader wish to include chromium, cobalt, copper, iodine, manganese, selenium, zinc, etc. The potential for helping cattle go further, it must be said that a great deal of motivation to balance, in terms of mineral metabolism, particularly in those situations of relative or marginal insufficiency provided the motivation, that is good. If not, then it as can be encountered on farms, is an interesting development should at least serve to dispel some of the mists of ignorance and suspicion that enshroud the art and science amount to material supplementation, but the aid to of both homoeopathy and acupuncture. This should absorption and metabolism, which they can offer, can

*allow for more productive cooperation between con-
amount to a significant benefit. The use of these poten-
ventional veterinarians and those who have diverged
tized salts, in place of some of the mineral supplemen-
into 'alternative medicine'. Those who choose to apply
tation now required by most farm livestock, should be
themselves to diligent study and to professional appli-
particularly attractive in the case of organic farm units,
cation of these methods will be able to enjoy a life full
where outside inputs need to be minimized (Day, pers.
of surprises and rewards commensurate with effort, but
comm.).*

will not have chosen an easy option.

Bach Flowers:

In the 1930s, another homoeopath,

Edward Bach, devised a system of medicine which

References and further reading

*would treat disease more via the psychology and per-
sonality of the patient than via the obvious physical*

*Ball, S. & Howard, J. (1999) Bach Flower Remedies for
signs. This system can be applied well to animals, but it*

Animals. C.W. Daniel, Saffron Walden.

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Bischko, J. (1983) Einführung in die Akupunktur, 13th edn.

Gilchrist, D. (1984) Greyhound Acupuncture. Pro-Prom Pty, Karl F. Haug, Heidelberg.

Mareeba.

Boericke, W. (1972) Materia Medica with Repertory. Boericke Goodwin, J.S. (1980) Biochemic Handbook. Thorson's,

& Runyon, Philadelphia.

Wellingborough.

Brock, K. & Nielsen, J. (1986) Veterinær Homøopati. DSR

Grosjean, N. (1994) Veterinary Aromatherapy. C.W. Daniel, Forlag, København. Saffron Walden.

Chancellor, P.M. (1971) Handbook of the Bach Flower Reme-

Hahnemann, C.F.S. (c1814) Homöopatische Heilkunde der dies. C.W. Daniel, Saffron Walden.

Hausthiere, Leipziger Universitätsbibliothek.

Clarke, J.H. (1982) Dictionary of Materia Medica, 3rd edn, Vols Hahnemann, C.F.S. (1833/42) The Organon of Medicine,

1, 2 and 3. Health Science Press, Saffron Walden.

5th/6th edn (transl. by Boericke & Dudgeon). B. Jain, New

Day, C.E.I. (1984a) Control of stillbirths using homoeopathy.

Delhi.

Veterinary Record, **114**, 216.

Hansard (1855) 14th May, pp. 557–8.

Day, C.E.I. (1984b) *The Homoeopathic Treatment of Small*

Janssens, L.A.A. (1984) Acupuncture Points and Meridians in Animals, Principles and Practice. Wigmores, London (now the Dog. Blondiau Print, Beersel.

republished as an expanded 3rd edition by C.W. Daniel,

Kaptchuk, T.L (1983) Chinese Medicine. Rider, Melbourne.

Saffron Walden, 1998).

Kent, J.T. (1986) Repertory of the Homoeopathic Materia

Day, C.E.I. (1985) Dystokia prevention. In Proceedings of Medica. Homoeopathic Book Service, London.

LMHI Congress, Lyon.

Klide, A.M. & Kung, S.H. (1977) Veterinary Acupuncture.

Day, C.E.I. (1986) Clinical trials in bovine mastitis using

University of Pennsylvania Press.

nosodes for prevention. International Journal for Veterinary Kothbauer, O. & Meng, A. (1983) Grundlagen der Veterinär Homoeopathy, 1 /1, 15.

Akupunktur. Verlag Welsermühl, Wels.

Day, C.E.I. (1987a) Isopathic prevention of kennel cough.

Lewith, G.T. (1982) Acupuncture, its Place in Western Medical International Journal for Veterinary Homoeopathy, 2/2, 57.

Science. Thorsons, Wellingborough.

Day, C.E.I. (1987b) A Guide to the Homoeopathic Treatment

MacLeod, G. (1981) The Treatment of Cattle by Homoeopathy. Beaconsfield Publications, Saffron Walden.

Beaconsfield.

MacLeod, G. (1983) A Veterinary Materia Medica and Clinical Repertory. C.W. Daniel, Saffron Walden.

Day, C.E.I. (1993) Acupuncture and homoeopathy combined.

Lecture delivered to IVAS Conference in Minneapolis,

Mahé, F. (1987) Evaluation of the effect of a collective homoeopathic cure on morbidity and the butchering quality in USA.

Day, C.E.I. (1998) Herbal medicines. In The Veterinary

ties in fattening calves. International Journal for Veterinary Formulary (ed. Y. Bishop). Pharmaceutical Press, London.

Homoeopathy, 2/1, 13.

Day, C.E.I. (1999) Herdenbetreuung (herd treatments). In

Schoen, A.M. (1994) Veterinary Acupuncture – Ancient Art to Proceedings of the 2nd Workshop: Veterinary Homoeopathy

Modern Medicine. Mosby, USA.

in Organic Herds. IAVH, Frick, Switzerland.

Schroyens, F. (1993) *Synthesis, Repertorium Homeopathicum* Daykin, P.W. (1960) *Veterinary Applied Pharmacology and*

Syntheticum. Homeopathic Book Publishers, London.

Therapeutics. Baillière, Tindall & Cassell, London.

Veith, I. (1972) *The Yellow Emperor's Classic of Internal Medicine* Garner, R.L. (1967) *Veterinary Toxicology* (rev. by E.S.C.

Medicine. University of California Press, Los Angeles.

Clarke & M.L. Clarke). Baillière, Tindall & Cassell,

Vermeulen, F. (1997) *Concordant Materia Medica*. Emrys bv London.

Publishers, Haarlem.

Chapter 66

Aspects of Bovine Surgery

G. Wyn-Jones

Introduction

1105

Secondly, use plenty of anaesthetic solution. It is cheap

Anaesthesia

1105

and the added expense of using 50 per cent more is

Preparation of the surgical site

1105

easily outweighed by better results. For example, most

Surgery

1106

descriptions of paravertebral anaesthesia advocate

Rumenotomy

1106

15–20 ml spread between the upper and lower branches

Dilatation and displacement of the abomasum

1109

of each nerve. Increasing the amount to 25 or 30 ml

Intestinal obstruction

1113

greatly increases the percentage of successful blocks,

Caesarean section

1115

Amputation of a digit or claw

1119

especially in relatively inexperienced hands. Thirdly,

Surgical correction of an umbilical hernia

1122

despite the refinement of the techniques nerve blocks

Dehorning of an adult

1124

are still relatively imprecise methods of inducing anal-

Eye enucleation

1126

gesia. To offset this, spread the anaesthetic solution

Teat surgery

1127

more widely to create a larger block of saturated tissue

and to increase the chances of affecting the target

nerve. Finally, do not 'line-block' at the site of the inci-

Introduction

sion if at all possible as this creates a sodden, jelly-like

tissue which is difficult to dissect and which has reduced

The following chapter deals with those surgical situa-

initial healing properties. Use either a regional block or

tions which most commonly arise in dairy practice.

a field block.

There are always several ways to achieve the same end

and there are probably as many variations in the tech-

niques described here as there are surgeons. The

Preparation of the surgical site

methods detailed in this account are only one variation.

However, they are based on many years of experience

Whenever we operate on large or small animals we

in practice and in the more rarified environment of the

have to accept that we cannot create a sterile field. At

university teaching hospital and every attempt has been

best we can hope to make it 'surgically clean' and

made to keep the contents practicable and pragmatic.

reduce bacterial contamination to a level where it can

Although each individual technique is dealt with com-

effectively be controlled by antibiotics and body

prehensively, there are some general points which

defences. The weakest link in sterility is always the

relate to them all and are most sensibly made at the

patient itself and this is where most attention should be

outset.

directed. Physical removal of hair and dirt is important;

use proper clippers whenever possible and clip a big

enough area to give a good margin around the incision

Anaesthesia

site. Initially wash the area with copious amounts of

water and antibacterial soap, although elbow grease is

Most procedures described here will be done under

probably more important than antisepsis at this stage.

local anaesthesia or regional 'blocks'. The author's

Finish with an antibacterial agent which will persist on

guide to success with these is based on several points.

the skin; although pure surgical or methylated spirits

Firstly, for most 'blocks' in cattle use 3 per cent ligno-

poured onto the skin is comforting it does not, in fact,

caine with adrenaline. The rate of development of the

achieve very much.

block is faster and it persists for much longer than when

Drape as extensively as possible. For abdominal

a 2 per cent solution is used. However, use the latter

surgery prepare several large drapes that will hang over

alone without adrenaline for subcutaneous infiltration

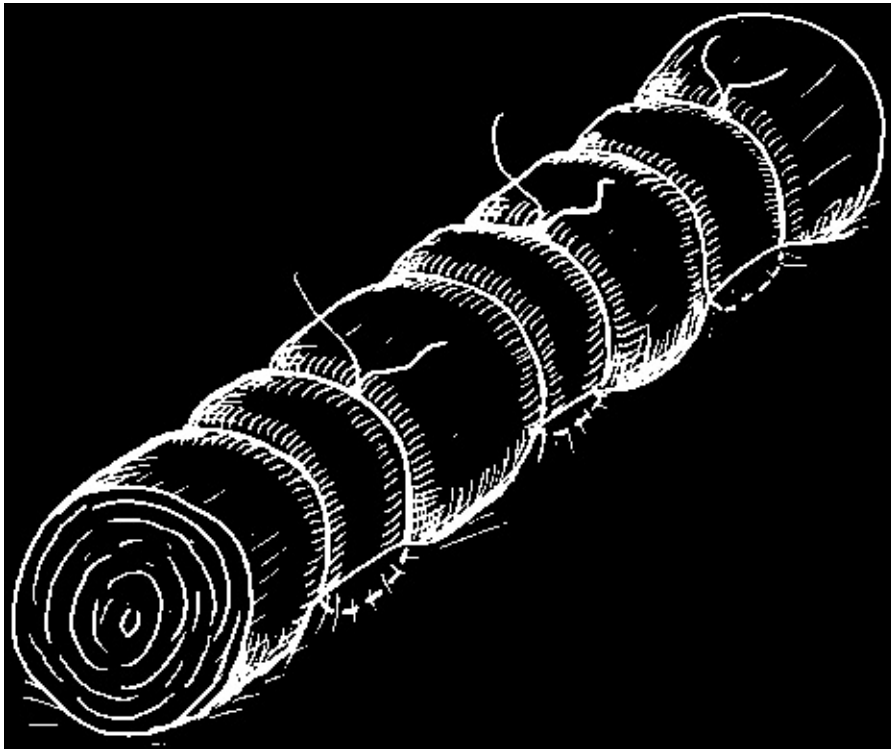
the animal's back, each with holes appropriate for a

as occasionally the vasoconstrictive effects of adrena-

certain technique. Only two variations are really

line can lead to local skin ischaemia, necrosis and death.

needed, one with two holes for abomasal and rumen



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approaches and one for caesarean sections. The synthetic material used for tent making is now widely available and is ideal, being impervious to fluids, mouldable and capable of being sterilized. The use of drapes makes the maintenance of a clean site so much easier and imparts a professional air to the operation.

The choice of suitable dress for the surgeon is difficult. Abdominal surgery requires extensive intra-abdominal palpation or manipulation. A long-sleeved

gown is inconvenient in that the material abrades the wound edges and rapidly becomes sodden and uncomfortable; in a rumenotomy it also becomes severely contaminated. For the same reasons the use of gloves for these procedures is often impractical too. A good

Fig. 66.1

A Stent bandage. The cylinder is made of rolled up gauze swabs; it should be sutured firmly, tenting the surrounding skin at the suture attachment points. It can be left in place sleeved gown worn over a disposable plastic apron.

for about 48 hours, although it must be removed earlier if it

Even if only freshly laundered and not sterilized they

becomes sodden. It protects the wound and exerts inward pres-

are still infinitely preferable to the calving gowns which

sure which prevents fluid accumulation.

many practitioners use. If these are the same garments

which are used for routine obstetrics they will

inevitably be grossly contaminated and will impregnate

the wound edges with a good variety of pathogenic

Cows can afford to lose a great deal of blood before

organisms.

showing any ill-effects, but even if no haemostasis is

Having eschewed gloves for abdominal surgery, there

done they will never lose more than a few hundred mil-

are some occasions when they should be used. The

lilitres during a routine procedure. Consequently only

hands of a dairy veterinary surgeon are, of necessity, fre-

those vessels which are a persistent nuisance should

quently in contact with faecal or septic material and

have time spent on them. The others will dry up very

even with a full surgical scrub they are unlikely ever to

rapidly. The most efficient method of dealing with them

be surgically clean. For those surgeries where maximum

is to clamp them in the tip of a pair of artery forceps

cleanliness is essential, e.g. in an umbilical hernia repair

then to twist them about eight to ten revolutions.

where non-absorbable sutures are to be buried, the

Although this advice may sound heretical to some, it is

practitioner should overcome his prejudices, accept the

sound, and surgery which is done rapidly but deftly

limitations and wear gloves!

without unnecessary frills will result in less tissue

trauma, less contamination from the environment and substantially better healing.

Surgery

For suturing, plain catgut is entirely adequate and cheap. Despite purist objections the variety obtainable

The old adage 'time is trauma' still holds true; for large in long lengths in plastic cassettes is perfectly acceptable. In its thickest size it is suitable for closing even the infection', 'time is tiredness' and practically 'time is

largest abdominal wounds and should there be infection, its rapid lysis is beneficial in that the sutures do not to be rapid but there are some things which help. Most

form a persistent nidus of sepsis. The suture wound can novices spend more time nerving themselves for the be protected effectively by means of a stent bandage next action than actually doing it and then more time (Fig. 66.1). This will help absorb wound seepage following contemplating what they have done. If these delays are lowing umbilical hernia repair or eye enucleation, and omitted then the duration of a procedure can be sharply

will effectively reduce post-operative swelling.

reduced without spending less time on the actual surgery. In addition, if gratuitous and unnecessary tissue

Rumenotomy (see p. 837)

handling is abolished, tissue trauma will be greatly reduced to the benefit of healing.

The indications for rumenotomy are: confirmation and

Another area where a great deal of time can be

treatment of traumatic reticulitis ('wire', 'hardware

wasted is that of haemostasis. The concept of a blood-

disease') and examination of the cardia, oesophageal

free field is dear to the heart of many surgical teachers

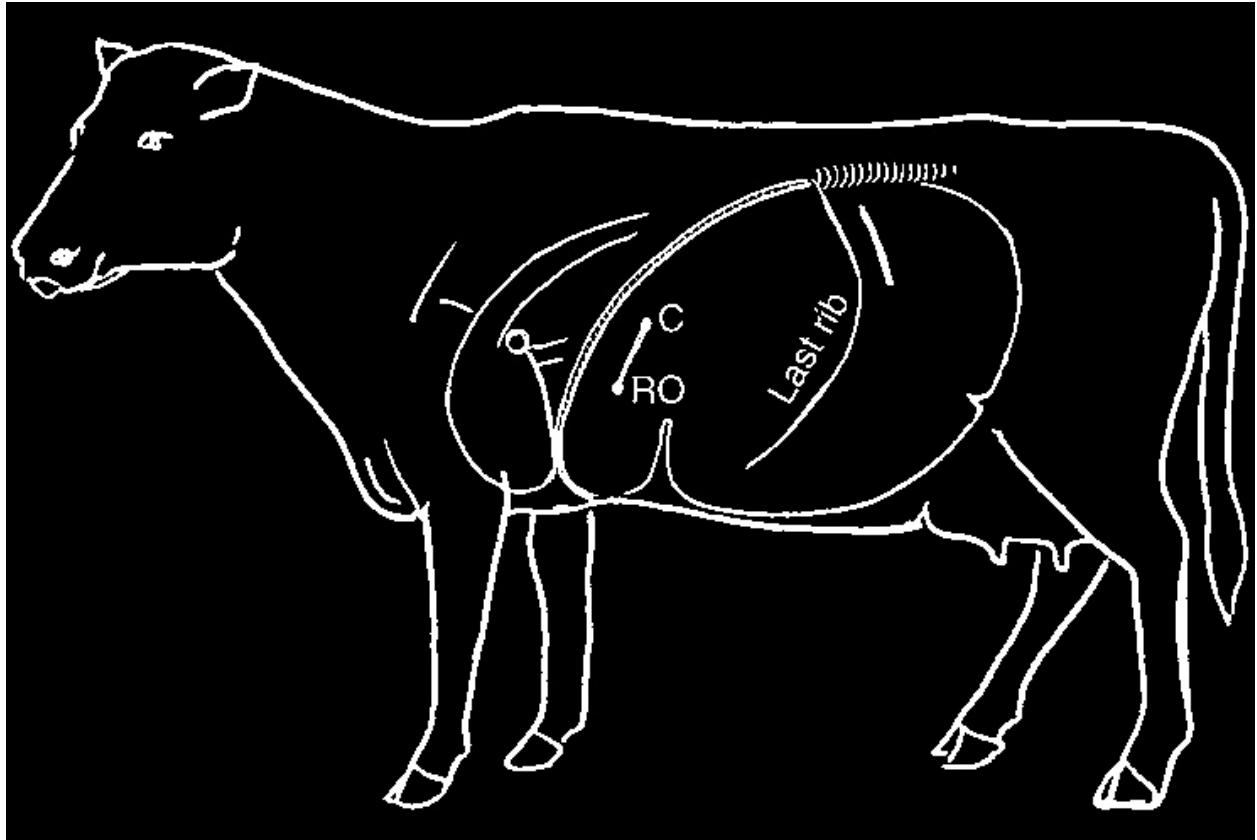
groove, reticulo-omasal orifice, reticulum and rumen.

and newcomers often devote a great deal of time and

The abdominal wall incision is also suitably placed for

effort to stopping every bleeder.

exploration of the left and caudal abdominal cavity.



Aspects of Bovine Surgery • 1107

Anaesthesia

The peritoneum must be cut carefully to avoid possible damage to the rumen wall, which is often tightly

This procedure must be done with the animal standing.

applied to its underside because of the concurrent

Fractious patients must not be given too much sedation

rumen tympany. The safest method is to pick up a fold

otherwise there is a risk of them going down during

of peritoneum in tissue forceps and to incise with a

surgery with disastrous results. In most cases, local scissor tip. Once a hole is created, air will rush into the anaesthesia of T13, L1 and L2 spinal nerves using the abdomen with a characteristic sound and the rumen paravertebral technique is satisfactory without need for should fall away. Insert two fingers into the hole and, sedatives.

with one each side of the scissor blade acting as tissue guards, lengthen the incision to its full extent.

Prior to entering the rumen it is worthwhile exploring the left cranial abdomen. Pass the right or left arm

Technique

No specialized instrumentation is needed for the technique described here. However, the surgeon should incision and then forward between the rumen and inner wear a short sleeved gown to avoid gross contamination of the sleeves and, for exploration of the rumen, the dorsal cranial rumen the free 'tongue' of the spleen gloves should not be worn as they promptly fill up with

can be palpated. In the case of a recent wire penetration the cow will begin to show signs of resentment at that they may not be able to reach the reticulum in a this point and the first adhesions may be encountered. large cow. Under these circumstances there is a definite In early cases, this feels as if the hand is separating two need for a long-armed assistant.

freshly buttered pieces of bread and their extent is surprising! The site for the incision is high up on the left flank With time, they become organized and more just caudal to the last rib (Fig. 66.2). This is so that the difficult to disrupt.

rumen can be entered above the likely level of ingesta If no adhesions are present then the reticulum can be and to minimize the distance from the incision to the reached and palpated. If this engenders no obvious pain reticulum. Following routine preparation of the area, response then it is unlikely that the animal is suffering start the incision some 6–7 cm ventral to the transverse from traumatic reticulitis and another cause for the

process and some 5–6 cm behind the last rib and con-
symptoms must be sought.

tinue ventrally for some 15 cm parallel to it. Remember

The problem of contamination of the peritoneal

that the aperture must be large enough to accommo-

cavity during rumenotomy has long worried surgeons

date easily the upper part of the surgeon's arm without

and many methods, some extremely complex, have

traumatizing the tissue. Once through the skin there is

been devised to avoid this. However the bovine peri-

no great muscle thickness, though it is important that

toneum is remarkably tolerant to the presence of the

the three mainly aponeurotic abdominal muscle layers

rumen contents in small amounts and, while excessive

be identified so that the peritoneum can be recognized

contamination is obviously to be avoided, these labori-

and dealt with carefully. During this stage, haemorrhage

ous methods are time-consuming and unnecessary.

is not usually a problem and only the occasional spurt-

In preparation for entering the rumen, place a small

ing vessel needs attention.

folded cloth in the ventral commissure of the abdomi-

L

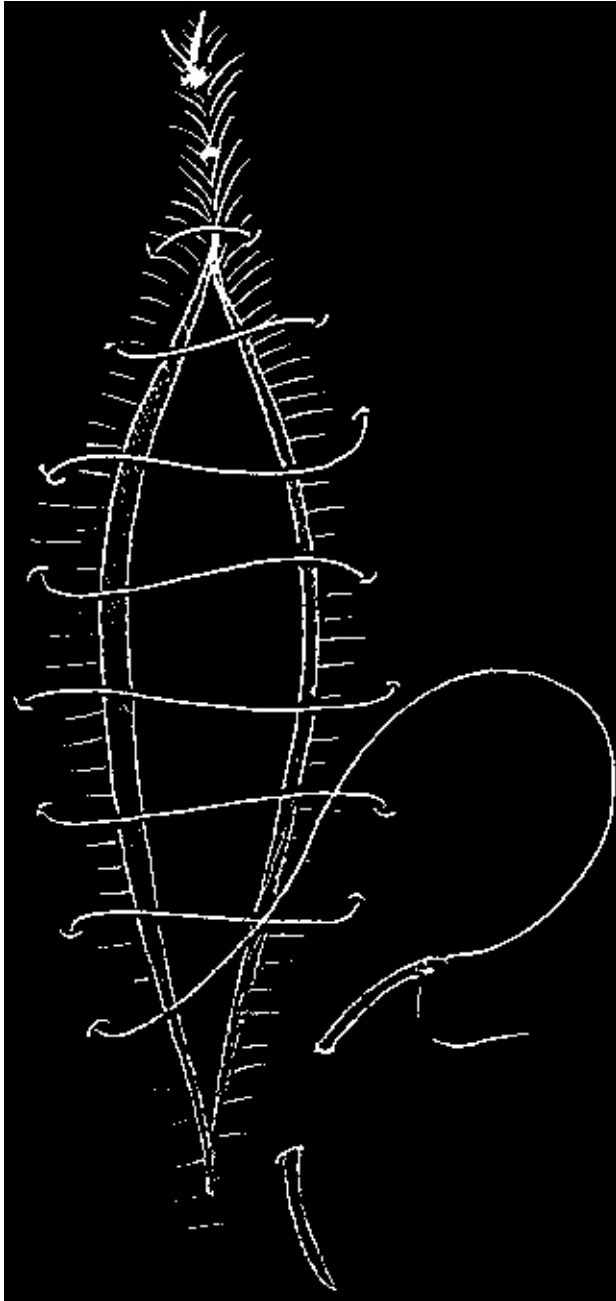
Fig. 66.2

This shows the position of the incision line (IL)

for a rumenotomy. Note that the reticulum and heart apex

are separated only by the thickness of the diaphragm. C,

cardia; RO, reticular-omasal orifice.



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nal incision so that half lies between the rumen and completely penetrated and has begun a migration peritoneum and half outside on the skin. This will mop through the abdominal or thoracic structures. While this

up most of the inevitable spillage of rumen contents.
is likely to have further grave repercussions, with
Pick up a fold of rumen wall from approximately 5–
chronic peritonitis, liver abscesses, pleurisy or peri-
6 cm caudal to the abdominal incision and exteriorize
carditis all being possible, the occasional wire can
it. Retain this vertical fold of rumen by clamping it
migrate out through the body wall or become encapsu-
firmly with two pairs of strong Vulcellum or other tissue
lated in omental tissue and be rendered harmless.
forceps, some 15 cm apart. The upper pair is held by an
At this point, terminate the search, withdraw the arm
assistant who stands on the right side of the cow and
carefully from the rumen and scrub up again while the
who is instructed to maintain a gentle upwards and out-
assistant maintains traction on the rumen incision,
wards traction.
holding it clear of the abdominal wall.
Incise the rumen fold between the two forceps and
Clean the surface of the rumen by gentle wiping with
expose the lumen. There is some haemorrhage from the

a moistened swab. Close the incision in the rumen with cut wall but haemostasis is rarely needed. Apply a third a continuous inverting pattern of absorbable suture tissue forceps to the mid point of the caudal cut edge material (Fig. 66.3). Catgut is ideal. Start the inversion and ask the assistant to maintain a mild degree of several centimetres dorsal to the dorsal commissure caudal traction to open up the rumen incision. Hold the and be sure to include the areas traumatized by the lower pair of forceps in the left hand and insert the right forceps in the inversion. Invert at least 2 cm of each hand and arm into the rumen. Follow the dorsal wall of margin and continue the inversion pattern for several the rumen downwards and forwards over the mass of centimetres ventral to the ventral commissure. Pull the contents. The reticulum is identifiable by being at arms suture material tight as each suture is laid to ensure a length, being full of fluid and having a distinct honey-watertight seal. One layer is usually sufficient but there comb pattern to its mucosa.

is always enough slack rumen for a second inverting

Sometimes the wire is immediately palpable, protruding from the reticular wall; if so, grasp it firmly and withdraw it. Note the angle and depth of penetration as abdominal cavity. Because the initial fold was taken these factors can influence the prognosis; e.g. a cranial from caudal to the abdominal incision the rumen suture penetration of even 1 or 2 cm could have impinged on the pericardium; a medial penetration could well result in the localized peritonitis affecting the vagus nerve and inducing 'vagus indigestion'. Bring out the offending wire, holding it firmly all the way, rinse the arm and explore the reticulum again. Further foreign bodies may be found within the silt that invariably lies on the reticulum floor as may all kinds of other, harmless items such as bolts, pebbles etc. Take the opportunity to examine all aspects of the reticulum, cardia, oesophageal groove, etc., for abnormalities and try to assess where the bulk of the adhesions lie. This is best

done by attempting to pick up folds of reticular wall. In the normal state this can be done fairly easily, but where induration of the wall and peritonitis exist it is impossible and attempts will be met by evidence of severe discomfort from the patient. The disposition of the inflammatory process also has implication for the prognosis; major involvement of the medial reticulo-rumen wall could well lead to damage to the stretch receptors located there.

If no foreign body or penetrating wire is discovered at the first examination, the reticulum wall must be searched carefully, honeycomb by honeycomb. If no trace of a wire is then found (and fresh adhesions are present) it must be assumed that either the wire has

Fig. 66.3

An inverting ('Cushings') type suture pattern which dropped back into the lumen and has been swept into may be used for closing an incision in the rumen, abomasum or the rumen food mass or, more commonly, the wire has uterus.

nal wall counterpart and the risks of adhesions and partial atony of the abomasum will lead to gas accumulation and instability of the organ. Normally the abomasum lies within the right, ventral aspect of the cranial abdomen; when it fills with gas it acts much as a hollow absorbable material in the peritoneum and transversus, ball pushed down into a swimming pool: it wants to rise

It is the author's practice to close the abdominal incision in three layers: an everting continuous pattern of abdomen; when it fills with gas it acts much as a hollow absorbable material in the peritoneum and transversus, ball pushed down into a swimming pool: it wants to rise a series of large simple interrupted sutures, again of and it can do so with some force! Whether the abomasum rises to the right or the left of the rumen probably depends on factors not yet fully understood, but an non-absorbable material, to close the remaining subcutaneous layers and, finally, horizontal mattress sutures of the abdominal wound appear somewhat traumatized, rumen relative to the abdominal cavity. In diets discoloured or contaminated, debridement by slicing a

*cient in roughage, as are many fed to the modern dairy
thin sliver of tissue off each incision edge will create
cow, the bulk of the rumen diminishes; its mesenteric
fresh bleeding surfaces for apposition and virtually
attachments force it to contract dorsally thus creating a
eliminate the potential for major infection and
potential space ventral to it. Nature and a closed cavity
dehiscence.*

*such as the abdomen abhors a vacuum and the nearest
In the normal course of events, removal of a pene-
mobile organ, the abomasum, becomes drawn down
trating foreign body should result in a dramatic clinical
and across to occupy a more ventral mid line position.
improvement over two to three days, recovery being
If distension occurs now, the greater curvature of the
hastened by the use of broad-spectrum, systemic
abomasum is able to rise on the left hand side. In coun-
antibiotics. Occasionally the penetrating portion of the
tries such as the UK, left side displacement seems to
wire will have instilled infected material into important
predominate while in some European studies, right side*

sites such as the pericardium and despite foreign body dilatation, displacement and associated torsion are removal the symptoms of pericarditis will progress. much more common.

Similarly induration of the medial reticulum/rumen can Factors which have been implicated in the aetiology result in the development of 'vagus indigestion' even if of atony include a high concentrate, high fat diet, surgery was successful (see p. 835).

chronic hypocalcaemia, stress of parturition, abomasal Locally, the most likely complication is the development of an abscess or abscesses at the incision line. This vagus nerve damage. Of these the most important are will show 10–15 days post surgery as a small lump or likely to be the high concentrate, high fat diets commonly fed to parturient dairy cows. Others, such as abomasal ulcers and silt impaction, are probably due to the the nidus of a buried suture and, for rapid treatment,

same aetiological factors or they are a mechanical consequence of the atony. sequence of the atony.

This is facilitated by having used interrupted sutures for closure of the bulk of the abdominal wall. Sometimes

Left displacement of the abomasum (LDA)

the sutures are difficult to locate and in this eventuality, if absorbable material such as catgut was used, the

Diagnosis is based on the presence of a non-pyrexia,

suture will lyse and disappear of its own accord, following

sudden onset 50–75 per cent drop in milk yield within

days which the abscess will dry up. If non-absorbable

the first few months after calving, coupled with a very

material was used the suture will have to be removed

characteristic selective appetite where the cow is reluctant

otherwise the infection will persist indefinitely. After

two to three weeks there is no risk involved in either

is normal, except when there is severe distension of the

searching for or removing these sutures. There will be

displaced abomasum. The faeces are characteristically

enough thickening and induration at the site to prevent firm and may be mucus covered, although occasionally accidental penetration into the abdomen, and enough and confusingly there may be diarrhoea. A ketonaemia healing to prevent dehiscence.

may be present but the condition is distinguished from simple ketosis by the local symptoms related to a distended, abnormally positioned abomasum.

Dilatation and displacement of

Auscultation over the last two or three ribs on the left ***the abomasum*** (see also pp. 839–842)

side will, when the abomasum is distended, often reveal

This is a 'production' disease. It is seen primarily in the characteristic splashing and pinging noises associated with a gas/fluid interface. However, if the cow is calving. The aetiology of condition is still not completely elucidated but is certain to be multifactorial.

lotment. This is best done by placing the right ear over

Any set of circumstances which creates a complete or

the mid point of the last-but-one rib and using one's

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knee or fist to rhythmically indent or ballot the left and the rumen and pushing the latter medially. (This lower abdomen. A second method is to tap the upper accounts for the hollow looking sublumbar fossa of last two ribs sharply while listening at the same point many LDA patients.) If one surgeon is operating on as before. If there is a gas distended viscus, i.e. aboma- both sides, open the left flank first, protect the wound sum deep to the ribs, the tapping noise will have a clear with a towel laid across it and then open the right side. resonant quality not heard in the normal cow.

The position of the right incision is critical to the

Both tests are easy to perform, take only a few success of the operation. Make a vertical incision about seconds and should be part of a routine examination of 15 cm long with its centre point about half-way up the any anorexic cow. If positive, they are virtually pathog- flank and some 6–7 cm caudal to the last rib. Higher, and nomic for a displaced abomasum. Although both 'vagus

there could be difficulty reaching the abomasum.

indigestion' and the much rarer pararuminal abscess

Lower, and the small intestine will persist in prolapsing

can under some circumstances produce similar noises,

through the incision. The internal abdominal oblique

these will extend much further caudally in the flank and

muscle is the thickest at this point and its fibres, cut

be associated with other symptoms.

obliquely, hide some large vessels. Only the largest of

The absence of these characteristic sounds does not

these need to be dealt with and they can be stopped

mean that an LDA is not the cause of the problem, only

quickly by clamping with artery forceps. Do not leave

that the abomasum is not distended with gas at the time

these hanging on the wound as they will inevitably fall

of the examination. Any previous vigorous movement,

off or be carried accidentally into the abdomen. Cut the

e.g. being brought in from the collecting yard, can result

peritoneum carefully with guarded scissors. Visible

in the escape of gas and fluid from the abomasum,

through the incision will be a curtain of fat-filled mesen-

either back to the reticulum or forward to the duodenum and omentum, with the descending duodenum. If possible, re-examine the animal in about half-running horizontally across the middle of the incision an-hour or come back the next day. Sometimes several and separating them.

examinations are necessary.

Lubricate the right arm and pass it into the incision downwards and slightly forwards between the abdomi-

Treatment: All the techniques described, and there are nal wall and the omental sheet. Advance across the mid

many, are directed towards returning the abomasum to line and as far over to the left as possible. Simultaneously the right and anchoring it there. The most commonly the assistant on the left starts to deflate the abomasum by rhythmic downwards ballotment using the author does recognize that others or variations may be flat of the hand or a fist. Even if at first the abomasum used successfully. The 'toggle' method is described on seems reluctant to empty, persistent intermittent pres-

page 842.

sure will usually cause release of gas and fluid through the pylorus or cardia with a characteristic burbling

The bilateral flank technique: Of those techniques done sound. As the body of the abomasum becomes slacker

in the standing animal, this is probably the most consistent. As it is pushed further and further down the left flank to consistently successful. Its great advantage is the ease with which the surgeon's awaiting hand. Grasp a large handful of the tissue which is being pushed ventrally to the right. This is especially important when the organ is distended and attempt to draw it across the mid line. Establish a tightly distended and apparently immobile. Its only real disadvantage is that it does need two people, though push-pull sequence and continue until the abomasum has completely disappeared from the left flank. Someone can be a lay assistant.

times the tissue being pulled can be felt to tear; this is almost certain to be omental tissue and not abomasal
Anaesthesia: Bilateral paravertebral anaesthesia of

wall. If this happens grasp a fresh handful and try again.

T13, L1 and L2 spinal nerves. Sedation is usually not

If the abomasum does not collapse with ballotment,

required, but if needed it should be used sparingly to

relieve the initial tension by puncturing the gas cap with

avoid the possibility of the animal going down.

a 14 g needle. Further rhythmic bouncing should then

cause it to deflate. If the organ still remains fixed in

Technique: No special instrumentation is needed.

position it is worthwhile checking for adhesions

Make the left side incision as for a rumenotomy. Incise

between it and the peritoneum or rumen. They can

the peritoneum carefully as the distended abomasum

form as a consequence of the abomasal ulceration that

may be lying up against its deep face and an accidental

is present in many cases.

incision in the organ would result in its collapse with

In the majority of cases the abomasum and its abo-

serious contamination of the abdomen with acidic and

masal attachments come over easily. Pull a handful of

irritant abomasal contents. Through the incision the

the displaced tissue up into the right incision. First to abomasum is seen lying between the abdominal wall appear will be folds of fat-filled omentum. Search then Aspects of Bovine Surgery • 1111

for an area where the fat deposits are thickest and being composed of rectus sheath and aponeurotic firmest. In this region lies the pylorus which is instantly rectus abdominis muscle. On entering the abdomen, recognizable as an obvious, shiny, serosa-covered blind- identify the abomasum which is likely to have risen to ending sac. Occasionally some degree of upwards trac- lie at, or near the incision line. Push the organ, prob- tion on the omentum is needed before the pylorus ably in a collapsed state, over onto the right side, identi- comes into view. Palpate the pylorus, the pyloric antrum fying the attachment of the greater omentum to the and the body of the abomasum to determine whether greater curvature. Grasp a fold of the greater omentum there is any abnormality such as gravel impaction. If all some 7–10 cm from, and parallel to the insertion of the is well proceed with the omentopexy.

omentum to the abomasum and incorporate it in the

The earliest techniques sutured the pylorus to the closure of the peritoneal incision. This is done with a inner face of the abdominal wound. However, this put continuous simple suture of catgut or similar. This has too great a tension on the abomasum and animals the effect of interposing a barrier between the abomasum and the left side, preventing displacement in that tice is to allow the pylorus to sink back to a more direction or upwards on the right.

anatomically correct position and anchor it via its

Close the fibrous aponeurosis with strong non-omental attachments.

absorbable sutures laid in simple interrupted fashion

Grasp a fold of thick omentum some 12–15 cm from

(absorbable material is unlikely to remain strong

the pylorus and fix it with Vulcellum or similar forceps.

enough for long enough in this high stress, poor healing

Start the abdominal incision closure with a continuous

site). Coapt the skin with horizontal mattress sutures

*simple suture pattern picking up peritoneum and trans-
and protect the wound with a Stent bandage (Fig. 66.1)
versus, but with every deep needle pass pick up a deep
sewn firmly in place.*

*bite of the omental fold. Do this for the whole length of the incision in order to
ensure a strong adhesion.*

*Other techniques: A non-surgical method for correc-
Then close both wounds in the routine manner as
tion of LDA is the 'rolling technique'. Although quite
described for the rumenotomy.*

*effective at correcting the displacement, its major draw-
With this technique there is usually a good strong
back is the very high rate of recurrence. It was popular
adhesion formed and the recurrence rate is low.*

*in the early days following the discovery of the condi-
However, some do tear away and the owner should be
tion, but it does have a place in modern therapy as a
instructed to lessen these chances by feeding a high
means of returning the abomasum to its correct posi-
roughage, low concentrate diet for 7–10 days and
tion prior to surgical omentopexy by a surgeon working*

keeping the animal quiet, in a loose box if possible.

through a single, right sided incision.

An alternative technique is performed through a

Cast the cow onto her right side and hobble her. Pull

paramedian incision. It has the advantage of needing

her gently onto her back (dorsal recumbency). Rock

only one surgeon, though the animal must be heavily

her gently over this 90° arc for two to three minutes to

sedated or under general anaesthesia. This in itself

allow time for the gas-filled abomasum to move from

carries some risk in the cow and the animal must be

the right to a higher position, i.e. into the ventral

starved of food and water for 24 hours prior to surgery

abdomen. Finally, allow her to roll into left side recum-

to minimize the risk of regurgitation. Also heavy faecal

bency when the buoyant abomasum should slide

matting of the ventral abdomen in some cows can make

around to her, now uppermost right side. If this is done

asepsis of the surgical site impossible to achieve.

as a prelude to surgery, the operation should commence

Position the animal on its back (wedge-shaped sup-

immediately as redisplacement can occur within a very short time.

and support her firmly. Prepare the area that lies in the

Another technique used by single-handed surgeons triangle between the costal arches, then if necessary

is to deflate the distended abomasum using a 14 g carry out a 'field block' to anaesthetize the incision site.

needle attached to a length of giving-set tubing. The

This lies some 5 cm from the mid line with its cranial

needle is taken into the abdomen guarded by the left

margin some 20–25 cm behind the xiphisternum. Be sure

hand, passed between the abdominal wall and the

to identify the course of the mammary vein before

omentum, lateral to the rumen and up to the abomasal

cutting as this runs within 10 cm of the mid line in places.

gas-cap. The wall is punctured, as much gas as possible

The 'milk-well' or foramen through which the vein

released and the partially deflated organ manhandled

enters the abdomen can usually be easily palpated and

into its correct position. The drawback of this method

this gives a guide to the course of the vein which, as the is that the needle often becomes blocked and it is some- animal lies in dorsal recumbency, is completely collapsed. times impossible to deflate the abomasum sufficiently Incise the skin parallel to the mid line; below, the to reposition it. A larger needle gauge would help avoid abdominal wall at this point is almost entirely fibrous, this, but would increase the risk of subsequent leakage.

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Some techniques have been developed which involve and, frequently, diarrhoea and melaena. A marked dif- operating through the left flank alone. One such ference between this condition and LDA is that right method attempts to create a barrier to redisplacement side dilatation and displacement frequently progresses by suturing either the omentum or the actual rumen to a complete torsion of the organ around either a wall to the peritoneum and musculature of the left mid caudo-cranial, vertical or transverse axis. This leads to flank. The abomasum is first repositioned and a series compromised blood supply to the organ, sequestration

of sutures is laid to create the adhesions. This is a rather of fluid and gas within the lumen, further distension and imprecise method and the results are distinctly variable. a marked increase in the severity of the symptoms. The The rumen can occasionally be penetrated resulting in animal becomes profoundly depressed, totally anorexic abscess formation. Another method involves placing and the heart rate increases to between 120–140 bpm. long, 100 cm (3–4 feet), heavy synthetic sutures through The aetiology of the condition is probably similar to the cranio-ventral fundic area of the abomasum. After LDA. However, in many cases of RDA large amounts deflation, the organ is repositioned whilst keeping the of sand or silt are found, often impacted into the pyloric free ends of the sutures outside the incision. These are antrum. There is still dispute as to whether this is cause threaded, one at a time, on to a large straight needle or effect, although it does suggest either a high intake which is then taken into the abdomen and used to pen- of mineral matter, e.g. through the feeding of root crops, etrate the ventral abdominal wall immediately caudal

or possibly a chronic, low-grade atony which leads to to the xiphoid process. The sutures are tensioned to the reduced passage and hence accumulation of this bring the abomasal wall into firm contact with the perisolid material. Other suggested aetiologies are mechanitoneum and then tied outside. Again, the aim is to cal strictures, or pyloric dysfunction consequent to abo- create adhesions which, this time, it is hoped will bind masal ulceration or maybe pyloric nerve damage. the abomasum into its correct position. The sutures are

Diagnosis is by detection of a distended viscus on the left in place for three to four days before being right side. Auscultation and ballotment of both flanks removed. It is suggested that because of the risk of take little time and should be a routine part of any leakage of abomasal contents, both intraperitoneal and clinical examination. The sounds are identical to those systemic antibiotics are used. Although the author has heard in a case of LDA and they occur in a mirror- no personal experience of these techniques, both have image position, i.e. over the last two ribs. If dilatation is

their advocates, with the latter method especially being severe or torsion has occurred, the distended viscus can popular in some countries.

often be seen distending the cranial, right sublumbar
A final technique, which is occasionally described is fossa just behind the last rib. The organ can also be felt known as the ‘ventral abdominal blind approach’. It has per rectum as a spherical, taut structure at arm’s reach few adherents because of its imprecision and the very on the right side of the abdomen. The animal’s general high risks of serious complications. The cow is cast in demeanour, and especially the heart rate, will indicate dorsal recumbency and an area behind the xiphoid is whether torsion has occurred.

surgically prepared. The animal is rocked from right to left until the abomasum is auscultated in its normal
Anaesthesia: A right side paravertebral block of T13, position. A large, full-curved needle threaded with 6 L1, and L2 spinal nerves or a field block.

mm (1/4 inch) cotton or synthetic tape is passed blindly through the abdominal wall, hopefully through the abo-

Technique: The only special item of equipment needed is a stiff plastic tube, about 1.5 m long and with a bore of some 1 cm. This will be used to syphon off the fluid allow adhesions to form before being removed.

sequestered within the abomasum prior to correction of the displacement or torsion. If available, a stirrup type pump is also useful to speed up the evacuation.

Abomasal dilatation and right sided

Make an incision in the right flank as if for an LDA displacement (RDA) (see p. 842)

correction. Palpate the swollen abomasum and try to

In the UK this variation is seen much less frequently assess visually the condition of its wall. In general terms, than left side dilation and displacement. As with LDA the larger the organ and the darker its colouration, the the symptoms are usually manifest within the first few less favourable is the prognosis; where torsion has been weeks after parturition. Insidious in onset, with inapparent for some hours it can be enormous, contain

petance, reduction in milk yield and a variable ketosis, many gallons of fluid and, when its blood supply has the animal's condition soon deteriorates with signs of been compromised, it will be a dull, dark reddish-blue abdominal pain shown by lifting of the hind legs in colour.

towards the belly and looking around at the flank.

The first step in the management of this formidable

There is elevation of the heart rate to about 100 bpm

looking organ is to reduce the tension in its gas-cap.

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Pierce the most dorsal accessible point with a 14 g

the section on LDA), grasp it firmly between the thumb

needle attached to a length of plastic giving-set tube.

and forefinger and incise parallel to the lumen axis

When the wall is reasonably slack, insert a purse-string

through the serosa, circular muscle and submucosal

suture in the serosa and muscularis layers in a position

layers until the mucosa bulges from the inside. (If the

above the fluid line. Make a small incision within the

mucosa is accidentally punctured it can be repaired with

purse-string and thrust the plastic tube through into the a catgut suture.) An alternative method is to carry out lumen, drawing the suture tightly around it. Foul- the procedure while the abomasal evacuation incision is smelling, coffee-coloured and often blood-stained fluid still open, inserting an index finger into the sphincter will run out of the tube and can be collected for disposal.

lumen and cutting down very carefully on to the site indi-The stirrup pump will speed up the process and can be

cated. When these procedures are complete the abomasum is anchored in the usual manner.

The next step is to reposition the abomasum, a relatively simple procedure if only dilatation is present.

abomasum after its deflation and repositioning. If the However, in a case of torsion the direction of the twist discoloured wall assumes a more normal healthy colour must first be established.

and acquires a degree of tone, the outlook is reasonable. After removing the evacuation tube and tightening

able. If it stays discoloured and flaccid the prognosis the purse-string suture, try and feel at the extremities is not good. Pre- and postoperative fluid replacement of the organ, i.e. the omaso-abomasal junction and the pyloric end, to see if the twisted tissue there gives some choice of fluid should take into account the massive indication of the torsion axis. If not, a process of trial sequestration of sodium and chloride ions that has and error repositionings often proves successful. In the occurred. Intravenous normal saline or Ringer's (not author's experience most torsions occur along a caudo-bicarbonate-containing Hartmann's) solution at 5 l/h to cranial axis with the direction of twist being anticlock- a 450 kg cow has been recommended. This can be wise when viewed from behind, so it seems logical to 'spiked' with potassium (up to 40 mmol/l). try correcting that version first. Using the flat of the left In the first days after surgery the patient should hand on the most dorsal part of the partly distended receive routine antibiotics and be fed small quantities of

abomasum, push downwards and obliquely forwards; if hay only, though with unlimited access to water. During this is the correct direction, the twist unravels itself and this period, foul smelling diarrhoea is inevitable but the area reverts to a relatively normal anatomical state. temporary.

If not, the organ will resume its previous abnormal position, begin to distend and another direction must be

Intestinal obstruction (see p. 847)

tried. Fortunately, gentle perseverance usually pays.

Once repositioned, the abomasum can be anchored by

The theoretical causes of intestinal obstruction include the standard omentopexy technique.

volvulus, gut tie, strangulation in scrotal or umbilical

Unfortunately, there is a high rate of recurrence of hernias, caecal torsion and others. However, intussus- this condition. Many authors, believing that pyloric dys- ception would seem to be the one most commonly function contributes greatly to the development and encountered in practice situations. It may be seen in any persistence of the syndrome, advocate either a pyloro-

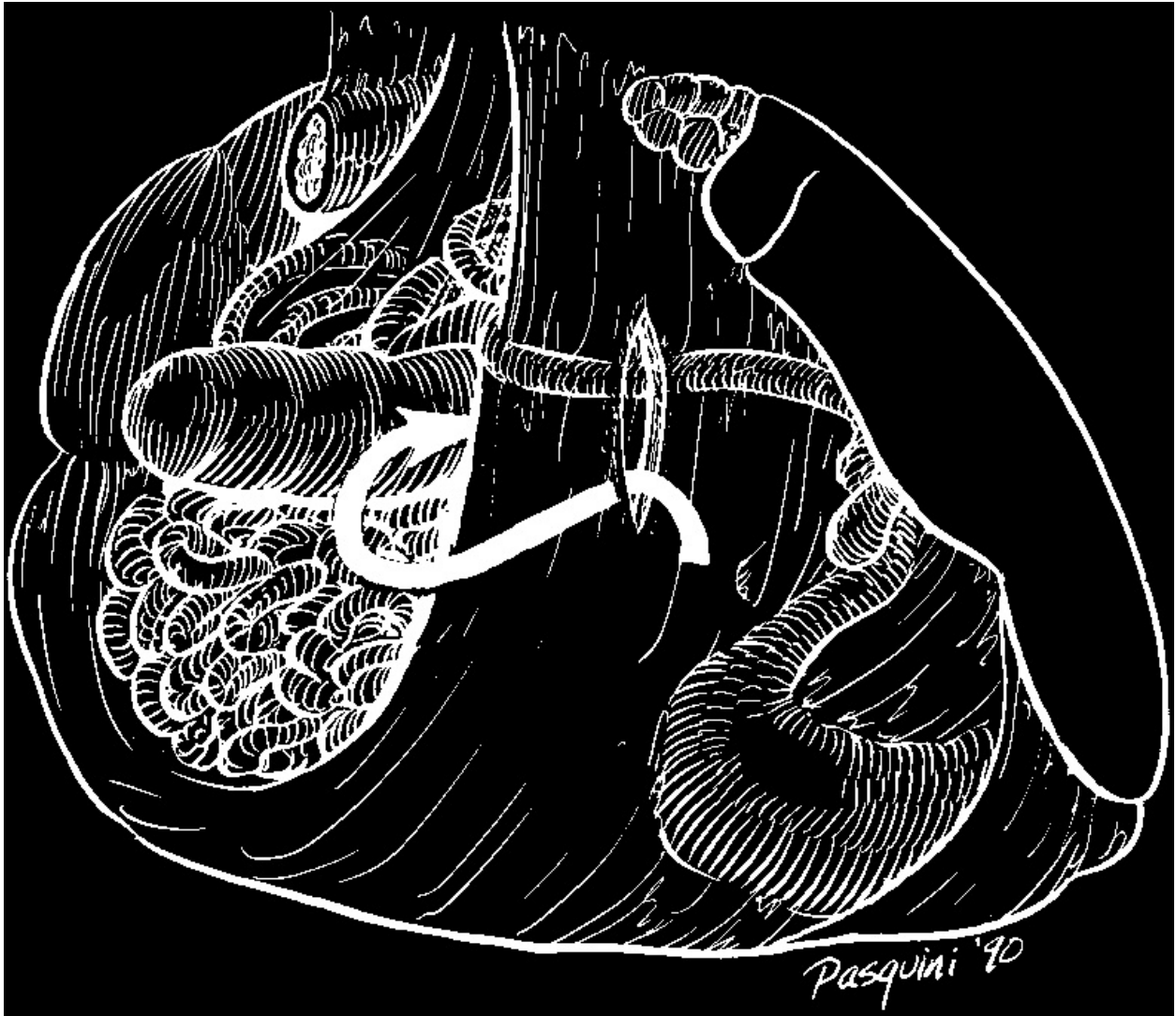
*age of animal, but most often is encountered in young
plasty or a pyloromyotomy with or without evacuation
stock.*

of silt.

*Acute intestinal obstruction differs from most of the
Although there is not enough information available
other abdominal conditions in that it causes abdominal
to decide which combination is the correct one, it seems
pain which manifests as a colic-like syndrome. The
illogical not to carry out the relatively simple pyloric
bovine reaction to this is much more subdued than the
surgery and to remove any silt which seems to be
more excitable horse, but it does show in restlessness,
forming an obstructive mass. This author has had
with frequent getting up and down, looking around at
encouraging results with the simpler pyloromyotomy
the flanks and lifting of the hind legs to kick forward at
coupled with silt removal.*

*the belly. Some people describe a 'trestle'-like stance
To evacuate the sand, exteriorize a portion of the
which affected animals adopt with a dipped back and*

flaccid abomasum, make a 10 cm incision in the wall and, spread legs. Rectal temperature can vary and is no having protected the abdominal wound margins with a guide but as the condition progresses the heart rate will sterile towel, manually remove the contents. Close the cross the 100 bpm barrier and reach a high of 120– wound in two layers; firstly the mucosa with a simple con- 130 bpm. Faeces are passed at first but on the second tinuous pattern and then the muscularis and serosa with day only scant, tarry material is seen. The colic phase a continuous inverting suture, both of catgut. To perform may only last 12 hours or so and the animal often looks the myotomy, identify the thick muscular spincter (see brighter for a time before becoming dull and depressed



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*as the obstruction leads to gut necrosis, peritonitis, tox-
preferably under paravertebral anaesthesia of T13, L1
aemia and shock.*

and L2 spinal nerves. Avoid making the incision too low

*This set of signs presented to the clinician fairly obvi-
in the flank otherwise small intestinal loops will persist*

ously indicates an abdominal problem and in theory in prolapsing out of the wound. The small intestinal there are a number of conditions to consider. However, mass is reached by first going caudally between the absence of a distended single viscus rules out LDA, duodenum and the flank, then medially and finally RDA or torsion. Hernias can be examined externally cranially into the omental sling, or recess in which the for signs of strangulation and traumatic reticulitis can bowel loops lie (Fig. 66.4). The intussusception is be virtually eliminated by, amongst other differences, a usually easy to feel and once located it should be failure to elicit pain by ballotment of the xiphoid region exteriorized. Sometimes the mass will not come to the and by the fact that it rarely causes such an elevated incision. In these cases surgery cannot be performed heart rate or signs of colic. and the procedure must be abandoned. Most, however, A rectal examination is important both for elimina- can be exposed although the mesentery is short and tion and possible positive findings. Where an obstruc-

access to the affected area and to the surrounding
tion is present, some distended small intestinal loops
normal bowel is always limited. This fact makes the
may be palpable in the right abdomen and occasionally,
help of an assistant essential since a constant traction has
in small cows, an intussusception can be felt as a
to be maintained to keep the mass in view of the
sausage-shaped mass about 6 cm in diameter and of
surgeon.

variable length. However, distended gut loops alone or
Several additional problems now have to be over-
the presence of taut bands of mesentery are a strong
come. In other species it is usual to remove the intus-
indication of an abdominal catastrophe and the need
susception along with a reasonable length of normal
for an exploratory laparotomy.

bowel each side so as to ensure viability of the anasto-
Bearing in mind the simplicity and straightforward
mosis. In the cow, however, the mesenteric frill is so
nature of the procedure, a laparotomy should be
short that only the intussusception itself and a minimal

regarded as a diagnostic tool; there is an immense margin of normal gut can be removed otherwise the two amount of diagnostic and prognostic information to be cut ends might not come together.

gained and there is the opportunity for immediate cor-

Before attempting resection, it is worthwhile seeing rective surgery; it is therefore surprising how often it is if the invaginated length (the intussusceptum) can be shunned by both veterinary surgeon and owner.

pulled clear of the enveloping bowel (intussusciens).

If this can be done easily and the wall looks pink and

Surgical correction of intussusception: Make the

healthy then there is no need for resection. Usually

laparotomy incision in the mid point of the right flank,

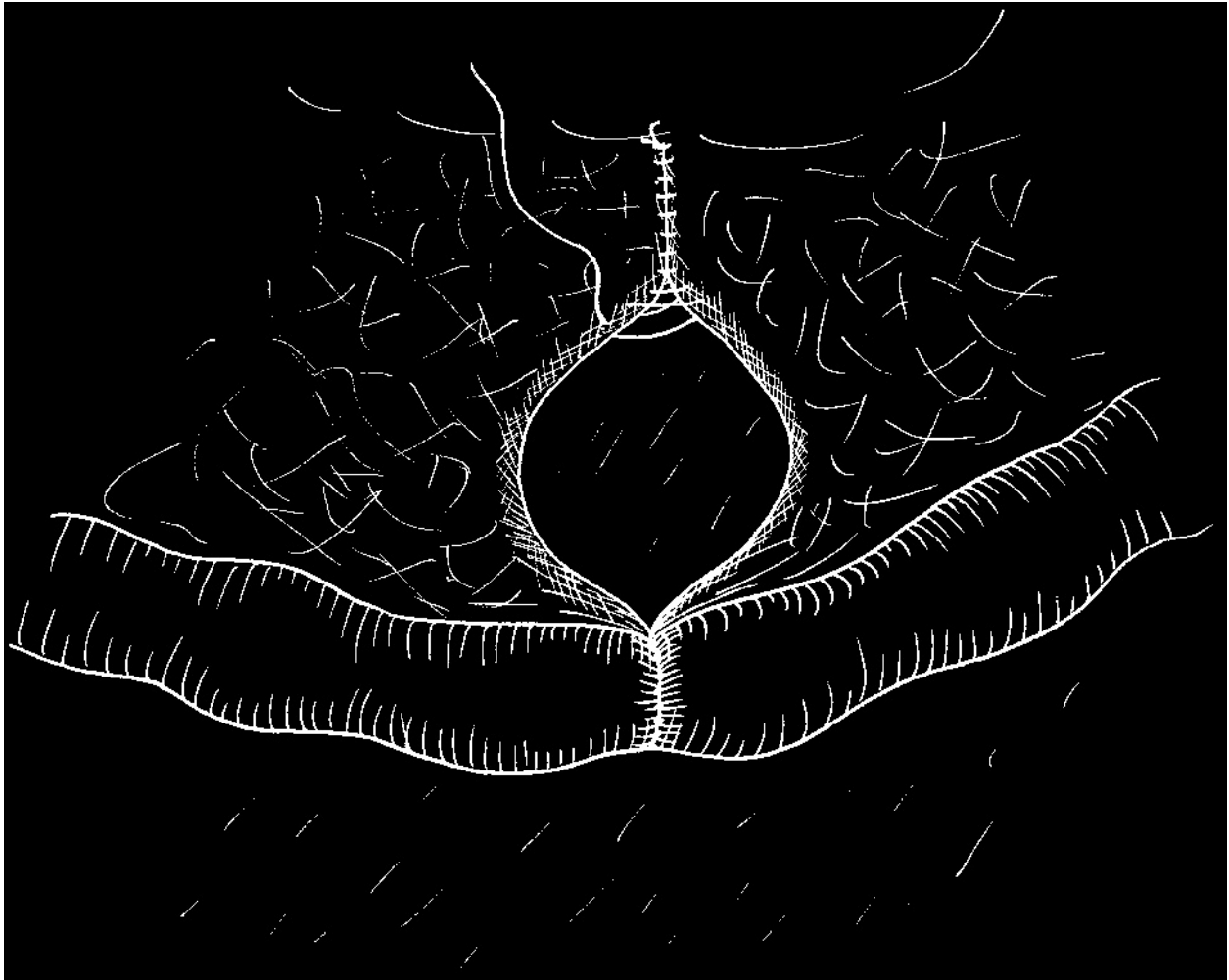
though, adhesions have fixed the two parts together or

Fig. 66.4

This illustrates the route taken to gain

access to the small intestine from a right flank

laparotomy incision. (Courtesy of Chris Pasquini.)



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Fig. 66.5

This illustrates the problem in closing the defect in the mesentery following a small bowel resection.

if they are separable the intussusceptum is discoloured

chosen, insert the first four sutures at the poles of the

and friable and must be removed.

lumen, the first being inserted at the mesenteric border.

Use bowel clamps to isolate the intussusception.

There is likely to be a disparity in size of the lumen of (Use the minimum compression needed to avoid the proximal and distal segments and if this disparity is trauma to the remaining gut.) Place each pair about large then apposition may be difficult. If there is enough 5 cm apart and try to ensure that the bowel between slack, the distal stump can be cut at an angle to try and them has a good blood supply. Again, unlike other equate the circumferences (see Fig. 66.5). An alternative species the cow provides unique problems, as the blood is to take longer bites of the proximal end to try vessels to the small intestine, although large, are completely obscured under a thick layer of mesenteric fat; and pull it down to the size of its opposing partner. When completed, test the seal by removing the ligating individual vessels supplying the intussusception clamps and massaging gut contents at pressure through is therefore difficult. To ensure that all are sealed start the anastomosis. If it holds, the next step is to close the the ligation procedure at the level of one of the inner hole in the mesentery. Use an absorbable material

clamps. Take bites of the mesentery and fat with strong (catgut) and a continuous simple pattern, starting from absorbable suture material and lay down an arc of over-the inner aspect of the defect and working outwards. lapping blind ligatures from one end of the intussus- Because of its short length that part of the mesentery ception to the other. Occasionally large lymph nodes adjacent to the bowel may not come together (Fig. 66.5) will get in the way and the ligature line must deviate and the hole so formed will have to remain.

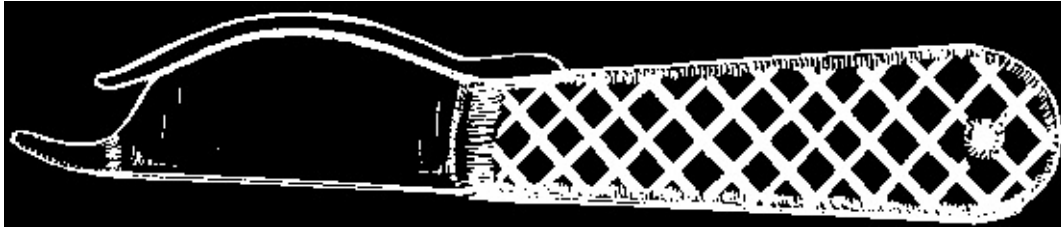
around these. When all the ligatures have been laid, Clean the anastomosis, return the bowel to its correct resect the affected length, cutting the bowel close to the position and close the abdominal incision routinely as inner set of clamps. This leaves a good length of free described elsewhere in this chapter.

bowel protruding from the remaining forceps and makes the subsequent surgery easier.

Caesarean section

Either an everting or inverting suture pattern can be used to join the two ends, although the latter gives a

This is probably the commonest major surgical procedure for securing a more secure union with better healing. Whichever is more durable in cattle practice. It is carried out in all sorts of cir-



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cumstances; from the ordered serenity of a teaching school clinic to the murky depths of small hill-farm barns at 3 am by torch or car headlight. It is therefore a procedure performed in sometimes difficult and adverse conditions, often by a surgeon tired by the

Fig. 66.6

A Charles Roberts embryotomy knife.

attempt to calve the animal, and frequently under pressure of other calls to attend. It therefore, more than most, requires the establishment of a routine which can not work. This is presumably because gravity causes the be carried out in all foreseeable circumstances. The technique anaesthetic solution to affect only the dependent nique described here is a routine which the author has

side and the straining reflex is an 'all-or-nothing' developed over the years and which has worked successfully and consistently.

Technique: This technique requires the use of a Charles Indications: These are dealt with exhaustively else-Roberts embryotomy knife (Fig. 66.6), but no other where. However, one point needs to be stressed: the special instruments.

decision to carry out a caesarean section is one that If the surgery is to be carried out in the standing position, put the patient's right side up against a barrier of many of these procedures are carried out after heroic some sort to prevent her swinging away. Mild restraint attempts at normal delivery and avoiding surgery have can be achieved with a nose twitch or 'bull-dog' but, if failed, with a bone weary clinician operating reluctantly at all possible, always have someone standing at her on an exhausted and traumatized patient.

head to exert control if necessary. If the cow is down, or cast, position her in sternal recumbency with her

Anaesthesia: A decision must be made early whether hind-quarters tipped towards the right. Hobble both to operate on a standing or recumbent animal. The fore and the right hind legs together but pull the left standing position is infinitely preferable so if the cow hind gently backwards to stretch the left flank. Take is tractable, use little or no sedation, merely manual care not to put too much traction on the leg as this can restraint. If the animal is wild enough to require heavy cause peripheral nerve injury. Again it is sensible to sedation then the author recommends that the animal have someone at the cow's head to restrain her if she be cast at the outset and hobbled to keep her down. attempts to get up. Some surgeons operate with the Nothing is more chaotic than operating on an ataxic animal in full lateral recumbency. This provides for cow struggling to keep her feet and collapsing, good restraint, but lifting a calf out of the abdomen at inevitably on the wrong side during surgery. arms' length with bent back is extremely difficult and Anaesthesia of the flank is best achieved by paraver-

uncomfortable, not to say dangerous and the author
tebral block of T13, L1, L2 and L3 spinal nerves, the
does not recommend it for this reason alone. In addi-
latter included because the top 2 cm or so of the inci-
tion there is the distinct impression that abdominal
sion often intrudes into this dermatome. In addition the
wound closure is more difficult with more tension being
author strongly recommends epidural anaesthesia for
required, probably because of the increased upward
all caesareans other than early elective cases. If not
intra-abdominal pressure and the convexity of the
done, any cervical stimulation, for example by the calf
upper flank.

moving against it, will trigger abdominal contractions
The only technique to be described here will be the
even in exhausted cows. At best these are disconcert-
sloping, left flank incision principally because the
ing, at worst they can cause massive prolapse of the
author has had no indication to do any other. Right side
rumen through the abdominal incision. However, the
'caesareans' should be avoided because of the propen-

epidural injection must be done with care since too much volume will cause the cow to start knuckling on the abdominal incision for the duration of the surgery. her hind legs (usually the right one first) and to become Occasionallly though, old left side lesions may make this unsteady on her feet. Only the barest minimum, sufficient to stop straining should be given and this is usually plication of operating on the right side.

the same as that required to produce vulval anaesthesia. Begin the abdominal incision about 12–15 cm ventral to the tuber coxae ('pin-bone') and, at an angle of about 30–35° to the vertical, extend it downwards and forwards. Make the incision long enough to remove the calf if already down, with the animal's hindquarters in a anticipated size of calf at the outset. Extending it later, horizontal position. If the animal's pelvis is tipped to

when the calf is stuck half way out, is difficult; an adequate size makes for easier delivery and much less

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trauma to the wound edges. Cut through the fatty subcutaneous fascia and the external abdominal oblique muscle whose fibres cross the incision at right angles of any damage being done with this instrument. Hold and which, at about this level, merge with the relatively the knife in the palm of the left hand with the blunt, avascular aponeurosis. Deep to this lies the principal probe-headed blade away from the palm and pointing muscle layer, the thick internal abdominal oblique with forwards. Push this blunt point vigorously through the its fibres running approximately parallel to the incision. uterus wall over the extremity of the calf, then push Reverse the scalpel and with its blunt handle separate the knife along the greater curvature of the uterus for a small gap in these fibres at mid incision level. Insert

the required distance, preventing loops of bowel from the index finger of each hand into this gap and pull possibly straying into the cutting area by using the first apart. The muscle then splits easily and bloodlessly two fingers of the same hand extended alongside the along the incision line to the required length.

knife guard. If the knife is sharp it cuts effortlessly, Below lies the transversus muscle whose fibres run, pushing cotyledons aside with its blunt probe tip and like the external oblique, across the incision. At the mid thus minimizing haemorrhage.

point there is usually a fairly large segmental nerve and To remove the calf grasp a leg, through the fetal accompanying blood vessels and it pays to ligate this membranes if they are not cut, and bring it to the bundle before cutting. For some reason this nerve abdominal incision. This may necessitate careful branch often seems to retain sensation, so it is wise to manipulation and flexion of the joints, especially in the stand away from the hind leg whilst it is quickly cut. case of the hind limbs. Deal with each leg separately,

Take care cutting through the peritoneum as the rumen and the head too if the calf is in posterior presentation. is usually tightly applied to its deep face.

Break through the fetal membranes and extract the calf

Pass the right hand and arm behind the rumen and by lifting it as gently as possible from the incision.

then medially to feel the uterus lying in the mid and

Remember that some part of the calf may be within the right caudal abdomen. Palpate it thoroughly to feel the pelvis and traction downwards and cranially may be

lie of the calf within. If the calf is in anterior presenta-

needed. If the calf is alive, try not to let it fall and snap

tion relative to the cervix, feel for the distinctive outline

the umbilical cord precipitously; rather move the calf

of the hocks and, if in posterior presentation, locate the

away from the cow slowly, breaking the cord in a con-

head and nose. Once these are found the uterine inci-

trolled movement. If possible, let someone else deal

sion can be made without delay.

with resuscitation of the calf and return to the cow.

The principles are: begin the incision over the extrem-

Even in this short time, the uterus will have continuity of the calf which is furthest away from the cervix (i.e. tracted considerably, but in the majority of cases it is over the hocks or nose of the calf) but not too close to perfectly feasible now to exteriorize the incised horn to the fallopian tubes or ovaries; continue the incision over make closure easier. This has to be done carefully to the dorsum or back of the calf, along the greater curvature of the uterine horn, making the aperture big enough flattened cupped palms and a scooping motion. Where to extract the calf without tearing the uterus in the the placenta has separated, the membranes can be process. However, the incision must not be so large that peeled away easily, but when the calf is delivered alive it extends too far towards the cervix and therefore to a this is not feasible and attempts to separate the fetal position which will be difficult or even impossible to and maternal parts of the cotyledonary attachments will reach and suture. As a general rule this means an incision result in severe damage and bleeding. However, if the

sion length of between 30–50 cm depending on the size placenta remains in its entirety, it will make closure very of the calf. Also the more friable the uterus (e.g. after a difficult. A simple remedy is to grasp the mass of membranes), the longer it should be to help prevent tearing. branes close to the uterine incision and to cut the bulk If these lengths sound excessive, remember that follow-away with a scalpel keeping about 10–15 cm away from ing removal of the calf, uterine contraction will substantially reduce them within minutes.

bleed transiently but this is not a problem. By this Many texts refer to exteriorizing the uterus or visualizing it before making the incision. However, few of the remaining membranes is not affected.

people are strong enough to manipulate, let alone lift Begin the uterine closure at the cervical end of the this huge and heavy organ (often 75 kg or more) at incision some 3 cm away from the wound commissure arm's length, and in trying to do so, accidental damage

and invert 2–3 cm of the wound edges to bring serosa in is often done. It is far better to learn to use a Robert's contact with serosa. Any continuous inverting suture knife which has a protected blade and is used to incise pattern will suffice but a Cushing's pattern (Fig. 66.3)

the uterus in situ. The prospect of using a knife blindly provides for the most rapid closure. Use an absorbable

in a site surrounded by small intestinal loops is daunt- material such as catgut and penetrate only the serosa

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and muscular layer. Pull each suture bite snugly as it is are far more likely in torsion cases so make sure that laid and be careful not to trap strands of placenta in the the uterine incision is made long enough to extract the closure. If a 'T' or 'Y' tear has occurred suture the calf easily. Also sutures tend to pull through the soft branch tear(s) first then treat the remainder as a single wall and it is invaluable to have an assistant scrub up to incision. If there is any doubt as to the seal achieved, help oppose the edges of the incision. Always oversee oversee the whole incision with a second inverting

in these cases and check that the torsion is completely layer. Clean the uterus of blood and fibrin tags and relieved before closing the abdomen.

replace it in the abdomen.

Close the peritoneum and transversus together with

Removal of a dead, contaminated calf: The bovine a continuous everting suture to appose peritoneum to abdominal cavity is, by and large, a tolerant organ, but peritoneum. This is not a weight-bearing layer so the the gross contamination by infected fluids, membranes, closure can be rapidly attained with large tissue bites.

hair, etc. that occurs in a caesarean delivery of long

Oppose the remaining layers of muscle and fascia with dead fetuses leads to a high incidence of maternal endo- a row of large, simple interrupted sutures. As there is toxemia, peritonitis and abdominal wound dehiscence.

tension on this part of the closure the knots can be dif-

However, with suitable modification to the basic technique difficult to tie, with the first throw slackening before the

nique the level of post operative morbidity and mor-

second throw is laid to lock it. If the first throw is made

tality drops to such a low level that surgery becomes a triple or even quadruple slippage does not usually viable alternative to sending the animal for slaughter. occur and tissue opposition can be maintained. The skin These modifications were adopted successfully by the is closed with horizontal mattress sutures of non-author and others at the Liverpool University Veterinary Field Station.

The choice of suture materials for the deep layers of In anticipation of the massive abdominal contamination the abdominal wound is the subject of controversy. tion give the animal 10 million units of crystalline penicillin G intravenously just prior to surgery. Open the material such as catgut is indicated. The thickest gauge abdomen in the conventional way but then attempt, if is easily strong enough and, in the event of infection, at all possible, to bring the gravid horn up to the these sutures will not act as persistent, individual incision. (Take care not to damage the friable uterine

foreign bodies, will lyse rapidly and will not have to be wall in the process.) Incise the horn over an extremity painstakingly removed before healing can take place. and try to extract the calf and membranes with the As soon as the surgery is over, release the cow and, minimum of spillage into the abdomen. To make this if it is alive, allow her access to the calf. In an uncom-easier, make sure that both the abdominal and uterine plicated caesarean section there is no indication to use incisions are of adequate length to prevent sticking or any drugs other than the routine pre and post operative tearing. Close the uterus carefully as described for antibiotics. However, if the uterus seemed slow to uterine torsion and oversew the first closure. Clean the contract down, as often happens when a dead calf is uterus, reposition it and then begin the task of cleaning removed, oxytocin can be given and, of course, if there out the abdominal cavity. This is achieved by repeated is any suspicion of hypocalcaemia, appropriate therapy lavage. Use 2 litres of saline each time, warmed and is indicated.

poured through the incision. Use the saline to literally wash the viscera before scooping as much as possible with cupped hands. Repeat this process twice. Instill 10

Special cases

million units of crystalline penicillin G into the peri-

Caesarean section in cases of torsion of the uterus: The toneal cavity and begin the closure.

operation proceeds routinely until the stage of incising

The margins of the abdominal wound will be severely

the uterus. If this is done without first correcting the

contaminated so remove a 4–5 mm strip of the incision

twist, suturing will be extremely difficult because in

margin of each muscle layer by sharp dissection. The

the, by then, untwisted uterus, the incision will lie on the

bleeding edges are then sutured in the usual way using

deep face of the horn. It is therefore the author's prac-

plain catgut. Strip the contaminated fascia from below

tice to try and correct the torsion at this stage if at all

the skin margins and close the skin with horizontal mat-

possible. Use the flat of each hand to start the heavy

tress sutures of a non-absorbable material. Use high

uterine body swinging in the copious peritoneal fluid levels of penicillin and streptomycin to supplement the that invariably is present in these cases. Be very careful initial intravenous therapy for about 5 days and to not to allow stray fingers to penetrate the oedematous, ensure the maintenance of high concentrations in the friable uterine wall and if this seems likely, or the twist peritoneal cavity inject 10 million units of penicillin G is resistant to correction, abandon the attempt and directly through the abdominal wall in the right sub-proceed to incise the twisted horn. Uterine wall tears lumbar fossa on the first and second post operative day.

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Despite these precautions, wound infection can occur whose success rate and ease of execution improves considerably, the earlier it is done. Too often the clinician at the ventral aspect of the wound which is painful on and owner delay the decision to amputate, just in case palpation. Needle puncture will confirm the presence of the animal improves, and are only persuaded that it is

pus. In these cases remove the sutures from the lower necessary when the infection has extended above the third of the incision and open the wound to allow hoof and the distal limb is swollen and excruciatingly drainage. Penetration of infection into the abdomen is painful. By this time, the unfortunate patient has lost a not likely at this stage and the only treatment needed great deal of condition and most of her milk, has taken is daily wound irrigation. It is in these circumstances up a great deal of time and money and is a prime candidate that the use of catgut sutures is vindicated. The material is a candidate for humane slaughter, not surgery.

Material lyses rapidly in the infected tissue and does not Probably the commonest instigating condition for form a series of permanent niduses of infection which this type of deep infection is solar ulceration. Typically would have to be removed before healing could take the lesion remains unnoticed because of its bilateral place. Whilst these open wounds look extremely nature and only when infection penetrates through the serious, provided drainage and irrigation are main-

keratogenic layer into the deeper structures does the
tained they heal well with minimal long term scarring.
lameness worsen sufficiently for the farmer to notice
and bring the case to the attention of his veterinary
Caesarean section for treatment of Hydrops allantois:
surgeon. By this time involvement of the deep flexor
Hydrollantois in the cow develops within the last three
tendon (DFT) at its insertion into the pedal bone has
months of pregnancy. It results in a massive fluid accu-
usually occurred. This extension of infection to the level
mulation within the allantoic cavity and caesarean
of the DFT is easily established by probing down
section is the treatment of choice. Initially a two stage
through the granuloma which is always present by this
operation was devised in an attempt to avoid the
stage at the ulceration site. If the probe tip strikes bone
splanchic pooling and vascular embarrassment which
or penetrates to a depth of 2.5–3 cm, then DFT involve-
might occur if the fluid was allowed to escape too
ment is certain. In these cases it is the author's practice
quickly. On the first day, a drainage tube was inserted

to excise the granuloma and the infected deep tissue into the uterus and fluid was evacuated slowly once only. If, on a repeat examination some 5–7 days overnight; on day two a routine caesarean section was later, the granuloma and sinus have reappeared, then, carried out. A development of this technique was to as the chances of success a second time are minimal, drain fluid through a larger bore tube over an hour or amputation is recommended. At this stage, infection is so before completing the caesarean procedure.

usually localized within the hoof and has not spread to The author has modified this technique further so the soft tissue of the pastern region or, worse, to the that delay is minimized.

flexor tendon sheath.

Begin the abdominal incision in the usual way but

Done at this relatively early stage, the operation is open only the top or dorsal 50 per cent of the wound.

easy to perform and is usually successful in its aims. The

The fluid-filled uterus invariably bulges out of the aper-animal heals rapidly and begins to improve in condition

ture. Make a small 2 cm stab incision into this bulging almost immediately after surgery. However, a warning portion and slowly release some of the fluid. As the note must be sounded: the remaining digit will now be intra-uterine pressure drops, slowly at first, then faster carrying all of the animal's weight. It will therefore be and faster, the abdominal and uterine incisions can be subjected to a great deal of extra wear and tear and is progressively enlarged. If the evacuation of fluid is unlikely to support the cow for only one or two more lactations; in small cows, with a good clean management show no sign of discomfort or vascular embarrassment system it can be much longer.

and the caesarean section can proceed as normal. Bear in mind, however, that the calf is likely to be small, so Anaesthesia: For the technique to be described here, the incision does not need to be as large as normal, and the animal must be sedated and cast to lie in lateral that the vastness of the uterus and prolificacy of the recumbency. For sedation use xylazine or chloral

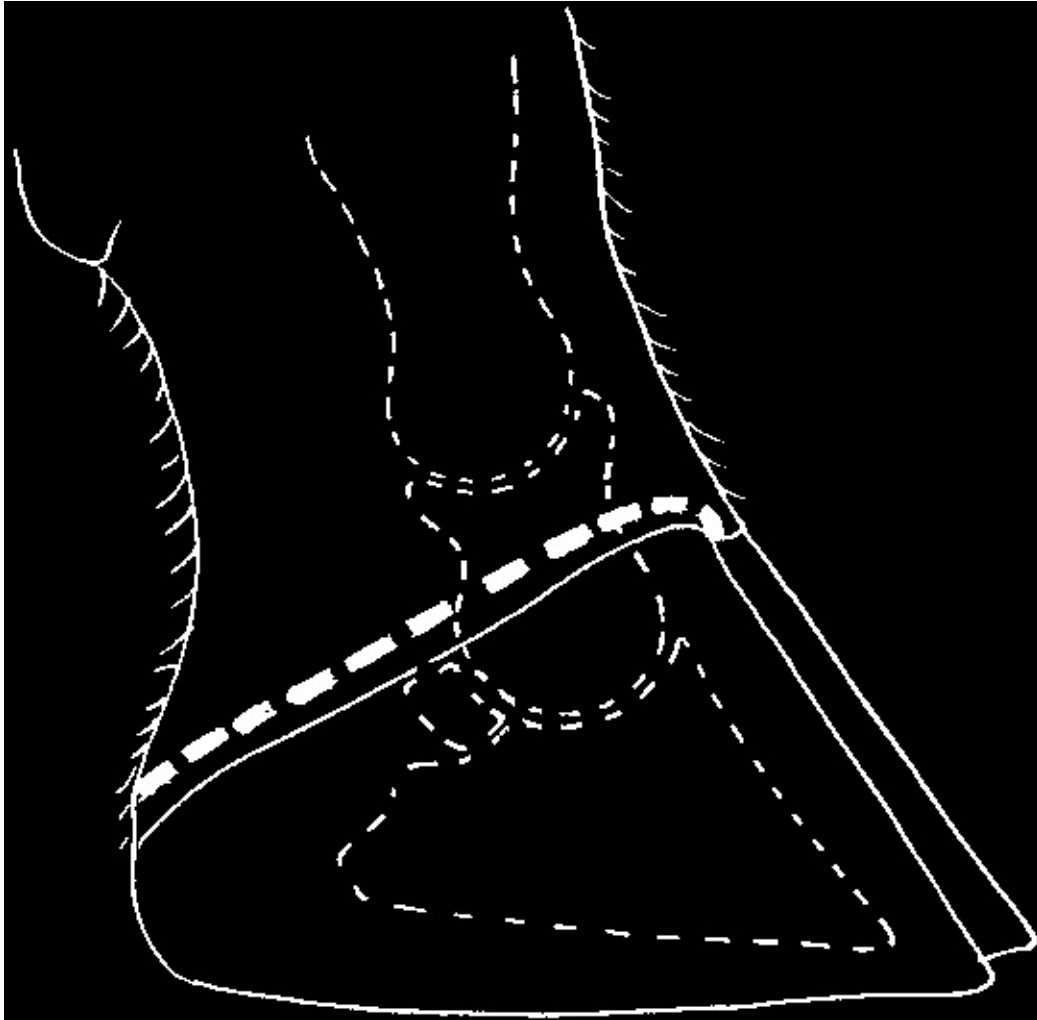
membranes can occasionally make locating that calf hydrate. The latter drug, now sadly out of fashion, is in rather difficult!

many ways superior to modern sedatives for this type of work and is also unlicensed in the UK. Onset of sedation is quiet and unmarred by bellowing or saliva-

Amputation of a digit or claw (see p. 430)

tion and the depth of sedation can be profound if so

The main indications for this procedure are severe desired. Its analgesic effect is good and its duration of trauma to the hoof and associated internal structures action, after oral dosing and a 10–15 minute induction and intractable infection of the foot. It is a procedure period, is 45 minutes or longer; this allows plenty of



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time for surgery by even the most inexperienced. The

Technique: No special instruments are necessary,

recovery phase is calm and uneventful and it is cheap!

although a proper curette is a great help.

The oral dose is between 60 and 90 g, depending on

With the cow in lateral recumbency, and the affected

the size of the animal, and it can be given through a

claw uppermost, prepare the foot for surgery. The claws
stomach tube, dissolved in about 1 litre of warm water,
can be difficult to clean and a combination of paring
or as a drench. If using the latter method allow the
and scrubbing with a stiff brush is often needed. To
animal plenty of time to swallow and to avoid inhala-
clean the interdigital area use three or four lengths of
tion of the liquid. Make sure that the solution does not
cotton bandage soaked in surgical scrub and see-sawed
contact the eyes as it is extremely irritant.

briskly backwards and forwards in the cleft.

Chloral hydrate can also be given intravenously, vir-
Make the incision about 5 mm proximal and parallel
tually to effect, although again its irritant nature means
to the coronary band–skin junction. Extend the incision
that care must be taken to avoid perivascular injection.
through the thick skin all the way around the outer

Whatever sedative is used, local analgesia of the foot
hemircumference of the pastern and into the inter-
is essential. The method of choice is, without doubt,
digital cleft. Here, cut the skin 2–3 mm axially to the

intravenous regional anaesthesia (IVRA).

horn–skin junction, carrying the incision through the

*The secret of successful IVRA is the application of
cleft to rejoin the skin wound. Always take care to cut*

an effective tourniquet. Use thick (2 cm diameter)

towards (or into) the affected claw. Some force is

rubber tubing of the type used in milking machine

needed to penetrate the thick skin of this region and

pipelines, applying it just proximal to the carpus or

one inadvertent slip of the scalpel blade into the sound

the hock. To improve compression in the latter case,

claw can ruin the animal. Once through the skin, angle

place 15 cm (6 inch) bandage rolls into the lateral

the incision proximally to aim for the proximal inter-

and medial depressions which lie just anterior to the

phalangeal joint. This lies in the dorsal (anterior) half

Achilles tendon. Wind the tubing around the leg at least

of the pastern thickness and some 20 mm proximal to

4 times applying maximum effort to ensure a high

the coronary band (Fig. 66.7). Careful dissection is not

degree of tension and compression; if necessary, use two

necessary and the fascia, ligaments, tendons and sheaths people. Whilst doing this do not lift the leg above the can be cut rapidly with fairly bold strokes.

horizontal or the limb veins will collapse and be diffi-

Finding the proximal interphalangeal (pastern) joint

cult to inject. Clip a strip over one of the superficial

can be difficult at times, especially if chronic infection

veins and clean it thoroughly. Using a wide bore (16 g)

has resulted in extensive fibrous reaction or the depo-

needle inject about 30 ml of 2 per cent lignocaine or

sition of periarticular new bone. The easiest way is to

20 ml of 3 per cent, rapidly into the vein; withdraw the

put an axial (inwards) force on the claw and then to

needle and syringe and apply pressure to the injection

make a series of cuts parallel to where the joint is

site immediately. (If the injection is done slowly the

believed to be. When one of these cuts is made in the

animal often kicks after about 50 per cent of the solu-

tion is injected and frequently the needle pulls out of

the vein.)

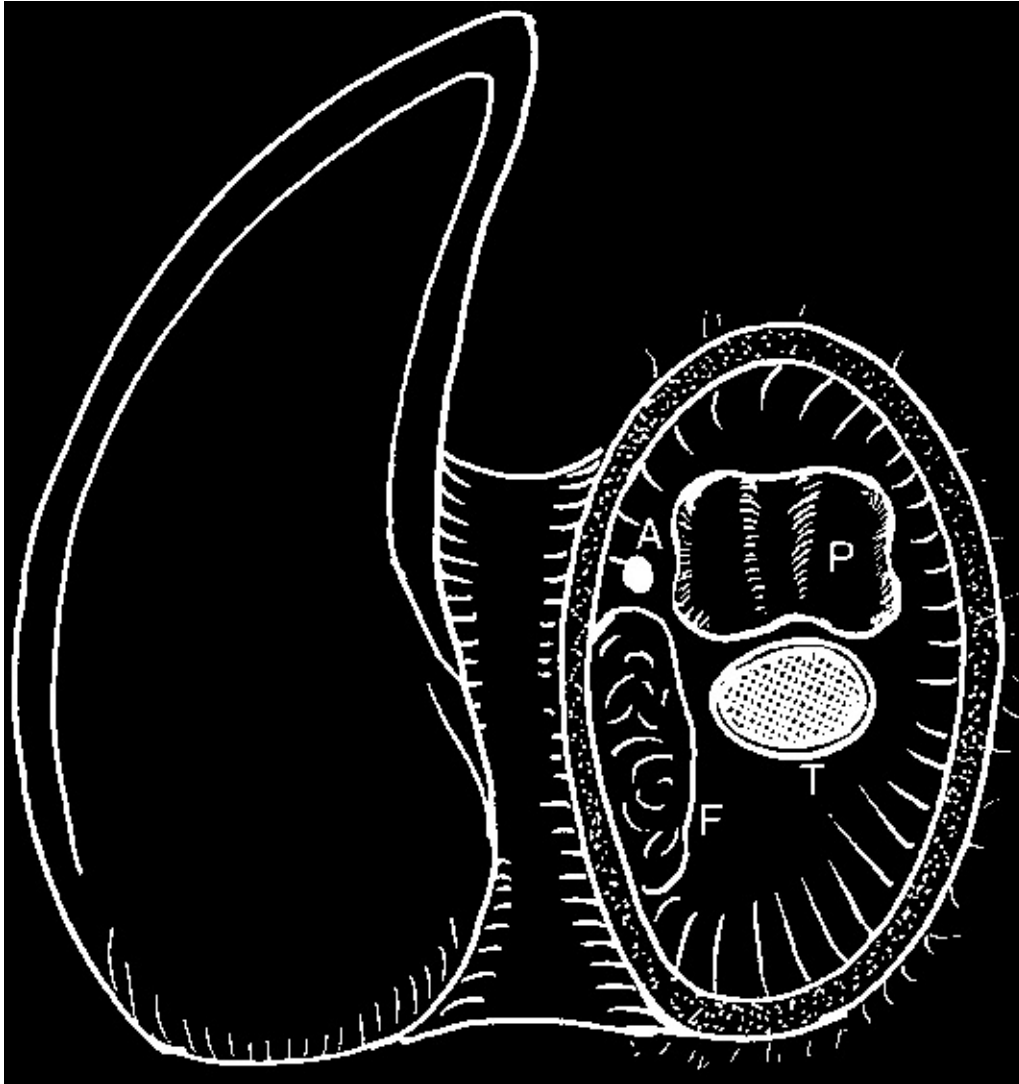
Once injection is completed, full analgesia will

usually develop in about 10 minutes or so. However, before starting the incision, this must be checked.

Within a volume of tissue subjected to IVRA the initial effects are random, with blocks of anaesthetized tissue lying adjacent or deep to other tissue blocks which are fully sensitive. It is therefore essential to check, not just by skin pricking, but by driving a needle down to the bone all the way around the claw. (It needs little imagination to picture the consequences of completing half the operation only to find the deep layers of tissue are entirely sensitive!) The interdigital cleft and especially its caudal aspects seem to be the last tissue to be affected and these areas should be carefully checked. If anaesthesia has only been partially achieved after 10–15 minutes, repeat the intravenous injection using the same amount of local anaesthetic solution. If it is still

Fig. 66.7

The heavy dotted line shows the course of the initial not fully effective then it is likely to be the tourniquet skin incision when amputating a digit. Note the position of the which is at fault. proximal interphalangeal joint relative to this incision.



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*correct place the tautly stretched collateral ligaments
tic sign as the sheath usually remains infected; it also
and joint capsule part easily with the release of some
occasionally communicates with the sheath of the
synovial fluid and an increase in joint laxity. Carry this
sound claw at the level of the fetlock. The final step is*

incision on and around the articulation, bearing in mind to curette the articular cartilage. This must be done its deeply curved profile. Take care when cutting its thoroughly to expose the subchondral bone over the deep attachments not to penetrate too far towards the entire surface of the articulation. If it is not done care-sound digit. During this phase twisting and turning the fully, granulation tissue will not grow from the bone partially amputated claw can help to expose and tense end, or adhere to it, and a persistent sinus will develop the tissues for cutting. At this point, scalpel blades are in the healing tissue. The use of a proper curette greatly often broken and it is a good idea to have a few spares simplifies this part of the surgery, but if one is not available. Complete the removal of the claw by sever-able a scalpel can be used as a substitute.

ing any remaining attachments.

Careful bandaging of the wound is vital, not only for During surgery, haemorrhage is not a problem its protection but as the only means of post operative because of the tourniquet; however, if some pre-

haemostasis. Place a layer of non-adhesive dressing on
emptive action is not taken it can be quite considerable
the exposed tissues then overfill the wound cavity with
once the latter is removed. The only vessel of real sig-
gauze, swabs or cotton wool. Wrap the stump and the
nificance is the dorsal digital artery and its cut end is
entire pastern region with a layer of cotton wool and
usually fairly easily found in the dorso-medial quadrant
use a cotton elastic bandage to keep it in place. Finally,
of the wound axial to the stump of the first phalanx (Fig.
apply a 10 cm (4 inch) elastic adhesive bandage.

66.8). Ligatures should not be used as the wound is to
Starting from the tip of the remaining claw, progress
heal by second intention. Instead, grasp the severed end
upwards, increasing the tension over the stump and
of the artery in forceps and twist it some 8 to 10 times
continuing up to and above the fetlock. The latter is
to seal its lumen. Debride the wound if necessary,
necessary to anchor the bandage securely; if it were to
cutting off any obviously infected or necrotic tissue. Do
come off the consequences would be disastrous! Take

not remove any of the large interdigital fat pad which care not to crush the accessory digits; the bandage can prolapses into the wound unless this too is infected.

be laid over them, but its tension should be slackened

Check the flexor tendon sheath: if the fluid that can be and they should be packed around with liberal wedges

milked out of it is turbid or flocculant it should be

of cotton wool prior to wrapping. This will prevent the repeatedly irrigated with saline and antibiotic solution.

skin necrosis that can occur deep to them if they are

Although not disastrous, this finding is a poor prognosis- subjected to too great an inward pressure.

Once the bandage is secure, the tourniquet can be

removed. By this stage it will probably have been on for

at least a half to three-quarters of an hour. This does

not seem to be harmful in any way and the author has

often seen tourniquets in situ for an hour or more

without deleterious effect. Remove the hobbles and

assist the patient onto her sternum. Do not force her to

her feet, rather let her get up in her own time. As soon

as she puts weight on the foot blood is frequently forced

through the weave of the bandage – if this is minor it can be ignored, but if it persists for 10–15 minutes or is excessive in amount, apply a second elastic adhesive bandage over the first and, if necessary a third. Do not, under any circumstances, remove the existing bandage.

Leave the bandage in place for four to five days. If it is taken off before that time the fragile clot is easily disturbed and haemorrhage can be severe. Thereafter, change it every five to seven days depending on the amount of exudate produced by the healing wound.

The wound cavity will begin to contract about seven to ten days after surgery rather like a flower closing and the wound area diminishes rapidly. Concurrently, gran-

Fig. 66.8

This shows the anatomic relationships of the structures remaining after amputation. A is the arterial stump; F is the ulation tissue grows from the cut surfaces and at five to

fat pad; T is the transected flexor tendon bundle and P is the six weeks is usually flush with the skin edge. Skin con-distal end of the first phalanx.

traction continues and when the wound is about 30 mm

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across the bandage can be left off. Exuberant granula-

*regarded as clean and never sterile. Sutured wounds in
tion tissue is not a problem at this site.*

*this environment always succumb to infection and to
Occasionally a cleft persists in the granulation tissue
post operative haemorrhage and swelling and always
bed and wound closure stops. This usually shows three
dehisce.*

*to four weeks after surgery and the likely cause is
incomplete stripping of the articular cartilage. If there*

Surgical correction of an umbilical hernia

*is no progress over one week, reanaesthetize the site,
cut out the granulation tissue and recurette the bone
The reasons why umbilical hernias develop in some
end.*

*animals are not fully understood. It is presumed that in
If surgery has been delayed, infection may have
some there is a failure of the normal closure mechanism
established itself proximal to the amputation site. In
(see p. 182), although there is no doubt that many pre-
these circumstances, and especially where tendon
viously normal calves are found to have hernias fol-*

sheath is involved, the lameness and swelling will persist following a bout of 'navel-ill' or umbilical infection. This despite some wound closure. No hard and fast rules can also accounts for the incidence of navel abscesses which be given as to when to give up hope of healing but, if can mimic hernias and for the fact that hernias and there is no improvement in the condition of the distal abscesses can co-exist.

limb or the lameness within two weeks of surgery, then Hernias are broadly classified into those where the resolution is very unlikely and the animal should be contents are easily returned to the abdomen, i.e. slaughtered on humane grounds.

'reducible', and those where the contents are not, the 'non-reducible'. The former, which are in the majority, Other techniques: The disarticulation technique de- are easily diagnosable and cannot be confused with an scribed here is somewhat more complicated than those abscess. The latter, however, present as a firm swelling which use an embryotomy wire, either to cut through which could be either an abscess or a combination of

the phalanges or to carry out the entire amputation. Its both abscess and hernia. In these suspect cases, use use is justified because transection of either the second needle puncture and aspiration to determine which is or first phalanx opens up their medullary cavity to an what. Puncture the swelling from both its caudal and infected environment, resulting in a fairly high incranial aspects, using a separate needle for each puncture of osteomyelitis following such procedures.

ture to prevent possible transmission of infection. Use In addition the nutrient foramen of the bone may a large gauge needle and much suction as the pus in also be destroyed leading to necrosis of the remaining these abscesses is often quite thick.

fragment. A second distinct advantage of the disarticulation method is that it produces a cup-shaped wound fingers' width or less, do not usually need repair. The cavity which maximizes the potential for healing by skin animal grows around the hernia and it is insignificant. contraction. In contrast, the use of an embryotomy wire

Some surgeons recommend closure because of the risk for the entire amputation produces a flat wound surface of strangulation of herniated viscera; this risk is very face. This does not favour skin contraction and wound small and the author has seen only one case in over 20 closure is often considerably delayed.

years. In addition, the hernial content is almost always

However, there is no doubt that this technique takes omentum rather than the vulnerable small intestine.

much less time than disarticulation and it can be per-

However, hernias which are 3 fingers' width or more are formed without casting the cow; for these reasons alone often quite pendulous and can become traumatized.

many busy practitioners prefer it. With the foot anaes-

They can also become significant cosmetic blemishes in

thetized, locate the wire loop in the interdigital space,

later life. Occasionally, the abomasum can become

checking that it begins to bite above the coronary band.

lodged in large hernial sacs although this does not seem

The aim is to amputate through the distal third of P1

to create any clinical problems for the animal.

(i.e. distal to the nutrient foramen) so initially angle the direction of cut proximally. When the wire is at the

Technique: No special instruments are needed.

correct level, change the plane of incision to lie at right

The calf should be prepared by being starved of all

angles to the long axis of the phalanx and complete the

food for 24 hours. As well as being a routine prepara-

amputation. It is worth checking carefully to ensure

tion for anaesthesia this creates a slack abdomen during

that it is indeed P1 that has been cut and not P2, oth-

surgery which helps with ring closure. The surgery

erwise ischaemic necrosis of the remaining fragment of

should be performed under general anaesthesia, or

the second phalanx is very likely. Otherwise postoper-

under heavy sedation, with the surgical site anaes-

ative management is the same as for disarticulation.

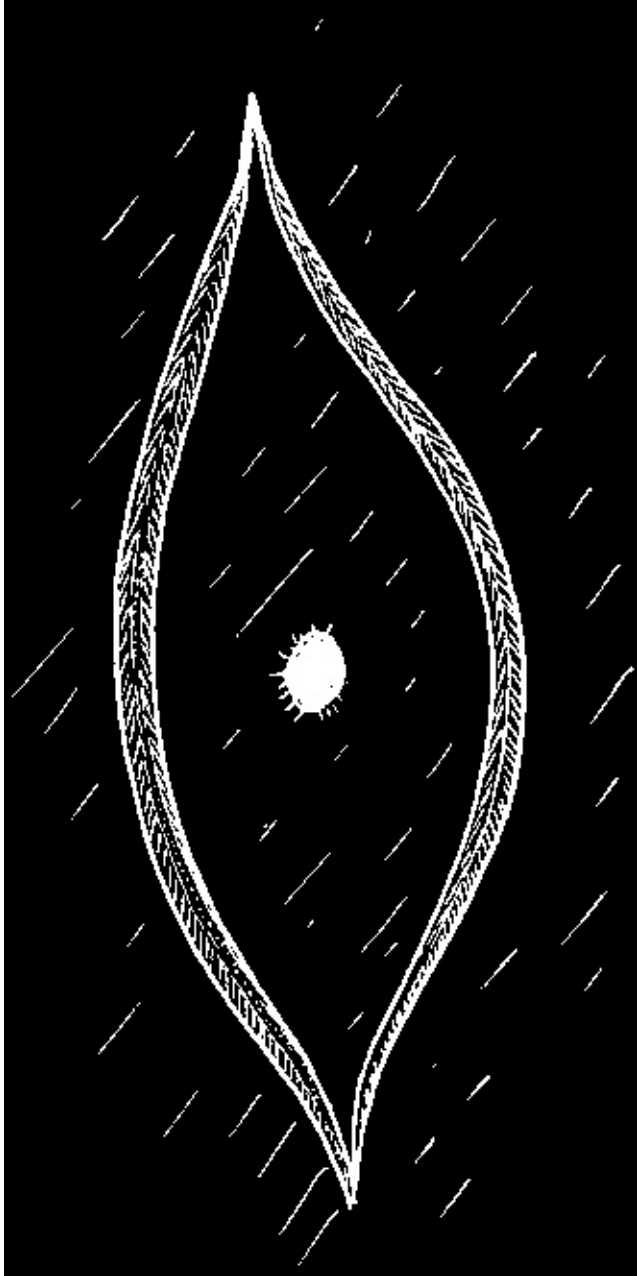
thetized by a field block. With the patient in dorsal

Avoid techniques which call for sutured flaps of skin

recumbency, excise a fusiform (Fig. 66.9) shaped area of

over the stump. This operation at best can only be

skin centred on the cicatrix at the hernial apex. Try and



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sac can be inverted into the abdomen. There is no need to suture the sac, only to close the now enlarged ring in the usual way.

For closure, use strong, non absorbable suture mate-

rial which forms a secure knot. Monofilaments are not ideal and the author prefers a sheathed multifilament type of suture. There is no need to use any complicated suture patterns to overlap the ring margins, e.g. the so-called 'vest-over-pants' technique, as a simple apposition with simple interrupted sutures is entirely sufficient. The former creates a bulky mass of tissue which in turn creates a poor cosmetic effect. Also there is no need to 'freshen' the ring edges.

Insert the first suture some 1 cm beyond the caudal or cranial ring edge, then progress along the ring laying sutures every centimetre or so to finish one centimetre beyond the ring edge at the opposite pole (Fig. 66.10). Use the finger of the 'spare' hand to protect the abdominal viscera from the point of the needle. Lay all the sutures before tying and clamp each pair of free ends with artery forceps to prevent them from pulling out

Fig. 66.9

A fusiform incision around the cicatrix.
or becoming tangled. Tie the sutures one by one, starting with the outer ones and progressing towards the

middle. Pull each suture tightly enough to appose the ring margins and then a little more to create a mild compression effect. If tied in the conventional way the first throw often slips as tension comes on it; to prevent this on the cautious side as more can be removed later. Separate the skin of this area from the underlying fascia being applied or, alternatively, make the first throw a triple rather than a double. This will usually resist the applied tension sufficiently for the locking throws to be section with a scissors tip, and beginning at the cicatrix, completed.

dissect back the multiple layers of fascia in a radial fashion to expose the discrete, thick, peritoneal hernial sac which lies deep to them. This is facilitated by grasp-

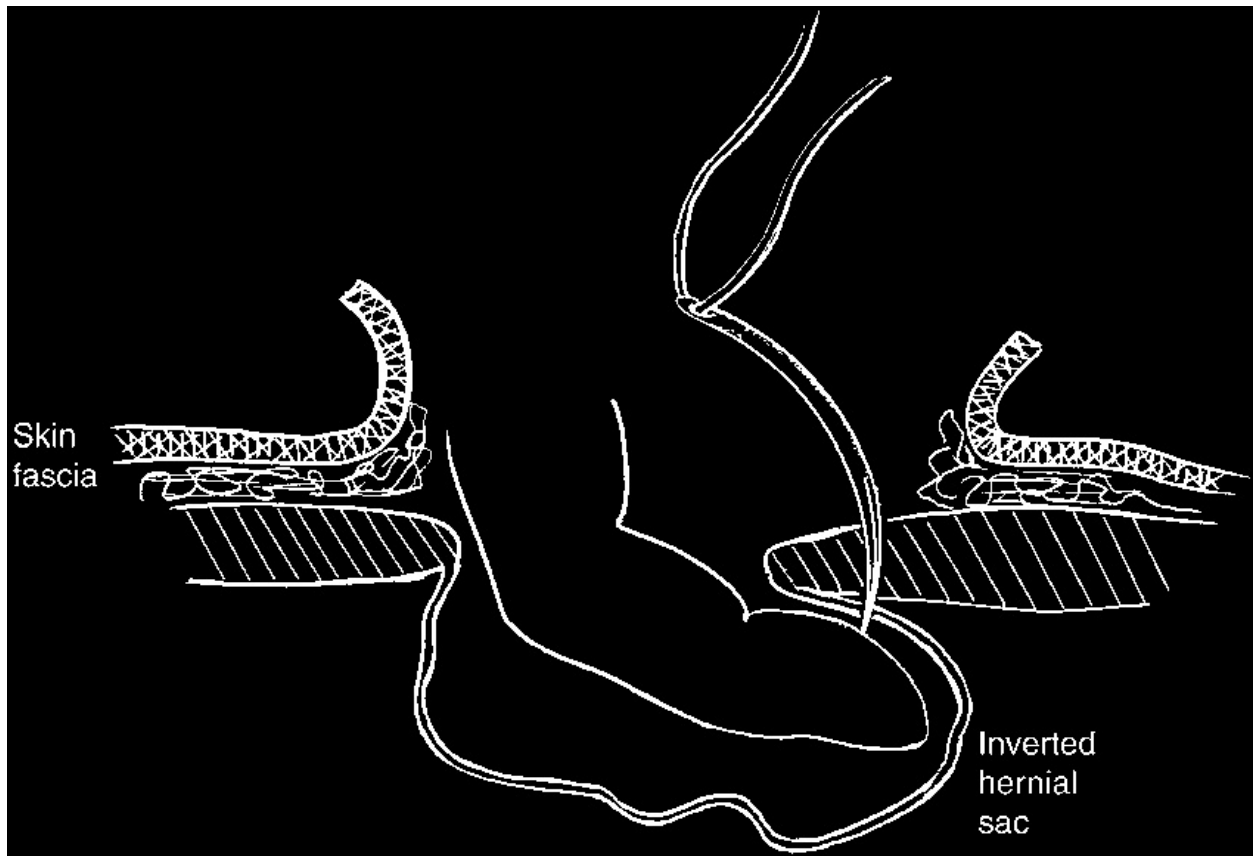
having first trimmed off any excess tissue that may ing the cicatrix with an Allis or similar forceps and prevent a flat closure. If the skin edges are still apart, applying upwards traction to tense the tissues. As the put in a subcuticular layer to bring them together. More fascial layers peel back, the sac becomes more obvious likely there will be some surplus skin and this too can and eventually becomes completely exposed, along be trimmed away, being careful to maintain the fusiform with the now easily discernible edge of the hernial ring. shape of the final deficit. Close the skin with either a Clear the fascia back from the edges of the ring for at simple interrupted or interrupted horizontal mattress least 1.5 cm all the way round so that there is a clear suture pattern. Finally, to apply pressure to this ventral shelf of aponeurosis into which the closure sutures can and dependent wound, and to protect it, sew on a Stent be laid.

bandage (Fig. 66.1).

Invert the sac and contents into the abdomen; if they go easily, as most do, there is no need to open the sac.

The repair of large hernias: To repair hernias which
If the contents are irreducible, the ring will need to be
have large rings over four to five fingers in width, most
enlarged. Carefully, so as not damage the hernial con-
surgery texts advocate the use of stainless steel or syn-
tents, make a small opening in the sac at the caudal or
thetic mesh to fill the defect. The author has had little
cranial ring margin. Using a grooved blade director if
success with these as, no matter how tightly they are
available, or any blunt instrument which can be inserted
sutured in place, they invariably sag under the weight
between the contents and the ring as a shield, enlarge
of the viscera so that the final result is little better than
the ring by cutting caudally or cranially as appropriate.
the original hernia!

In most cases the ring only needs to be enlarged to a
An alternative and effective method is to create a
small extent before the contents and subsequently the
slack abdomen by depriving the animal of roughage for



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Fig. 66.10

*Illustration of how the index finger
is used to guard the advancing needle tip
when laying sutures to close a hernial ring.*

*some 48 hours prior to surgery and then to carry out
if several animals are to be done, anaesthetize and
the repair as described above. Be sure to use strong
operate on them a batch at a time. Be sure to use an
suture material and to take good solid bites of the*

anaesthetic solution containing adrenaline which has a fibrous aponeurotic tissue surrounding the ring. One long duration of effect, and be sure that there is enough modification which helps a great deal is to have an assistant apply upwards traction equally and simultaneously crush a second time!

on all the laid suture ends to bring the ring edges

Animals with very large horns and therefore a broad diameter at the skin–horn junction often require an additional ring block for full analgesia. If in doubt carry

largest hernias can be repaired satisfactorily. Using this technique and preoperative fasting, even the additional ring block for full analgesia. If in doubt carry

out this procedure at the outset to avoid undue pain and the consequent non cooperation of the patient. The skin at the base of the horn is tightly applied to the skull and

Dehorning of an adult

infiltration can be difficult. Slide a long, relatively

In the past this procedure was commonplace; nowadays narrow gauge needle (e.g. 19G) under the skin and

it is done less and less since most cattle are debudded
inject the solution as the needle is withdrawn. Concentrate
on the caudal aspect of the horn base as the 1st
the procedure performed at all.

and 2nd cervical nerves can innervate this area of
Dehorning should be done in the winter months
skin-horn junction and they will not have been affected
when the risk of fly-strike is at its lowest.

by the cornual block.

*Anaesthesia: This is a technique which relies heavily on Surgical technique:
There are many methods for removal-good restraint of the animal and the use of a
'crush' and*

*ing the horns; the commonest are shears, embryotomy
the help of a strong and competent assistant are essential
wire and the butcher's saw. Shears can be used for young
if a rodeo is to be avoided.*

*stock where the horn and cornual process is soft, but
For most routine cases the standard cornual nerve
they are well nigh impossible to use if the horn is mature
block is sufficient. A common mistake is to use too little
or the horn base is very wide, as in the Welsh Black*

anaesthetic solution: at least 10 ml should be infiltrated
breed. They allow very rapid removal of horns, though
across the path of the cornual nerve. Redirect the
if used carelessly they may not cut the horn low enough
needle several times during the injection to saturate a
and therefore not amputate a complete circle of the ker-
large volume of tissue and increase the chances of a suc-
atogenic tissue which lies at the skin–horn junction. In
cessful block. Bleeding from the injection site is normal
addition to the long term complication of horn regrowth
and a good indication that the needle was in the correct
which may result, there is the more immediate problem
place. Another error which can lead to problems is to
that a cut made too high amputates the cornual vessels
attempt surgery too soon after the injection was made.
after they have entered the bone of the cornual process,
Wait for at least 10 minutes before beginning to cut and,
making haemostasis extremely difficult.

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*Embryotomy wire is silent in operation, resulting in
of the remaining bleeding points stop or slow to a level*

better patient cooperation; it allows for the direction where they can be ignored.

of cut to be changed and provides good intrinsic

In those cases where the horn and cornual process haemostasis. However, the push/pull action needed is have been sectioned too high the vessels will continue very tiring, the wire often jams in the cut, especially if to bleed from the vascular canals within the bone. They it is used too fiercely, and it is very prone to break.

cannot be picked up in artery forceps and are a nui-

The author's preference is for a heavy butcher's sance. The time-honoured way of dealing with these is saw which cuts with its own weight and whose use to insert a sharpened matchstick point into the hole to involves a more congenial action than the embryotomy plug it. Although seemingly crude in these days of wire.

modern technology, it works, and the presence of the

With the animal in the crush, the assistant should, wood fragment does not appear materially to affect using a 'nose lead' ('nose grip', 'bulldog'), bend the

healing in any way.

animal's neck sharply to one side to move one horn away from the crush door. This often causes the head

Post operative course: Treatment of the resultant to tilt to one side and it should be straightened as much wounds with antibacterial powders and aerosols is as possible before cutting begins. At this stage check widely practised. However, its effects are likely to be that the anaesthetic has worked and that the whole of cosmetic only and the author uses no wound dressing the horn base is desensitized. If it has not been done without apparently affecting the speed or course of already, clip the hair from around the base of the horn healing.

then position the saw blade some 2.5 cm from the

Post operative care must involve examining the medial horn base. Angle the blade towards the point animals every few hours for the first day or so in case where the ear meets the head and begin sawing. Occa- there is secondary haemorrhage; thereafter daily mon- sional animals object violently even when the horn is

itoring will be sufficient. Advise the farmer to feed hay apparently completely desensitized and the blocks have off the ground and not from overhead racks as this been repeated or supplemented with a ring block. In diminishes the amount of seed and dust contamination these cases it is likely that they are reacting to the noise of the exposed frontal sinus. In the normal course of of the saw in their heads and a change to an embryo-events the hole will scab over within one or two weeks, otomy wire will solve the problem.

with complete healing by skin contraction and epithe-
Saw swiftly through skin and bone to emerge just
lialization taking several months.

above the ear. Take care not to lift the skin from the ear
and, if this seems likely, cut the remaining skin tag with

Complications: Secondary haemorrhage is not
scissors.

common provided the largest vessels have been dealt
with properly. However, fighting or rubbing of the
Haemostasis: When the horn is finally detached from
wounds can occur as the analgesia wears off and bleed-

the head, haemorrhage can be impressive. However, it
ing can be severe. Identification of the source of the
is not life-threatening and can easily be temporarily
haemorrhage is almost always impossible as the side of
stopped by finger pressure. Many techniques of
the head and the surgical site are covered with a mass
haemostasis have been described and advocated but the
of blood clot which effectively hides the bleeding point.
simplest and most effective is torsion or twisting of the
The area is now completely sensitive again and the
bleeding vessels.

animal usually vigorously resents its handling. The
With a robust pair of artery forceps, grasp the vessel
quickest and least painful remedy is to apply pressure.
ends firmly, apply gentle traction and spin them about
Place a large pad of cotton wool directly over the horn
six or so revolutions or until they break off. This will, in
stump and bind it down tightly with several rolls of
most cases, completely stop the bleeding or at worst
10 cm (3 inch) adhesive elastic tape wound around the
reduce it to a small point of seepage. The largest vessels

head. Leave the ears protruding, as they help to prevent are always to be found at the ventral, rostral edge of the the bandage slipping, and take care not to constrict the skin wound and, if the cut is made low enough, will be larynx or trachea. Although apparently crude, it is a exposed for several centimetres of their length. If they very effective method and avoids the need to re- are not, grasp the bleeding, severed end of the artery anaesthetize and explore the site in an often fractious and, using gentle leverage over a finger, expose a suffi- animal. Some individuals may be extremely anaemic cient length to be twisted. Do not attempt occlusion by after such haemorrhage and a small number may snapping off the vessel end, as is occasionally recom- require blood transfusion. As a rough guide, if the mended; a high proportion break way below skin level animal is able to stand and move around without being and continue to bleed from inaccessible depths. If these too ataxic it is not likely to require such measures. large vessels are occluded in the manner described most However, if in doubt, transfuse.

Long term complications are sinus empyema and fly-dental rupture of the conjunctival sac and contamination of the wound. Farmers must be instructed to call for veterinary

attention if a recently dehorned animal becomes dull or

To penetrate into the orbital cavity, retract the upper eyelid skin and use a finger tip to locate the bony edge

if necessary by lifting the scab. If infected, or affected of the mid upper orbit. With a scalpel cut just below

by fly strike, give systemic antibiotics and treat locally the bone edge and parallel to it. Once an access hole

by irrigation with copious amounts of water to remove

has been created, use a curved-on-flat scissors to extend

the pus or larvae. Repeat the irrigation daily until the

the aperture laterally and medially, keeping close to

sinus is clean and healing is progressing well.

the orbit rim all the way round. Leave the medial

canthus until last as the largest vessels lie here and their

bleeding can be difficult to control. Up to this point

Eye enucleation (see p. 922)

haemorrhage is not usually a problem and it can be con-

The common indications for eye enucleation in cattle

trolled by conventional means. When the medial attach-

are panophthalmitis and conjunctival or scleral squa-

ments are severed the globe becomes free and

mous cell carcinoma. The presence of infected or

relatively mobile. If bleeding from the medial vessels is

tumour material within the conjunctival sacs in these

severe then hard finger pressure for a minute or so

cases precludes a subconjunctival approach through the

should reduce it to manageable proportions. These

palpebral fissure and so most large animal enucleations

vessels are often difficult to identify and even more dif-

are done by a transpalpebral technique. With this

ficult to grasp with artery forceps so if first attempts fail, method, the eyelids are
sewn tightly together to trap

apply pressure to the area for a little longer and then

infection or neoplastic material with the conjunctival

proceed with freeing the globe from its deep attach-

sacs and the globe is removed intact to maintain a

ments. This is done by putting outward traction on the sterile wound.

globe, inserting a heavy pair of curved-on-flat scissors into the gap between globe and orbit and cutting the

Anaesthesia: In tropical countries where ocular squamous cell carcinoma is rife, enucleation is a common it can be pulled further from the cavity and access

procedure and is usually done under retrobulbar block.

improves. Ideally the muscles should be detached close

This is a relatively simple technique which is described

to their insertion on the capsule so that as much tissue

in many textbooks but, if only the occasional enucle-

as possible is left within the orbit to minimize dead

ation is done, and facilities are available, then the lack

space. Practically, poor access, retrobulbar fat and the

of movement and relaxation of general anaesthesia

inevitable haemorrhage mean that cutting is usually

makes for much easier surgery!

done blind and the muscles, fat and optic nerve are sec-

tioned randomly. As soon as the globe is free lift it away

Technique: Prepare the eye by fine clipping of the eye and attend to any haemorrhage. Unlike the dog and cat,

lashes, lids and periorbital region, followed by removal

bleeding from the optic artery is not dramatic, there is

of debris and discharge from the conjunctival sac. Sew

no need for ligation, and at worst blood wells up slowly

the shorn eyelids together tightly with an O or I non-within the cavity and can be contained by firm finger

absorbable suture, laying the sutures close to the eyelid

pressure maintained for four to five minutes. It is not

margins. The lids and surrounding area can now be

necessary to stop haemorrhage completely so as soon

prepared for surgery.

as it has slowed to manageable proportions suture the

Incise the skin of the upper lid in a line parallel to

remaining skin of the eyelids together with horizontal

the lid margins and some 4 mm outside the suture line.

mattress sutures of non-absorbable material. With the

Deepen the incision carefully to expose the subcuta-

suturing completed, squeeze out excess blood from the

neous fascia. Using Metzenbaum or other similar scis-

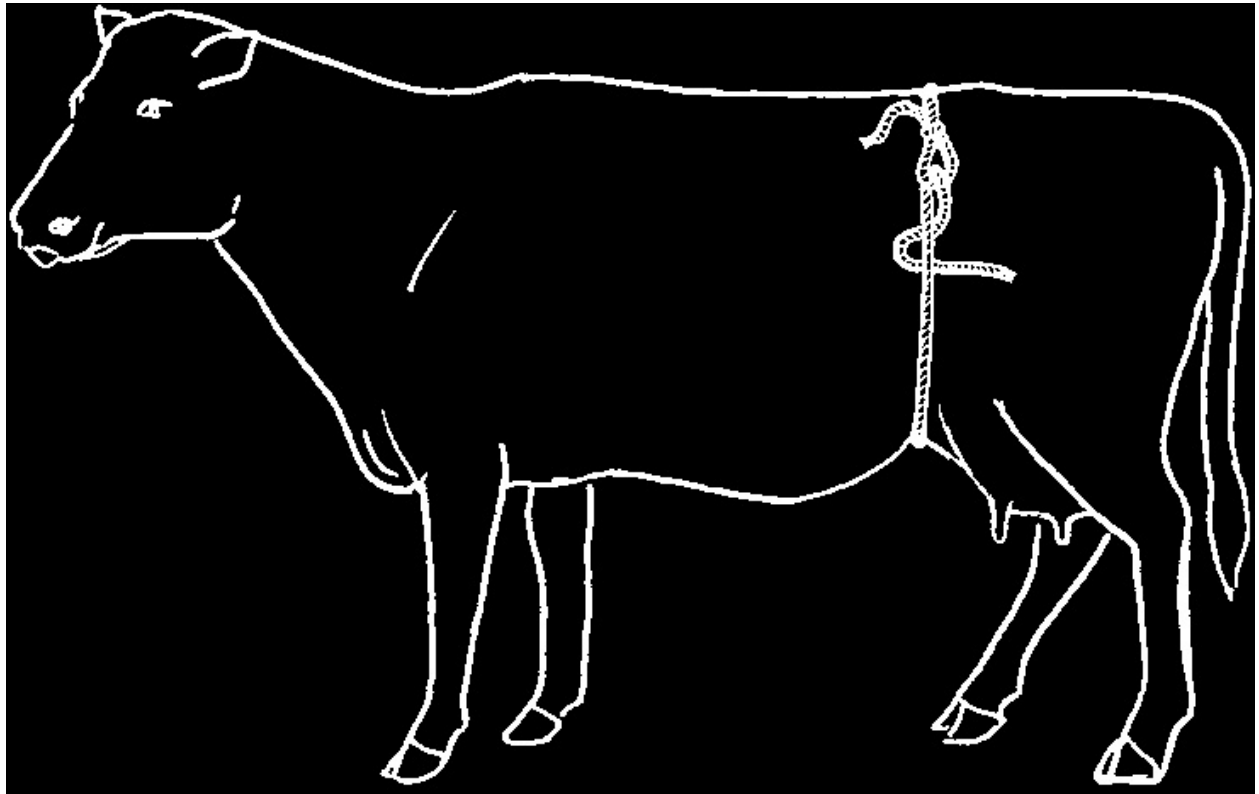
socket through the incision line and sew a stent bandage

sors blunt dissect under the skin of the eyelid, through (Fig. 66.1) firmly in place over the wound so as to exert the circular muscle of the eye (orbicularis oculi), until a firm inward pressure. This controls residual haemorrhage and protects the wound.

reached the bony rim of the orbit. Extend the skin inci-

If the conjunctival sacs and globe were removed
the length of the upper lid and the dissection plane intact, healing is likely to be uncomplicated. Remove to the remainder of the dorsal orbital rim. Repeat the 'Stent' at two to three days and the sutures at about process on the lower eyelid so that the incisions above 10 days. Some authorities recommend instilling antibiotics into the orbital cavity before wound closure, but lateral canthi in a fusiform shape. Make sure that the this is probably not indicated unless there is some con- dissection planes within the lids remain superficial until tamination and routine prophylactic and post operative the orbital rim is reached; this will help prevent acci-

antibiotics normally suffice.



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*Where there is known contamination of the wound,
In practice the former two requirements are met by
first intention healing may still occur with vigorous
using plastic indwelling cannulae, of which there are
antibiotic therapy, but an alternative method is to pack
many designs. Use them liberally; they are relatively
the cavity with sterile cotton bandage coated with anti-
cheap and work well. However, bear in mind that as
bacterial cream and layered concertina fashion. The*

well as being a boon, they can also act as very efficient free end is laid in the medial commissure of the wound introducers of infection if used incorrectly. If they drop which is sutured in the usual way apart from its medial out, they must never be reintroduced, but must be 1–2 cm. The bandage is removed through this gap after replaced by a new one from the packet. Supply them in two to three days and the socket eventually fills with granulation tissue. There is a persistent discharge from and the cleansing of the teat before insertion.

the drainage hole and routine management involves To protect against infection be scrupulously clean maintaining the patency of the aperture and clearing up when introducing anything into a teat and use intra-the discharge. Irrigation of the cavity does not seem to mammary antibiotic preparations routinely after any materially affect the healing process. When the socket form of teat surgery or while a cannula is in place. Systemic antibiotics too, although not often used, are indi-

This process can take several weeks.

cated in cases of trauma and surgery to aid successful healing.

Teat surgery (see Chapter 27)

Restraint: It is impossible to be clean and deft when the Milk production is the raison d'être of the modern dairy patient's hind legs are flying around the surgeon's head!

cow. Inefficiency in any of the factors involved leads to

Use restraint and lots of it! For simple examinations,

her becoming unprofitable. The care of teats and the

tail restraint, i.e. forcing the root of the tail vertically, is surgery that is frequently involved therefore deserve

often adequate; it discourages kicking, although it will more attention than they often receive.

not stop a reaction if there is a sufficiently painful stim-

There are three enemies of successful teat surgery:

ulus. A second level of restraint is a 'nose lead' or

the 3 Ms! Milk, milking machines and mastitis must

'bulldog' clamp; again its effect is of discouragement

always be taken into account by the surgeon.

rather than prevention of reaction. A more effective

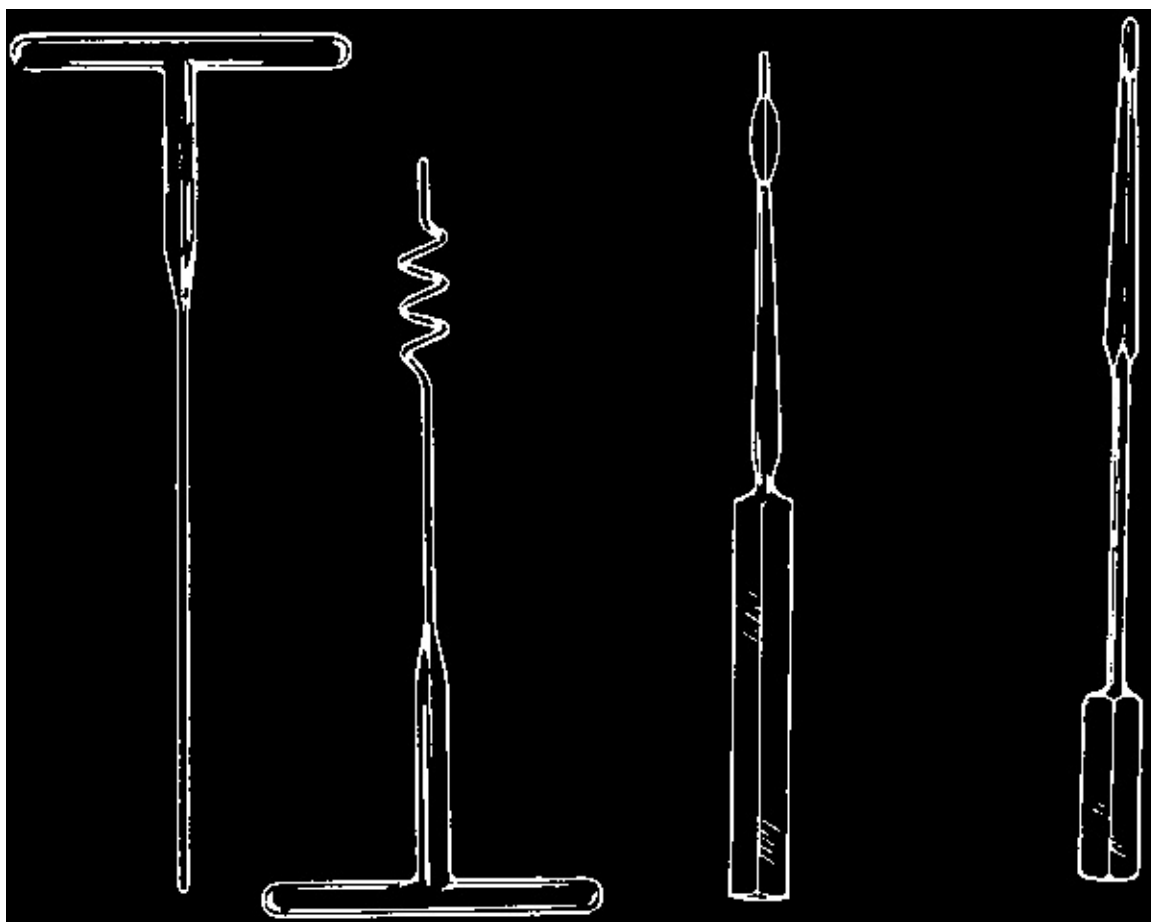
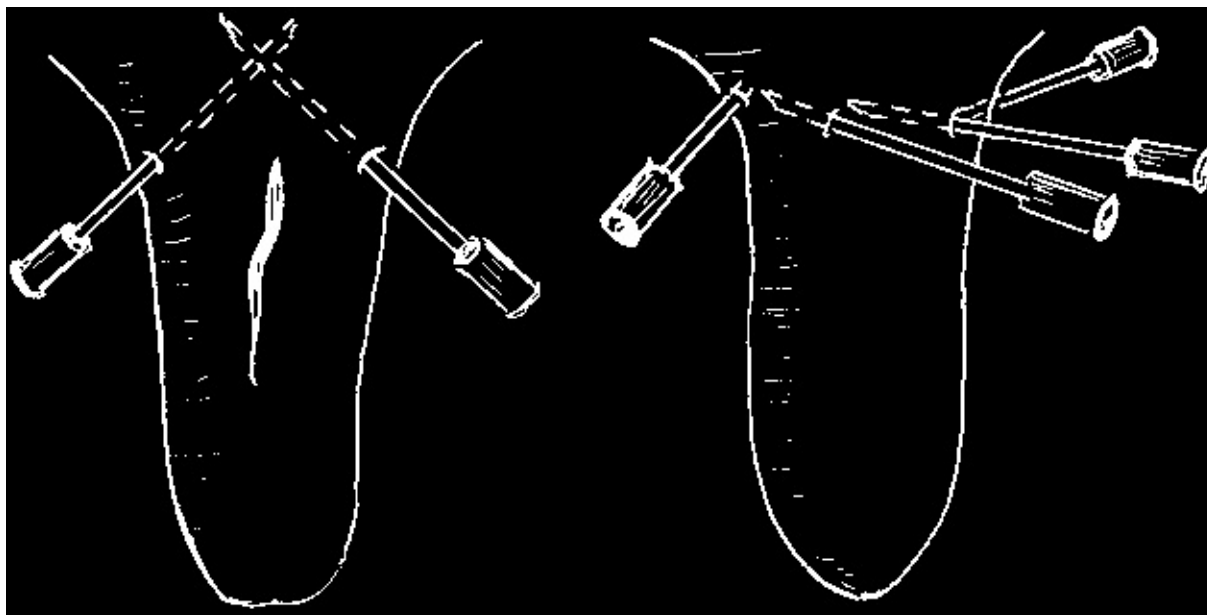
A simple way to avoid these problems in the small

method is the 'udder cinch'. This is a length of rope with proportion of surgery which is elective is to delay until a loop in one end which is passed around the cow's the cow's next dry period. Often this option is not open abdomen just in front of her udder. The free end is as the majority of surgical procedures are emergencies. threaded through the loop and pulled against it to In these cases the surgeon must take steps to ensure tighten the cinch around the belly. Pull it tight enough that milk pressure does not build up within the teat, that to cause the cow to sway slightly then secure the free the teat and surgical site do not have to bear the trauma end by tucking it partially under the taut portion to of being milked, either by machine or by hand, and that form a quick release (Fig. 66.11); do not pull too tightly adequate antibiotic cover is given.

or the animal may go down! Used in combination these

Fig. 66.11

An udder cinch.



three techniques of restraint will dissuade the cow from kicking even in the face of a considerable pain stimulus. Use them routinely as a cow's kick is quite capable of causing major injury!

a pause whilst the lubricant with which the cannula is filled dissolves, the milk and some of the antibiotic will closure of a lacerated wound, the success rate will be run out.

much better if the cow is first cast, restrained and positioned to allow good access to the wound, better lighting and a more comfortable position for the surgeon.

Streak canal stenosis: This long-term sequel to teat trauma is caused by fibrosis of the sphincter region. It results in an increase in time needed to milk the quarter

Local anaesthesia: The usual method is a ring block at and is a nuisance in a parlour system which does not

the base of the teat or an inverted V block (Fig. 66.12).

tolerate delays.

In the former technique, the cow need only feel one

*In the first instance, dilatation of the canal can be
needle prick, as the subsequent injections are made
tried, using a plastic teat cannula; this is inserted and
through the area of skin already desensitized. For
left in place for 7 to 10 days. If this is ineffective use a
streak canal and teat sinus surgery, more localized anal-
teat dilater. This device has two arms which, when the
gesia can be obtained by putting a tourniquet around
instrument is in a closed position, form a probe-like
the base of the teat and infusing local anaesthetic solu-
shape. After insertion into the canal a knurled knob is
tion up through the streak canal.*

*turned at the end of the instrument and the two arms
separate, stretching the lumen of the canal. These two*

*Crush injuries: In days gone by when cows were
methods are relatively atraumatic and should be used
shipped-housed, crush injuries were commonly caused
if possible; however, their effects are usually temporary.
by one cow swinging round to tread on her lying stall-*

A more permanent result is obtained by incising the partner's teat. Nowadays most cases are in older cows canal walls with a McLean's knife or similar. This is flat, who tread on the hind teats of their own pendulous double-edged (Fig. 66.13) and is used with a push/pull udders whilst getting up. These self-inflicted injuries action. The teat is grasped firmly as if for hand milking, usually consist of gross contusion to the teat walls tensed and the probe end of the knife inserted into the with much haemorrhage into the substance of the teat, canal. A sharp push causes the leading edges to cut swelling, pain and acute stenosis of the streak canal. as the blades pass through the canal. Once inside the If no major laceration has occurred, the treatment is blades are rotated through 90° and pulled sharply out, one of systemic antibiotics for several days and inserting a cruciate incision. Use the narrowest blade size tion of an indwelling cannula to ensure that milk can be first and if after its single use the milk flow is good, insert released from the quarter and that milking is avoided. a plastic cannula and allow the healing phase to take

Leave the cannula in place until the swelling and pain place around it. If the milk still does not run out easily, have subsided and the streak canal is functioning again; repeat the procedure with a larger diameter knife. this can take up to 14 days.

Inserting the cannula into the painful and swollen teat can be difficult. After gentle cleansing of the teat and the canal area, locate the latter with a blunt ended probe. Insert the atraumatic end of a tube of intra-mammary antibiotic and lubricate the canal with the

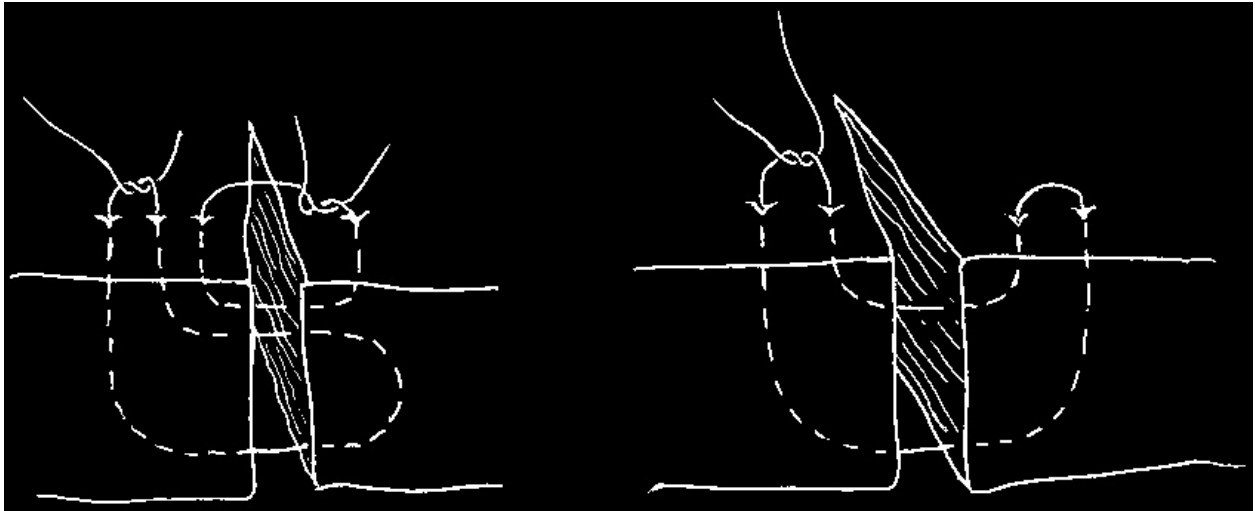
Fig. 66.13

Some common teat instruments; from left to right: a

Fig. 66.12

Ring block and field block techniques.

probe, a Hudson's spiral, a McLean's knife and a Hall's knife.



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Fig. 66.14

Two suture patterns which provide apposition of both the deep and superficial layers of the teat wall.

Although effective in the short term this is also traumatic debridement of the wound margin. To achieve this, trim to the canal and sphincter, and post operative fibrosis a thin (0.5 mm) sliver of tissue off each wound edge will usually result in a sphincter of varying competence, along the whole of its length, extending the wound with contraction of the scar tissue almost always causing fissures slightly at each end. This results in fresh bleed-
a recurrence of the problem at some later date.

ing tissue being apposed and a consequent rise in the chances of primary union.

Lacerations: For the purpose of deciding on the appro-

Use the suture patterns shown (Fig. 66.14) if possible

priate treatment, teat lacerations should be divided

as these compress both the superficial and deep layers, yet

into 'penetrating', i.e. those where the wound pene-

can be removed later. Sometimes the teat wall is excep-

trates into the lumen of the teat or canal, and 'non-

tionally thin and these more complicated patterns are dif-

penetrating', where the deep layer(s) are intact.

ficult to use; in these cases use simple interrupted sutures.

Using fine, non-absorbable material, lay sutures fairly

Non-penetrating wounds: Teats heal extremely well if

close to each other and do not penetrate the teat lumen.

given the opportunity. If it is certain that there is no

On completion, allow the cow to rise then apply a

penetration and no leakage of milk through the wound,

bandage to the teat. Degrease its surface carefully with

then allowing the wound to heal by second intention or

alcohol and allow it to dry. Place a small patch of

granulation is less risky than attempting to suture and non-adhesive wound dressing over the incision line and, possibly entrapping ischaemic tissue and infection.

using a 2.5 cm wide adhesive bandage, start at the

There is then the distinct possibility that enclosed sep-sphincter end and layer it directly on to the teat skin.

sis will erode through the deep layers of the wound

Overlap each winding by about 30 per cent and secure

and create a fistula into the lumen. Only if a non-

the upper, free end of the bandage to the skin with a

penetrating wound is very fresh and exceptionally clean

single superficially placed suture. Insert a cannula into

should suturing be considered. If there is doubt, treat

the streak canal and remove the cap; this will prevent

by physical cleansing of the wound and by inserting a

any build-up of milk pressure within the teat. Give

cannula to remove the trauma of milking. Systemic and

systemic, broad-spectrum antibiotics. The use of

intramammary antibiotics are indicated and usually

intramammary cerates within the first few hours is

healing is rapid with minimal scarring.

questionable as their base is very pervasive and may penetrate the suture line if they contact it before there

Penetrating wounds: Where the lacerations penetrate is some adhesion between the wound edges. After the into the lumen there is not the luxury of deciding first 12–24 hours they are definitely indicated as a whether to suture or not. The wound must be closed to barrier to any infection entering the teat.

prevent the development of a fistula and inevitable mastitis and, because the surgery involved is delicate

Special cases: Sometimes the teat is lacerated horizontally and must be done carefully, there is every reason to cast

tally near its end so that the cut lies close to or enters the teat and conduct the operation in a controlled

streak canal. To discover whether there has been penetration in a comfortable manner.

tration, hand milk the teat and look for milk emerging

In cases where the cut is recent and is minimally contaminated, simple cleansing and suturing may be all that is needed. However, most wounds are much older when

Where the streak canal has been spared, the decision

is needed. However, most wounds are much older when

to suture or not will be based on the criteria already laid seen and are often badly contaminated. In these circumstances a much better rate of healing is achieved if 180° around the teat circumference and so it usually the cleansing of the wound is supplemented by surgical does not gape too much. With a teat cannula in place

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to minimize milking trauma, but otherwise left to their dental amputation an inch or so remains it is worth-own devices, teat lacerations usually heal extremely while attempting some reconstruction. Trim the end of well, being barely visible in a few months. This makes the stump so that it lies roughly at right angles to the the possible repercussions of suturing and the trapping long axis of the teat, then insert a purse-string suture of infection much less acceptable.

around its circumference close to the cut end. Draw On the other hand, when the streak canal has been the purse-string tight to compress the sinus opening cut its early closure is essential. However, before sutur-

snugly around a plastic cannula. Fashion two or three ing begins, try to make some estimate of whether the stay sutures to anchor the cannula in place and remove distal tissue flap has an adequate blood supply for survival and healing; there is no point in trying to repair end will heal around the cannula into a semblance the defect if it has not! Sometimes it is obvious that the of a streak canal. There will, of course, be no sphincter attachment of the flap is too small for a good supply to and milk leakage and bacterial ingress are major be present and in these it is best to cut it off immediately in the sure knowledge that it is not viable. With a allow completion of the lactation and even a subscannula inserted in the remaining streak canal while quent one.

healing takes place, the resulting slightly truncated teat is likely to be completely functional, although more

Repair of congenital and acquired teat fistulae: Con-
liable to bacterial ingress and mastitis.

genital fistulae are usually first noticed when a heifer
In other instances it is obvious that a good blood
calves and comes into milk. The most likely explanation
supply exists and repair can proceed; in yet others it is
for their presence is that they are the streak canals of
not so obvious and in these cases it is wise to give the
otherwise vestigial, supernumary teats. Acquired fistu-
tissues the benefit of the doubt.

lae are the result of wounds to the teat and failed repair
Before beginning to suture, debride the cut surfaces
of penetrating lacerations. They are a nuisance in that
carefully, if necessary cutting a thin sliver off both sur-
they leak milk, which causes bacterial multiplication on
faces of the entire wound. Also, and most importantly,
the teat surface and hence milking machine contami-
insert a teat cannula through both parts of the streak
nation. They also allow ingress of infection into the
canal so that they will be aligned during suturing and in
quarter and attract flies. To avoid the complications
subsequent healing. In the post operative period,
associated with open teat surgery, delay if possible until

examine the wound often and remove the sutures from the next dry period. Then with a cast, restrained cow any part that looks as if it may dehisce. In this way pus and the maximum cleanliness available, excise the pressure is not allowed to build up and disrupt healing fistula through all the wall layers by means of a fusiform further.

incision. This shape causes the minimum of distortion on closure of the wound. Use the suturing techniques Transection of the teat: Milking machines need very described above, apply a bandage post operatively and little length of teat to hold on to, so if after the acci- give systemic antibiotic cover for several days.

Adult Cattle

*The Interaction of the Animal, Environment
and Management*

Chapter 67

Stress and the Pathogenesis of Disease

P.J. Hartigan

Introduction

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different occasions. Hence, when stress has adverse

The stress system

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effects on health, different patterns of disease may

The stress response

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emerge amongst animals exposed to the same stressors.

Coping with stressors

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Historically, biologists have formulated paradigms of

The biological costs of stress

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stress based on the concept of an integrated stress

The transition to pathology

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response jointly regulated by the sympathetic–adrenal

Concepts of allostasis and allostatic load

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medulla axis (Cannon's fight/flight response to threat)

Allostatic load and the veterinary practitioner

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Clinical implications

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and by the hypothalamic–pituitary–adrenal cortex axis

Concluding comments

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(the general adaptation syndrome described by Selye).

Glossary

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The two neuroendocrine axes have the capacity to alter metabolism and to redistribute metabolites to help the animal maintain homeostasis as it responds to stressors.

Introduction

While these feedback systems are capable of coping with the many uncomplicated and discrete stressors

It is generally believed – and frequently repeated in the literature – that ‘stress’ is the major challenge to the that animals encounter every day, it has been realized that the operating characteristics of typical feedback-welfare, health and productivity of animals in intensive controlled homeostatic systems are too narrow to

commercial cattle enterprises. However, when it comes account fully for the complexity of the interactive to drawing practical conclusions from the literature it responses that culminate in stress-related pathology. By becomes clear that scientists have been working with definition, homeostatic responses serve to maintain the paradigms of animal stress that have done little to help constancy of the internal environment; each strives to the industry to cope with the biological imperatives that return a regulated parameter to a set point that repre- it must address as it seeks to manage intensive units sents the central tendency of that activity. For instance, with the minimum of animal distress. There is a lack of a fall in serum calcium concentration elicits an imme- unanimity and clarity about which stimuli are so stress- diate increase in the secretion of parathyroid hormone, ful that, on occasion, they can undermine the dynamic which should promptly bring the calcium concentration equilibrium established and maintained by a battery of back to set point, its 'normal value'.

homeostatic responses. Any stimulus that challenges

Many stress disorders may not be amenable to such homeostasis can be described as a stressor. It is a con- a straightforward resolution. Stressors may create per- dition of life that homeostasis is under constant turbations within several interacting organ systems and challenge by a multitude of stressors. Most of those they may require compensatory responses that elicit encountered by cattle in a well-managed enterprise are changes in the activities of other body systems that are amenable to the routine coping strategies of the governed by other regulatory actions. In those circum- animals; however, there are occasions when particular stances, the regulatory systems must have sufficient stressors seem to overwhelm the homeostatic flexibility to enable them to recalibrate operating set responses, to the detriment of welfare, health and points and to adjust the dynamic ranges of compensa- production. Apparently, the amount of stress an animal tory processes so that the interactions between the perceives depends on the intensity and duration of chal- brain, the autonomic nervous system and the endocrine

lenging stimuli and on the animal's physiological and system are geared to defend welfare, health and psychological state when challenged; as a consequence, ductivity in the face of ongoing and evolving challenges. the effects of a particular stressor may differ between That type of construct is exemplified by the physiological adaptations of a mother during pregnancy: as gestation progresses the set points and operating ranges of

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a plethora of homeostatic systems continually readjust the core of the stress system consists of two axes that in such a manner that the organ systems remain a harmonious team. serve to integrate neural and neuroendocrine activities:

in one, a neural pathway links the sympathetic division In this chapter we provide a synoptic overview of the of the autonomic nervous system with the adrenal homeostatic components of the 'integrated stress

medulla (the SA or sympathoadrenal axis); in the other, response' before proceeding to introduce a relatively hormones link the hypothalamus with the adrenal new paradigm of stress that recognizes that stressors cortex (the HPA or hypothalamic–pituitary–adrenal may activate multiple interactive mechanisms, that axis). The stress system is on continuous service: in the functional set points are subject to change, that disturbance of significant challenge it has a baseline, circadian pattern of activity that delivers homeostasis at rest; changes in one body system may elicit compensatory changes in another and that compensatory changes when challenged by perceived stressors it makes the come at a biological cost which may make the animal appropriate adaptive changes to maintain physiological vulnerable to other unrelated stressors – which cost is equilibrium, delivering what might be described as a determinant of the clinical consequences of environmental stress-related homeostasis.

mental stressors. We shall then examine the paradigm Anatomically, three elements of the nervous system

in relation to aspects of the more common problems interact to provide the regulatory core of the stress encountered in intensive cattle enterprises: lameness, system (Fig. 67.2):

mastitis, infertility, metabolic disorders, welfare, behavioural problems and levels of production.

- *Groups of noradrenergic neurones in the brainstem (the locus coeruleus–noradrenaline system; LC/NA);*

The stress system

- *Peptidergic neurones in the hypothalamus, mainly in the paraventricular nuclei (PVN), that release*
For didactic purposes, it is convenient to refer to the corticotrophin-releasing hormone (CRH) and arginine vasopressin (AVP);

work of the ‘stress system’ (Fig. 67.1). As mentioned,

- *Central neural projections that enable the LC/NA neurones and the PVN neurones to innervate and activate each other.*

Mutual interactions between the two axes are not

Stress

restricted to local neurocircuits. They are regulated in parallel by an extensive battery of humoral messengers: hormones, opioid peptides, cytokines, growth factors, metabolites – some stimulatory, some inhibitory and

CNS

HPA

SA

almost all counter-regulated by glucocorticoids

axis

axis

(Chrousos, 2000). For instance, the secretion of ACTH (which can be stimulated by hypoglycaemia, social

CRH

CRH

PNS

CNS

stress, fear, physical stress, exercise, acute illness or

Sympathetic

Cerebral

inflammatory cytokines) is inhibited by oxytocin,

Pituitary

Brain

nervous

catecholamine

gland

somatostatin and opiates, and is modulated by negative

system

systems

feedback by glucocorticoids. We shall have occasion to

return to the importance of counter-regulation again,

ACTH

and again.

Noradrenaline

Adrenal

Adrenal

Adrenaline

cortex

medulla

The stress response

Noradrenaline

Glucocorticoids Adrenaline

The principal function of the stress system is to release

Noradrenaline

appropriate amounts of glucocorticoids and cate-

cholamines from the adrenal glands and of noradrena-

Hormonal response

Neural response

line at peripheral nerve terminals, specifically to assist

the animal to cope with the demands of metabolic,

Fig. 67.1

Schematic overview of the stress system. CRH,

corticotrophin-releasing hormone; HPA axis, hypothalamic-

physical, social or psychological stressors. The SA axis

pituitary-adrenal axis; PNS, peripheral nervous system; SA axis, elicits responses much more rapidly than does the HPA

sympathoadrenal axis; ACTH, adrenocorticotrophic hormone.

axis; that is largely because it releases catecholamines

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HPA axis

SA axis

Humoral messengers

Brain

Hypothalamus

Brain stem

Neural projections

PVN

LC/NA

CRH

AVP

Cytokines

Spinal cord

Anterior

Lymphoid

pituitary

tissue

gland

Negative

ACTH

feedback

Adrenal gland

Cortex Medulla

Peripheral

sympathetic

system

Adrenaline

Glucocorticoids

Systemic circulation

Noradrenaline

Fig. 67.2

A schematic representation of cross-talk between the neural, endocrine, and immune systems when challenged by stressors. The cell bodies of the peptidergic neurones that govern the hypothalamic-pituitary-adrenal (HPA) axis are located principally in the paired paraventricular nuclei (PVN) within the hypothalamus. They secrete corticotrophin-releasing hormone (CRH) and arginine vasopressin (AVP; also known as ADH), both of which stimulate the release of adrenocorticotrophic hormone (ACTH) from the anterior pituitary gland. ACTH stimulates the adrenal cortex to release glucocorticoids (principally cortisol). The glucocorticoids exert negative feedback at hypothalamic and pituitary levels of the HPA axis and they counter-regulate the activities of both the sympathoadrenal (SA) neurones in the brain stem (locus coeruleus-noradrenaline: LC/NA) and (but not shown) the central humoral messengers that modulate the stress response. Neurones in the PVN and in LC/NA mutually innervate and activate each other. The SA axis releases noradrenaline within the brain (not shown), adrenaline and noradrenaline from the adrenal medulla and noradrenaline from peripheral sympathetic nerves. The cells of the lymphoid tissues release various cytokines that interact extensively with the neural and endocrine systems (see Fig. 67.6). Solid lines represent activation; dashed lines represent inhibition.

that can achieve their objectives by modifying pre-

Response of the SA axis

existing substrates within their target cells, whereas the

HPA axis releases steroids – hormones that direct the

When activated by stressors, the SA axis releases synthesis of new proteins that the target cells must noradrenaline from neurones within the brain and manufacture before they can proceed to do the business from peripheral sympathetic nerve endings; in addition. When the stress system is being aroused, the hypothalamus, it releases both adrenaline and noradrenaline from the PVN and the noradrenergic neurones at the PVN and the noradrenergic from the adrenal medulla. The noradrenaline released from the LC/NA activate each other and both centrally results in greater mental acuity ('arousal'), contribute to the adaptive behavioural and physiological changes whilst the catecholamines released peripherally promote changes in metabolism and in the activities of

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Noradrenaline

Box 67.1

Metabolic responses to stress.

- *Initially, insulin secretion is suppressed*

CRH

LC

decreased uptake of glucose by cells: hyperglycaemia

CRH

*breakdown of stored triglycerides: increased fatty acids
in blood*

Spinal

cord

- *Raised concentration of plasma catecholamines*

breakdown of stored glycogen:

in liver \rightarrow increased blood glucose

in skeletal muscle \rightarrow increased blood lactate

*breakdown of stored triglycerides \rightarrow increased fatty
acids in blood*

Pituitary

increased secretion of glucagon

gland

Adrenaline/noradrenaline

decreased secretion of insulin

*increased gluconeogenesis (from glycerol, lactate,
amino acids)*

- *Increased secretion of glucagon*

ACTH

breakdown of stored glycogen

Glucocorticoids

breakdown of stored triglycerides

increased gluconeogenesis

- *Rapid rise in secretion of glucocorticoids*

breakdown of skeletal muscle

Adrenal

breakdown of stored triglycerides

glands

*inhibition of uptake of glucose by tissues (other than
brain)*

Fig. 67.3

Schematic diagram of the functional inter-relations

increased gluconeogenesis

between corticotrophin-releasing hormone (CRH) and the locus

- *Increased release of arginine vasopressin (AVP, ADH)*

*coeruleus–noradrenaline (LC/NA) system. ACTH, adrenocorti-
retention of water*

cotrophic hormone. Adapted from E.O. Johnson et al. (1992).

- *Increased release of renin*

Solid lines represent stimulatory effects; dashed lines represent activation of angiotensin–aldosterone system

inhibitory effects.

retention of water and sodium ions,

loss of potassium ions

the cardiovascular and respiratory systems to increase

cardiac output, to facilitate oxygenation of blood, to

raise blood glucose concentration and to deliver the

enriched blood selectively to the tissues upon which the

The secretion of adrenaline is the more rapid response

stressors are making the greatest demands.

that underpins the flight/fight reaction, whereas the glu-

cocorticoids give a slower response that helps to real-

locate resources to meet the challenges to homeostasis

Response of the HPA axis

and, equally important, to curtail the potentially

The paraventricular nuclei secrete CRH and AVP in

harmful effects on health and welfare that could be

response to stressors. In cattle, the two peptides appear

exercised by exaggerated or unregulated autonomic

to be equipotent in stimulating the pituitary gland to

responses. The importance of counter-regulation of the secrete adrenocorticotrophic hormone (ACTH) which, autonomic component of the stress response is brought in turn, stimulates the adrenal cortex to secrete glucocorticoids. As the final effectors of the HPA axis, glucocorticoids (chiefly, cortisol) participate in a multitude of activities that maintain whole-body homeostasis, including feedback regulation of both axes of the stress contraction of arterial smooth muscle and at the same system (Fig. 67.3) and the metabolic readjustments time induce metabolic changes (insulin resistance, (Box 67.1) required to sustain the physiological adaptations invoked to cope with stressors.

pose to arterial damage and the formation of atherosclerotic plaques in the coronary arteries.

Counter-regulation of the SA response is a key

Counter-regulation

element in an animal's ability to cope with environ-

The stress response can increase the concentrations of mental stressors without incurring a residual physiological toll of inordinate proportions.

logical toll of inordinate proportions.

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Coping with stressors

tract. Another consequence of general activation of the sympathetic system is the release of renin, leading to

Animals may employ active coping strategies to reduce activation of the angiotensin–aldosterone system, and the aversive effects of stressors or they may use passive consequent conservation of sodium and water, accompanying strategies to minimize the biological cost of the panied by excretion of potassium ions.

stress response evoked by the stressors. Moberg (2000)

identified four general types of biological responses

Neuroendocrine responses

available to an animal to cope with stress: behavioural, autonomic, neuroendocrine and immunological (Fig.

Within minutes of the LC/NA response, the HPA axis
67.4). Although all four biological defence systems are
is called to arms but, because steroids take longer than
available to respond to a stressor, an animal does not
amines to elicit responses, there is a time lag before the
always call on each of them to deal with each stressor:
glucocorticoids begin to exercise dominant control –
different stressors elicit very different types of biologi-
with adrenaline, growth hormone and thyroid hor-
cal responses. Nevertheless, in the vast majority of
mones in supporting roles. The combined activities of
instances the response will contain some element of all
these hormones result in metabolic adjustments (Box
four.

1) while the animal continues to resist the stressor(s) or
until its reserves of energy have been exhausted. The
first priority is to maintain blood glucose concentration

Behavioural responses

and to conserve the glucose for utilization by the neural

Behaviour offers the animal choices: to confront the

system. The liver can liberate glucose from stored

stressor aggressively, to remove itself from the stressor glycogen. Skeletal muscles do not possess the full complement of enzymes necessary to do this; there the impact of the stressor. In many instances a behavioural response is the simplest and most biologically economical reaction to a stressor; for instance, simply walking away. However, some displacement activities (e.g. stereotypies such as compulsive walking) may expend an enormous amount of energy to no purpose.

down of triglycerides stored in adipose tissues to yield the required fatty acids plus glycerol. These glucose-sparing actions are augmented by the synthesis of new Autonomic (LC/NA) responses

glucose from endogenous substrates: the glycerol

When the situation is not immediately resolved by a released from adipose tissues is made available for the behavioural response, the reaction is directed by a general synthesis of glucose in the liver (gluconeogenesis), as is generalized activation of the sympathetic nervous system the lactate released from muscle glycogen. Concurrently, the same hormones induce proteolysis in skeletal muscle, releasing amino acids that the liver also uses by a heightened state of arousal, by increases in blood pressure, heart rate, respiratory rate, blood flow to manufacture glucose. The process of gluconeogenesis is regulated by glucocorticoids (HPA axis), by skeletal muscles and consumption of energy and by glucagon and by adrenaline from the adrenal medulla decreased blood flow to skin, kidneys and digestive (SA axis).

Behavioural

response

Autonomic

response

(SA axis)

Stressor

Host perception

Arousal

Allostatic load

Neuroendocrine

response

(HPA axis)

Fig. 67.4

Coping with stressors. SA axis, sympathoadrenal axis; HPA axis, hypothal-

Immune

response

amic–pituitary–adrenal axis.

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Immunological responses

ish the body's metabolic reserves and how does this impact on health and welfare?

The two axes of the stress system are at all times

In the first instance, the biological cost of stress is met engaged in bidirectional communication between from the body's reserves of energy. For many short-the central nervous system and the immune system. The lived stressors that cost is negligible: it is met from cross-talk is intensified during the stress response. The reserves without any impact on other biological func-brain issues messages through the SA and HPA axes tions. As an example, catecholamines secreted during that affect the activities of the cells of the immune stress stimulate the conversion of stored glycogen into system, while products elaborated by various immuno-glucose; once the stress has been alleviated, the glyco-cytes affect the functions of the neurones at the centre gen stores are quickly replenished by gluconeogenesis; of both axes. In brief, the corticosteroids and the cate-there is no lasting deficit. On the other hand, when cholamines down-regulate the activities of the immune there are insufficient biological reserves to meet the system: they inhibit many of the functions of leucocytes, costs, resources must be diverted from other biological

macrophages and lymphocytes, and they decrease the functions: for example, if a young heifer reared under production of many cytokines and mediators of inflammatory stressful conditions has to divert a significant fraction of the energy normally expended on growth and released in response to stress are, in general, immunoreproductive it is likely that her growth will be retarded suppressive. Early in the response to stress there is and puberty will be delayed. In other words, for some increased release of growth hormone and prolactin, persistent or intense stressors the cost is high, a significant burden on the body that places the animal 'at risk both of which enhance the immune response; however, the circulating levels of both hormones are decreased of developing pathologies' (Moberg, 2000). The gap later in the stress response and, also, when there is between those two extremes is filled by examples in recurrent or chronic stress. Thus, the net effect of stress which the hidden costs of coping strategies so deplete is immunosuppression and increased susceptibility to

biological reserves that the animals develop 'pathological infectious diseases, particularly those that target the respiratory system or the digestive system. There is considered to be rather minor, inconsequential factors. Such are the cases that raise the questions that need to be addressed by the clinician in the field: how does that attenuate the immune response.

stress influence the pathogenesis of disease and what accounts for the variation in susceptibility to stress-

Physiological toll

related diseases amongst animals in the same, or similar, environmental circumstances?

If stress is intense and prolonged, the coping strategies may become part of the problem. Rapid production of glucocorticoids in response to a stressor is beneficial,

The transition to pathology

provided an efficient shut-off can occur; chronic elevation of glucocorticoids persisting after the stressor has

There are several routes by which coping strategies can gone indicates poor adjustment and is detrimental to progress into pathology.

health. Prolonged elevation of glucocorticoids in the circulation can cause excessive breakdown of muscle

- If stress is intense and long-lived, the coping strategies may become part of the problem. The potential pathological consequences of prolonged reaction, increased susceptibility to infection and elevation of glucocorticoid concentrations have delayed healing of wounds. The principal mechanism by been mentioned above. Persistent activation of the which the body is protected from these deleterious SA axis, which stimulates the renin–angiotensin–effects is feedback inhibition of the HPA axis by the aldosterone system, may also translate into pathology: initially, by progressively increasing blood volume and raising blood pressure; later, as a*

consequence of the alterations in neuromuscular

The biological costs of stress

excitability due to hypokalaemia.

- *Obviously, whenever coping strategies have*

All stressors, no matter how innocuous, have some exhausted the biological reserves of the animal it is biological costs. There is a price to pay, even when the to be expected that further challenge(s), even by homeostatic balance is restored promptly. The real inconsequential stressors, may translate into

questions are: to what extent does the challenge diminish pathology.

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- *A coping strategy may increase the animal's exposure to environmental factors that damage its health*
A simple example: if, for whatever reason, blood (e.g. the housed subordinate cow that copes with glucose concentration falls, the neuroendocrine axes dominant cows by standing and lying at a distance in

will set about correcting the deficit by typical homeo-
damp, dirty, unsheltered locations) or it may deny
static responses that implement glucose-sparing meas-
the animal the opportunity to participate in activi-
ures augmented by gluconeogenesis.

ties that promote its health and welfare (e.g. limit its
It is entirely appropriate that those components of
access to feed or, in the extreme, an animal exposed
the internal milieu that are absolutely essential for life
to chronic, unavoidable stress may develop 'learned
(for instance, oxygen tension, pH, body temperature)
helplessness', become recumbent and stop eating).

should be governed according to strict homeostatic

- A coping strategy may involve a physiological
principles that operate within narrow ranges about
response that directly contributes to a pathological
fixed set points. However, life expects the body to be
process (e.g. a very thirsty, salt-deprived animal
capable of adapting to marked variations in its natural
consumes excess water and develops haemolysis
environment and that is possible only if the animal can

and cerebral oedema: water intoxication).

continuously readjust a number of parameters toward

- A coping response may attenuate or suppress an new set points and wider operating ranges that are important component of the animal's normal appropriate to the challenging circumstances in which defence system and leave it vulnerable to a disease it finds itself. Put very simply, different circumstances unrelated to the original stressor (e.g. attenuation demand that the several interacting systems vary their of the immune response by transportation stress homeostatic set points to ensure that their activities renders an animal susceptible to respiratory remain in synchronesh. Allostasis enables the SA axis, disease).*

the HPA axis and the cardiovascular, metabolic and immune systems to effect the changes in the internal In each scenario the transition to pathology is pred-milieu that make the most beneficial use of the animal's icated on a changed (or changing) internal milieu that biological resources; in brief, they induce a new steady

cannot be explained in terms of strict homeostatic state based on a panel of readjusted set points in dogma. The principle of homeostasis is that the body counter-balancing systems.

must hold all its regulated parameters within very narrow operating ranges centred on the relevant set points. Allostasis is a useful concept but, as McEwen and Stellar (1993) pointed out, it does not take into consideration the strain on the body produced by the activities that that is not how things are in the real world. It is recognized that an animal exposed to intense or persistent stressors may have to vary many parameters of the impact of wear and tear on a number of organs and its internal milieu and match them appropriately to environmental demands. Sterling and Eyer (1988) introduced the term allostatic load to describe the referred to this principle as allostasis, meaning 'stability

aggregate biological costs that result from chronic over-through change’.

activity or underactivity of allostatic systems.

As McEwen (1998) perceived it, there are three phases in the body’s response to challenge: it must

Concepts of allostasis and

mount an adequate allostatic response, it must maintain

allostatic load

that response until the stress ceases and it must shut off

the response when the threat has passed. Thus, allosta-

When an animal perceives a situation as stressful it

tic load can accrue when:

makes the behavioural and physiological responses that

it considers most appropriate. Experience will help the

- *There is an inadequate response that leads to*

compensatory hyperactivity of other regulatory

animal assess how much of a threat is posed and what

systems;

it requires to do to ward off that threat. If a particular

stressor has been encountered previously and been

- *The body is subjected to repeated challenges from*

multiple stressors;

found to be easily controlled, the response will be measured accordingly; habituation will ameliorate the

- There is failure of habituation to repeated challenges of the same type;*

physiological perturbation and the biological cost will be reduced. The responses are mediated by the SA and

- Delayed shut-down prolongs the response after the threat has passed.*

HPA axes and, if the stressor is mild and short-lived, homeostatic regulation will modulate the perturbations

When circumstances improve, with environmental within tolerable operating ranges until the regulated stress consistently at a low level, the animal may

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discharge much of the allostatic load, to the benefit of parameters persistently outside their normal operating its health and production.

ranges – whether because of many significant chal-

On the other hand, if life remains stressful the load lenges or because the allostatic responses remain

will accumulate and the animal will find it progressively turned on when no longer required – the body incurs more difficult to make effective readjustments in a cumulative biological burden that constitutes its response to further challenges: in a figure of speech, a allostatic load.

point may be reached at which all the slack in biologi-

The biological costs of each challenge to homeostatic reserves has been taken up and the next coping

sis are influenced by the genetic background of the strategy will propel the animal into overt dysfunction.

animal, its previous history, the magnitude of the allo-

In many instances, the final challenge that breaches the static load it has accumulated already and the func-

boundary between the compensated state and ‘pathological priorities of the current phase of its life cycle, as

ogy’ may be relatively minor and, if it is identified, it

well by the nature of the stressor, its novelty, its inten-

may be assigned an exaggerated degree of causal

sity, its frequency, its duration and the types of

importance that is an impediment to the exploration of

responses it elicits.

the real sources of allostatic load within the herd, which

Thus, when clinical disease is manifest the cascade of

must be the ultimate target for the dedicated clinician.

environmental factors that have contributed to the

More immediately, failure to see the 'big picture' may

current state of the sick animal will have operated at

militate against the efficacy of treatment of the diag-

two levels: the proximate and the ultimate. The proxi-

nosed ailment – it is unlikely that targeting what, in

mate factors (infectious, metabolic, toxic or whatever)

essence, may be just the tip of a dyshomeostatic iceberg

have made the immediate challenges that caused the

will successfully restore the animal to full health and

presenting signs. The ultimate factors have been

production. Recognition of the bases from which the

responsible for the allostatic load, for the depletion of

allostatic load has accrued should enable the clinician

energy reserves and the cumulative wear and tear of

to implement a more holistic, more successful, approach

vital organs and their regulatory controls that have

to the problem.

predisposed the animal to the ravages of the proximate

In summary, the concept of allostatic load provides a

stressors. In brief, allostatic load is the burden of costs

rational hypothesis to account for the contribution of

that have been paid at several toll-bridges on the road

environmental stress to the pathogenesis of dyshome-

to clinical disease. Increasingly, trends in intensive

ostasis, a sometimes precarious state of readjusted

systems of animal production will force us to bring that

parameters that, when propelled into decompensation,

toll into sharper focus in our deliberations on the patho-

can be manifest as any of a range of different ailments

genesis and treatment of ailments and on the husbandry

in individual animals within a herd.

and welfare of farm animals.

The stress response can be mobilized in response to

physical or physiological stimuli, and also in expecta-

Allostatic load and the

tion of them. As is evident from the early part of this

veterinary practitioner

chapter, the brain is the coordinator of the body's responses to the stimuli: it decides whether or not a How should the concept of allostatic load inform the potential stressor poses a threat, and how the animal veterinary practitioner in the field? How does it relate should cope with it. The cognitive appraisal of the environmental stimulus determines the response: if it is not enterprises: lameness, mastitis, infertility, metabolic disorders, welfare, behavioural problems and levels of On the other hand, novel stimuli will be perceived as production? threats ipso facto, even though they may not be particularly aversive in character; the intensity of the responses will decline as the novelty wears off. It is 'Life is stress, stress is life' neither possible nor desirable to isolate livestock from Life is a minefield of potential stressors that each creature has to negotiate by recourse to its repertoire of

dynamic environment of the modern intensive cattle
cognitive, behavioural, neural, endocrine and immuno-
enterprise. Some types of mild stimuli are beneficial; for
logical modalities. The ability to adapt successfully to
instance, they induce a state of arousal in which meta-
the multifarious challenges of life – or, in the words of
bolic processes are adjusted for rapid expenditure of
Sterling and Eyer (1988), the ability of the ‘organism to
energy and they ‘tune’ the cognitive and motor mecha-
vary parameters of its internal milieu and match them
nisms that the animal will call upon whenever it is chal-
appropriately to environmental demands’ – is called
lenged by a significant stressor. Furthermore, there is
allostasis. When the adaptive responses put those
convincing experimental evidence that when the stress
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system is activated in that manner, productivity is
Partitioning of metabolic fuel
improved and some immune functions are enhanced, at
Each organism has a limited energy budget from which
least in the short term.

it must allocate resources prudently if it is to maintain the body and perpetuate the species. When the supply

Practical considerations

of energy falls below total requirement, the available metabolic fuels are partitioned according to priorities

The scientific literature, particularly the medical literature, has concentrated unduly on the negative aspects

of stress, on its role in the pathogenesis of disease; in

either because the intake has been reduced below

reality, the stress system has evolved as an essential

normal expenditure, or because the expenditure has

defence mechanism that plays a very positive role in the

survival of organisms beset by stressors. The task is to

manage the sources of environmental stimuli to the

major stressors. In the trade-off between competing

mutual benefit of the animal and of the commercial

energetic requirements, environmental stressors can

imperative; that requires attention to the structure exercise a defining role. Top priority is given to main- and hygiene of the physical facilities, and to the social tenance of basic cellular functions, thermoregulation contexts, metabolism, physiology, vulnerabilities and and locomotion for foraging (not a problem in a coping strategies of the animals. Several of those topics modern cattle enterprise). When the short-fall is severe, are dealt with in some detail elsewhere in this book; less and less of the intake goes to growth, fat storage or here, under several subheadings, we cherry pick some reproduction; in extreme circumstances these activities clinical conditions that illuminate the central thesis: that will be suspended until they can be resumed when the allostatic load is a significant factor in the pathogenesis energy balance has been restored.

of many ailments, that sick animals should be seen as sentinels of stress within the herd and that the thera- Counter-regulation by glucocorticoids peutic, prophylactic and managerial measures are unlikely to be entirely satisfactory unless that fact is

As mentioned, negative feedback by glucocorticoids taken into consideration. Although the vast literature plays a significant role in protecting the organism on the stress response has yet to generate a unitary paradigm against 'overshoot' by either limb of the stress system adigm that would inform the clinical judgement of a (Fig. 67.3). In addition, they contribute to the effort to veterinary surgeon in relation to those decisions, there conserve energy whenever stressors threaten home- are two less ambitious constructs, mentioned above, ostasis: they exert inhibitory effects at more than one that are relevant to the message of this essay: the paradigm point in each of the endocrine axes that regulate titioning of metabolic fuels and the counter-regulatory growth, thyroid function, reproduction and immune activity of the corticosteroids.

function (Fig. 67.6).

Procurement

Metabolism

and ingestion

and partitioning

Expenditure

Liver

Reproduction

Thermoregulation

Oxidizable

FOOD

metabolic

Cellular maintenance

Fig. 67.5

Partitioning of metabolic fuels. When

fuels

the supply of energy is insufficient to meet

demands, non-essential processes such as repro-

Locomotion

duction and growth are deferred, and fat stores are

mobilized to ensure that the essential processes

Growth

of basic cellular metabolism, thermoregulation and

Adipose tissue

locomotion are maintained. (After G.N. Wade &

fat stores

J.E. Schneider, 1992).

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(a) Reproduction

(b) Growth and thyroid function

(c) Immune function

b-endorphin

GHRH

CRH

TRH

CRH

GnRH

CRH

STS

STS

GH

ACTH

TSH

Cytokines

ACTH

(IL-1, IL-6, TNF)

LH, FSH

ACTH

Cortisol

T4

Mediators of

inflammation

Glucocorticoids

SmC

T3

(eicosanoids,

Oestradiol

PAF, serotonin)

Cortisol

Testosterone

Target tissues

Target tissues

Target tissues

Target tissues

ACTH: Adrenocorticotrophic hormone

SmC: Somatomedin C

CRH: Corticotrophin-releasing hormone

STS: somatostatin

FSH: Follicle-stimulating hormone

T3 and T4: thyroid hormones

GH: Growth hormone

TNF: Tumour necrosis factor

GHRH: Growth hormone-releasing hormone

TRH: Thyrotrophin-releasing hormone

IL-1: Interleukin-1

TSH: Thyroid-stimulating hormone

IL-6: Interleukin-6

Fig. 67.6

Interactions of the HPA axis with the systems that subserve (a) reproduction, (b) growth and metabolism, and (c) immune function. Solid lines represent direct or indirect activation; dashed lines represent direct or indirect inhibition. (After Stratakis & Chrousos, 1997.) **Clinical implications**

Social stress may be a source of significant allostatic load.

Social context (see Chapter 55)

Foot lameness (see Chapter 31)

Each herd of cattle develops its own social structure, a hierarchy in which social rank is held at a price. Social

It is accepted that poor quality horn is a significant interactions within the group can be stressful: deter-

factor in the pathogenesis of foot lameness in lactating
mined subdominant animals challenge the dominant
dairy cows. The defect in the horn is associated with
animals; in turn, dominant animals bully subordinate
impaired keratinization in the epidermis of the horn, a
animals. Several lines of evidence suggest that the mag-
process that is known to be influenced by the dominant
nitude of the allostatic responses to social stressors is
hormones in late pregnancy and early lactation. Particu-
influenced greatly by the social rank of the animal: the
larly relevant to the present discussion is the evidence
biological costs are greatest at the two extremes of the
from hoof explants in organ culture that glucocorticoids
hierarchy. The allostatic load – and the loss in produc-
inhibit the synthesis of hoof protein. It has been sug-
tivity – depends on the animal's perception of the threat
gested that the elevated systemic concentrations of
to its well-being and on its coping strategy. The active
glucocorticoids during lactation could contribute to the
coping strategy of the dominant animal can be as costly
deterioration in horn quality in the lactating cow. It is

as the passive coping style of the subordinate animal.

conceivable that other stressors that induce the release

When an animal is exposed repeatedly to the same

of more glucocorticoids would exacerbate that deterior-

predictable stressors, the intensity of the stress reaction

ration. For instance, poor quality horn that predisposes

tends to decline progressively. This process of habitua-

to foot lameness is not uncommon in recently-calved

tion appears to be stressor-specific; the animal retains

heifers joining the milking herd for the first time; in

the capacity to make an acute response to a novel stres-

those circumstances, horn is being produced by an

sor. Therefore, within a settled herd the toll of social

immature animal whose endocrine organs are secreting

stress can be attenuated by good stockmanship that

unprecedented quantities of lactogenic hormones aug-

seeks to shield the animals from all that they will per-

mented by the hormones released in response to the

ceive as novel, frightening, painful, unpredictable,

social stress – and whose metabolic stability is being

frustrating or uncontrollable.

challenged by the unaccustomed demands of her first

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lactation. Thus, the presenting ailment may be a mani-

reproduction is suppressed while the animals are in

festation of a 'big picture' that incorporates a number

negative energy balance: that the hypothalamic–

of elements of systemic dyshomeostasis – abrupt expo-

pituitary–gonadal (HPG) axis is inhibited while the cow

sure to a novel hormonal milieu, unrelenting demand

'milks off her back'. Most high-producing dairy cows

for an unprecedented quantity of energy, and social

enter the breeding season in a negative energy balance;

stress.

some will not have achieved a positive balance before

Allostatic load may find expression in a presenting

the end of the third month after calving, at which time

ailment that has no obvious anatomical link to the

they are expected to be in-calf again. From an energetic

systemic dyshomeostasis that has made the host

perspective, this means that the biological costs of reac-

animal vulnerable to 'pathologies'.

tivating cyclic activity of the HPG axis and its associated organs have to be abstracted from a budget that is in negative balance because it has to support, either in Infertility (see Chapters 33–37)

sequence or in parallel, the extensive hormonal changes

Reproductive functions in the female are more susceptible (qualitative, quantitative and temporal) that accompany the transition from pregnancy to lactation; the biological costs of uterine involution; the extraordinary investment of metabolic resources over a much longer time; secondly, because stressors are prone associated metabolic adaptations; the extensive adaptations to disrupt the precision with which a number of hormones must be secreted in appropriate quantities and biological costs of immune defence of organs newly opened to ascending infection (uterus, mammary

in order to establish a pregnancy. It is known that short-gland); the transition from housing to pasture and the term stressors (such as fear engendered during herding, associated meteorological and nutritional changes; handling or transport, electric prods or stray voltage) together with any number of contingency costs. Despite can interfere with fertility if they challenge the cow at all that massive drain on body reserves, approximately a critical juncture. To a considerable degree, the half of the mated cows conceive to first service. It is response of the female to stressors is predicated by a clear that although the energetic perspective provides desire to conserve energy until the threat is past; clearly, a rational account of the drain on body resources, it that is the case when she falls into a negative energy does not provide a comprehensive explanation for balance and suspends her reproductive activities pro either the infertility of a sizeable number of clinically tem. Fortunately, the suspension leaves the regulatory normal cows or for the relatively poor response to neuroendocrine apparatus intact and normal business

hormone therapy in those animals.

should be resumed when the energy balance has been

It is likely that much of the infertility reported in

restored. In reality, spontaneous resumption often takes

clinically normal cows is due to unrecognized allostatic

longer than is ideal for the commercial enterprise; this

load; in other words, it is not a disease in the traditional

is particularly significant in relation to the low calving

sense, rather a suspension of service due to a fault in

rates achieved in the early post partum period – which

the signalling system. Therefore, the protocol for clini-

can be a cause of considerable economic loss, especially

cal assessment of cows being prepared for the breeding

in a herd where a seasonal breeding programme is in

season should include a check-list of potential stressors

operation.

to which the animals due for examination are most

Over the past 20 years, the dairy industry in the UK

likely to have been exposed; the prophylactic manage-

has switched from a predominantly British Friesian cow

ment of the breeding herd should take account of the

population to predominantly North American Holstein findings.

cows and, further, it has engaged in intense selection for

The effects of stress on reproductive functions are

milk yield. The records show that over that period the

complex; however, it is reasonably close to the truth to

pregnancy rate to first service has declined at an

say that most of the adverse effects are mediated by

average rate of approximately 1 per cent per annum,

interactions between the HPA axis and the HPG axis

and that atypical ovarian hormone patterns have been

(Fig. 67.6(a)). Corticotrophin-releasing hormone (CRH)

contributing factors to that reduction (Royal et al. , can suppress the secretion of gonadotrophin-releasing

2000). The response to hormonal therapy has not been

hormone (GnRH) via an opioid-mediated mecha-

consistently good; it has fallen considerably short of

nism, supranormal levels of androgens from the adrenal

that which current knowledge of reproductive biology

glands can distort the negative feedback regulation at

would lead us to expect.

the hypothalamus and pituitary gland and glu-

*A plausible hypothesis advanced to account for the
cocorticoids can exercise inhibitory effects at all critical
decline in fertility in high-producing cows was that
points in the HPG axis (and even as far as the target*

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*tissues for gonadal steroids). It is probable that the most
logical defences. When the stored fat is mobilized, some
important factor in the suspension of reproductive activ-
is deposited in liver, kidneys, adrenal glands and muscle.
ity is interference with the pulsatile release of GnRH by
The fatty infiltration of the liver can be severe in cows
the hypothalamus, which alters the pulsatile release of
that are overfat at calving and in cows that mobilize fat
LH by the pituitary gland that, in turn, regulates ovarian
faster than it can be utilized. Subclinical fatty liver has
activity. However, in practice, inhibition at other points
been linked to increased susceptibility to ketosis, milk
in the HPG axis may be just as significant if it reduces
fever, mastitis, lameness and infertility.*

the prospects of success when exogenous hormones are

The important features here are that metabolic perturbations used to 'kick start' the inactive axis. For instance, exogenous GnRH may be an adequate stimulus to provide a functional pulsatile release of LH, but its ability to period and that the magnitude of that load can be a significant factor in determining how she copes with the lead to the removal of inhibition or to the acquisition of subclinical metabolic problems that invariably arise in essential receptors at other points around the HPG axis the early weeks after calving. Failure to cope renders or in the peripheral target organs.

her vulnerable to a plethora of stressors that are rife at that time and act as proximal factors in the pathogenesis of several clinical diseases, metabolic or infectious. challenges experienced during early lactation exert a

Inappropriate nutrition can be a significant factor

residual inhibition on the biochemical activities of the
in the pathogenesis of a variety of metabolic and infec-
HPG axis and that this inhibition can be of varying
tious diseases.

duration depending on the magnitude of the allostatic
load and the ability of individual animals to cope with
Coping with pathogens
it.

Allostatic load may find expression as a bio-

By definition, pathogens are stressors. The magnitude
chemical lesion.

of the biological costs they impose on their hosts is
determined by the virulence, infective dose and novelty
of the pathogen and by the nature, location, intensity
Metabolic diseases (see Chapters 46, 47)

and duration of the host response, together with the
The peripartum period is a stressful time in the life of
pre-existing allostatic load of the host. In turn, stress is
a cow. During the ‘transition period’ (the last four
a risk factor for a variety of infectious diseases, partic-
weeks of gestation and the first four weeks of lactation)

ularly in the periparturient period and especially in cows experience enormous changes in the hormonal individual cows who enter the transition period with milieu, a negative energy balance and a plethora of appreciable allostatic loads, and in cows that are genetically predisposed to exaggerated responses of the HPA and SA systems to stressors.

portion of the metabolic and infectious diseases to Risk of infection can be increased when the stress which cows are subject occurs during the transition response leads to behavioural changes that increase phase, many of them having metabolic perturbation as exposure to pathogens, when it impairs the immune the ultimate source of allostatic load. Metabolic perturbation (whether to an invading pathogen or to a latent resident), when it contributes to the progression of an existing pathological process or when it is

It is recognized that absorption of glucose by the prolonged unnecessarily.

intestine contributes little to the energy homeostasis of

It is accepted that stress has a negative effect on the lactating cows. The large and immediate requirement of immune response and it is often said that the effect is the lactating mammary gland for glucose is met by a largely due to the corticosteroids released during the reduction in utilization by several other tissues and by stress response. The reality is a very much more an increase in synthesis from fats and proteins by the complex scenario that involves the nervous, the liver (gluconeogenesis). The key role is played by lipid endocrine and the immune systems. The different types metabolism that provides a rich supply of substrate for of immune cells possess receptors for the signals issued gluconeogenesis. The amount of energy reserves upon by the HPA and SA axes; in turn, immunocytes release which the postparturient cow can draw is a product of cytokines that modulate the activities of the stress the feeding regime during the recent dry period. At the

system.

onset of lactation there is extensive mobilization of

Recent research indicates that stress may boost

body fat and in many cows this gives rise to a state of

humoral immunity as it suppresses cellular immunity –

subclinical ketosis, a condition that impairs immuno-

a response that is due to the effects of glucocorticoids

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and catecholamines on the differentiation of T helper

future challenges. This strategy is used in dealing with

cells. The stress hormones modulate the secretion of

a range of microbes and parasites and, in addition to

different types of cytokines by antigen-presenting

enhancing the host's immune repertoire, it represents

cells (macrophages and other phagocytic cells); those

prudent use of energy reserves since the likelihood is

cytokines influence the differentiation of antigen-naïve

that the biological cost of total elimination would be far

T helper cells (Th0) into two subsets (Th1 and Th2)

greater than the net cost of maintaining a resident

that, in turn, secrete cytokines that are involved in the

population to which the host becomes habituated – and acute phase response, in sickness behaviour and in the which brings such benefits as on-going priming of local differential regulation of cellular and humoral immunity to the pathogen and competitive inhibition. The cytokines secreted by Th1 cells promote of other pathogens that might attempt to invade the cellular immunity, those secreted by Th2 cells promote location.

humoral immunity. The Th1 and Th2 responses are Given that infectious agents impose an increased mutually inhibitory; it is thought that the Th2 shift that metabolic and nutritional load on their hosts, it is astonishing that voluntary reduction in food intake appears important implications for the course and outcome of to be a beneficial coping strategy in some viral, bacterial, infectious, allergic, autoimmune and neoplastic diseases. For instance, there is experimental evidence that when mice infected with *Listeria*

instance, lymphocytes can synthesize ACTH, growth monocytes were force fed to the same energy hormone and prolactin – messengers that probably act intake as their uninfected controls, they had a higher locally in a paracrine fashion. In addition, there are mortality rate and a shorter survival time than did the functional contacts between nerve fibres and immunoinfected mice that were anorexic. Clearly, in this cytes: for instance, lymphoid organs are innervated by instance, force feeding nullified the benefits of a natural nerve fibres that release noradrenaline or neuropeptides which interact with the immunocytes to cause death. It is legitimate to question the wisdom of force changes in cellular trafficking, lymphocyte proliferation feeding sick animals.

tion, antibody production and cell lysis. The physiological and pathophysiological consequences of all this infection and anorexia – are somewhat at variance with cross-talk are most evident to the veterinarian in the

the thrust of conventional wisdom; nevertheless, their periparturient cow.

adoption by diverse species indicates that they bring

*The transition period is recognized as a time of relationship significant benefit to the host. Therapeutic interventions
tive immunosuppression and increased susceptibility to
should not run counter to the way of nature.*

infectious diseases – whether viral, bacterial or para-

Therapeutic interventions in infectious diseases

sitic. The periparturient cow is at serious risk of infec-

should be consonant with coping strategies, including

tion because, at parturition, the uterus and the udder

those that appear to be contrary to conventional

lose the physical barriers that have protected them

wisdom and expectation.

from ascending infections; thus, bacterial pathogens

may enter while the defences are at their weakest.

Altered behaviour

Leukocytes play key roles in the defence of both

organs: prompt migration of neutrophils (PMNL) is

Changes in behaviour may be evoked as coping strate-

essential to overcome the bacteria; however, the activities (confrontational, submissive or socially neutral) or activities of the leucocytes are depressed as a consequence they may emerge in the course of stress-induced illness of the negative energy balance, the subclinical ketosis, – in the form of listlessness, exaggerated sleepiness, the fatty liver and the raised circulating concentrations weakness, depressed locomotor activity, shivering, of glucocorticoids. Some recently-calved cows develop diminished social interaction, anorexia, decreased a severe systemic illness when the influx of PMNL does foraging and absence of body-care activities. Current not materialize in response to invasion of the udder by opinion amongst those who study infectious diseases is *Escherichia coli*. However, in most instances the cellular and humoral responses control the proliferation of an adaptive response to antigenic challenge and that the invading bacteria without achieving total elimination; they are elicited by the pro-inflammatory cytokines that for instance, *Staphylococcus aureus* frequently is released from immunocytes to mediate both the

relegated to subclinical residence in the udder. This is local (inflammatory) and the systemic (febrile and an important adaptive strategy: its immediate effect is behavioural) components of the reaction to foreign to protect the organ from serious tissue damage, while antigens. In that interpretation, sickness behaviour and it sets up a mechanism by which the organ can maintain a satisfactory level of local immunity to cope with mental concern to the therapist: each is an important

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component of the natural defence system; in excess, Quite often the downer cow syndrome is seen as an each can be detrimental to the host.

incomplete response to treatment for milk fever: it is Cytokine-induced anorexia can be interpreted as a said that the longer the interval between the time the way to reduce the availability of some nutrients (e.g. cow becomes recumbent and treatment for milk fever, iron, zinc) necessary for multiplication of the the greater the incidence of the downer cow syndrome.

pathogens. Sleepiness and reduced activity can be inter-

One has to entertain another possibility: that some

preted as ways in which to conserve energy that the sick

recently-calved cows, already burdened with substantial

animal will re-allocate to new priorities geared toward

allostatic loads and with the added stress of acute

victory over the pathogens (e.g. inflammatory and

hypocalcaemia, may find rapid intravenous injection of immunological responses). Therefore, as far as is possible, essential ions to be a stressor too many – sufficiently

ble, our treatment modalities should not nullify the

stressful to necessitate a period of recumbency in which

natural benefits of sickness behaviour – as force feeding

to recover the will and the confidence to resume normal

did to the Listeria-infected mice.

activities. When management fails to recognize the

The behavioural changes evoked by the stressors

cow's immediate need for a period of relative in-

may be transient (e.g. failure to groom during illness),

activity she will be subjected to repeated premature

persistent (e.g. stereotypies) or life-threatening (e.g.

attempts to force her to stand; this will increase the risk

learned helplessness). Learned helplessness is an of physical damage and, by the same token, it is likely unmotivated state in which an animal gives up trying to to reinforce the state of helplessness that now is at the cope with the exigencies of life, is indifferent to its own core of her problem.

well-being and is unresponsive to therapeutic or punitive interventions. Originally, the term was used by may have a morphological basis somewhat analogous scientists who assessed the coping abilities of dogs to the neuronal damage seen in the hippocampus of exposed to repeated electric shocks. They found that chronically stressed humans, shrews and rodents. If that dogs that had been subjected to unavoidable shock were the case, the evidence from the other species treatments in a preliminary experiment just sat and would be reassuring: the neuronal damage is neither took the shocks when the experiment was repeated in terminal nor irreversible. Most of the affected cattle a situation from which there was a fairly obvious means

should respond to standard treatment, always provided of escape. Subsequently, it has been recognized that the clinical problem is not compounded by musculoskeletal damage incurred during ineffectual attempts species (including humans), notably when they have to get the cow on her feet before she is ready to do so endured repeated failed attempts to achieve an of her own volition or by complications arising from objective.

prolonged recumbency.

The author believes that he has encountered this
These observations on altered behaviour highlight cognitive aberration in cattle originally thought to two significant aspects of animal health: firstly, sickness be suffering from metritis, mastitis, acetonaemia or behaviour is an adaptive response to infectious or non-hypocalcaemia, but most convincingly in a subset of infectious stressors; it provides the allostatic base from downer cows that did not have the physical or biochemical which the recuperative process develops and it is instru-

ical defects to account for their predicament (pp. 439, mental in setting the endogenous rhythm for that 797). From clinical experience, he suspected that some process; secondly, any therapeutic intervention that of the underlying allostatic load in the helpless downer jolts the periodicity of the process becomes an additional stressor that is likely to delay or distort perhaps, from previous lactation(s). If the conjecture is resolution.

correct, it follows that a comprehensive record of health **Sickness behaviour is a strategy that may be invoked** and production parameters should reveal more about **to cope with either infectious and non-infectious stressors; interventions that target the behaviour rather** the bases from which the load accrued than would the most recent metabolic profile. Learned helplessness may **than the stressors are liable to delay, distort or prevent** be associated with either infectious or non-infectious **the recuperative process.**

conditions; it is regarded as a putative animal model of

depression. The pathophysiological mechanisms have not been elucidated but some evidence from animal

Concluding comments

experiments hints at an imbalance between the activities of the two limbs of the stress system, with the HPA axis

The primary aim of this chapter is to draw attention to in the ascendant. It would not be prudent to treat such the concept of allostatic load and to encourage practitioners to appreciate how important a role it plays the downer cow syndrome.

in the pathogenesis of most of the ailments they

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encounter. More often than not, clinical disease is the

Habituation: extinction of a conditioned response by repetition of a cascade of environmental stressors: some of the conditioned stimulus; perceived threat diminishes as novelty wears off.

responsible for the allostatic load that has made the

Hypothalamic–pituitary–adrenal (HPA) axis: the hormonal

animal vulnerable to the proximate stressors (infectious, metabolic, toxic) that are the immediate causes of the ailment. If we wish to keep pace with current trends from the hypothalamus, adrenocorticotrophic hormone in intensive systems of animal production we will have (CRH) and arginine vasopressin (AVP; formerly ADH) (ACTH) from the pituitary gland and cortisol from the adrenal cortex. to take greater stock of ultimate stressors than has been

our wont. Increasingly, our task will be to devise and monitor herd health programmes that manage the hormonal linkage between the hypothalamus (releases GnRH), the pituitary gland (releases the gonadotrophins, FSH and LH) and the gonads (secrete testosterone, oestrogens, progesterone). sources of environmental stimuli to the mutual benefit of the livestock and the profit-and-loss account. In that context, it does not require great insight to realize that

curtailment of allostatic load is the key to improvement

Locus coeruleus: a small pigmented cluster of noradrenergic

neurones in the dorsal brain stem on the floor of the

in the overall health and productivity of the herd.

fourth ventricle. It contains more than half of the nora-

When the accumulated allostatic load attenuates the

drenergic neurones in the brain; they project widely

animal's ability to cope with further challenges, the host

throughout the CNS and they are highly responsive to

becomes vulnerable to all sorts of stressors, even those

alerting/stressful stimuli, including those that activate the

that are only mildly aversive. In that situation, a rela-

HPA axis.

tively minor proximate stressor may propel the host

Paracrine: hormone (or cytokine) acts on a different cell type into clinical disease, particularly one that reflects the

which lies in close proximity to the producer cells.

functional priorities of the current phase of the animal's

Proximate cause: an environmental factor that acts as

annual life cycle. For that reason, the differences in pre-

an immediate stimulus for a biological event (e.g. a

senting signs shown by herd mates should not obscure disease).

the fact that each sick animal has had a significant allo-

Sympathoadrenal (SA) axis: consists of two components: the sympathetic nervous system and the adrenal medulla. The static load and, in that sense, each clinical case should major biologically active products, the catecholamines be seen as a sentinel of stress within the herd.

(adrenaline and noradrenaline), serve both as neurotrans-

Allostatic load is not something that can be measured mitters and as hormones. Within the CNS they act as neuro- by standard biochemical or physiological techniques.

transmitters; outside the CNS, adrenaline acts principally as

Research scientists use hormone assays and heart rates

a hormone, while noradrenaline acts principally as a neuro-

to monitor responses to acute stressors, but those

transmitter at sympathetic post ganglionic terminals.

parameters are not indicative of allostatic load. The best

Ultimate cause: an environmental factor that increases

sources of the required information are comprehensive

the probability that a particular biological event will be

life-time records of health and production parameters.

elicited by another factor that is seen as the immediate

The onus is on the herd veterinarian to ensure that the

stimulus.

appropriate records are compiled, that they are used to

identify both ultimate and proximate stressors and

References

that the information so gained is taken into account

whenever therapeutic or prophylactic interventions are

Chrousos, G.P. (2000) The stress response and immune func-

contemplated.

tion: clinical implications. Annals of the New York Academy of Science, 917, 38–67.

Johnson, E.O., Kamilaris, T.C., Chrousos, G.P. & Gold, P.W.

Glossary

(1992) Mechanisms of stress: overview of hormonal and

behavioural homeostasis. Neuroscience and Biobehavioral

Arousal: the state of general alertness. It is reflected in

Reviews, 16, 115–30.

conscious state, in cognitive activity, and in behaviour.

McEwen, B.S. (1998) Protective and damaging effects of stress

Autocrine: hormone (or cytokine) acts on the cells that produce it.

mediators. New England Journal of Medicine, 338, 171–9.

McEwen, B.S. & Stellar, E. (1993) Stress and the individual: Brainstem: the stem-like region that links the cerebrum with

mechanisms leading to disease. Archives of Internal Medi-

the spinal cord; consists of the midbrain, pons and medulla

cine, 153, 2093–101.

oblongata.

Moberg, G.P. (2000) Biological response to stress: implications Cytokine: protein or glycoprotein (produced by one or more

for animal welfare. In The Biology of Animal Stress, pp.

cell types) that regulates the activity of other cells.

1–21. CABI Publishing, Wallingford.

Endocrine: hormone is secreted directly into blood stream

Royal, M.D., Darwash, A.O., Flint, A.P.F., Webb, R., Woolliams,

and acts on target cells elsewhere in the body.

J.A. & Lamming, G.E. (2000) Declining fertility in dairy

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cattle: changes in traditional and endocrine parameters of

Stratakis, C.A. & Chrousos, G.P. (1997) In Endocrinology: fertility. Animal Science, 70, 487–501.

Basic and Clinical Principles (ed. by P.M. Conn & S.

Sterling, P. & Eyer, J. (1988) *Allostasis: a new paradigm to Melmed*, p. 205.
Humana Press, Totowa, NJ.

explain arousal pathology. In *Handbook of Life Stress*,

Wade, G.N. & Schneider, J.E. (1992) *Metabolic fuels and*

Cognition, and Health (ed. by S. Fisher & H.S. Reason), reproduction in female
mammals. *Neuroscience and Biobehavioral Reviews*, 16, 235–72.

Behavioral Reviews, 16, 235–72.

Adult Cattle

Global Variation in Cattle Practice

Chapter 68

Diseases Related to Management in

Europe

G.H. Wentink and A. de Kruif

European dairy farming trends

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per hectare may be as high as or even higher than in

Infectious diseases

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The Netherlands and Belgium.

Metabolic diseases

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Total milk production in Europe is fixed by the quota system introduced in 1983. Placing a ceiling on milk production per country puts a limit on the number of

European dairy farming trends

dairy cows. As a result of fruitful breeding programmes and efficient feeding regimes the annual milk yield per Europe stretches from the 70th degree of latitude cow increased during the last decades. The number of (north of the polar circle) to the 40th degree of latitude dairy farms and dairy cows will be reduced even more into subtropical regions. Many different climatic conditions during the next decade, with an increase in the number of cows per farm.

differences work out in very variable disease problems The diets of dairy cows in The Netherlands and and different husbandry systems. In Denmark, northern Belgium contain substantial levels of concentrates. The Germany, The Netherlands, northern Belgium, north constituents of these concentrates are partly grown in west France and in Italy at the borders of the river Po,

Europe, but for the larger part, in particular protein, flat land with fertile pastures is located. These areas are they are imported into Europe. Nitrogen, phosphate suited for dairy farming. Annual milk production is high and other minerals are actually imported into these and may reach 8000 kg with 4 per cent fat corrected countries, but a limited amount of minerals is exported milk (The Netherlands). However, in Sweden, southern as milk and meat. Therefore, an imbalance exists Germany, southern Belgium, eastern France and between the amount of minerals imported into these Austria a hilly or mountainous landscape exists. These areas and the amount exported to other areas of areas are less suited for dairy cows, but more suited for Europe or to other parts of the world. The mineral suckler cows and beef production. Traditionally, in these surplus is applied into the ground as manure. This areas dairy herds with limited numbers of cows occur. causes an accumulation of elements (nitrogen) and puts Italy, Spain, Portugal and Greece have a rather hot a further stress on the reduction of the number of cows

climate during summer time; dairy farming is difficult on environmental grounds, especially in The Netherlands and of reduced extension. The average annual milk lands and Belgium.

production is 3000 kg (Greece), although exceptionally

During the last decades, the average education level dairy farms with a high production level occur (Portugal). In this chapter attention will be focused mainly on veterinarians must take another role. The veterinarian dairy cattle management in north west Europe.

will be called in to take their part in prevention of dis-

In 1998 just over 82 million cattle, including 21

eases and to sustain the milk production level on dairy

million dairy cows over 2 years old, were present in

farms rather than to cure sick animals. Therefore a shift

Europe. In Table 68.1 the number of cows and the agri-

in the veterinary skills is inevitable: the veterinarian has

cultural area per country are given. In The Netherlands

to support the management system of the client, mainly

and northern Belgium the ratio of cattle to agricultural

by doing the thinking for prevention of diseases and to area is more than two animals per hectare, in Ireland optimize production levels.

1.5, while the other countries have less than one cow In the past, much attention was paid to certain per hectare agricultural surface. This quotient applies to aspects of food quality, e.g. the cell count in milk, the agricultural area and gives an indication of animal but nowadays the consumer's attention is even more density in a country. In countries with a low quotient focused on food safety. Although the quality of prod-cows may be kept on a limited part of the agricultural ucts for human consumption increased during the last area and in some areas the actual number of animals decade, the confidence of the consumer in these prod-

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Table 68.1

Cattle population and agricultural area in Europe 15 (source FAO).

Country

Agricultural area ¥ 1 000 000 ha

Number of cattle ¥ 1000 Cattle/agricultural

ha

(1999 data)

(2000 data)

EU 15

143.018

82.332

0.58

Austria

3.419

2.150

0.63

Belgium-Luxembourg

1.521

3.085

2.03

Denmark

2.644

1.850

0.70

Finland

2.272

1.086

0.48

France

29.900

20.572

0.69

Germany

17.031

14.658

0.86

Greece

9.020

590

0.07

Ireland

4.418

6.708

1.52

Italy

16.268

7.184

0.44

Netherlands

1.967

4.200

2.14

Portugal

4.142

1.245

0.31

Spain

29.980

6.203

0.21

Sweden

3.235

1.713

0.53

UK

17.219

11.133

ucts continuously declined. The latter aspect is emphasized by BSE. The supposition that BSE prions might have caused the vCJD in man (see p. 909) and the possibility that prions entered the human food chain without appropriate check on the source of the animals

Infectious diseases

for food production caused a shift in public opinion. The threat of disease caused by products of animal origin and the threat of antibiotic resistance in human diseases have been eradicated, e.g. rinderpest, BCPP, bacteria due to the application of antibiotics in food tuberculosis, brucellosis and EBL. In 2001, FMD re-animals increase the attention paid by the consumer to emerged in north west Europe and caused severe (UK), the chain in which human food is produced. The chain moderate (Netherlands) or mild (France) epizootics, must be completely transparent and the methods to

focusing attention on this disease again. Until this outbreak the processes must be supervised by independent people.

of BSE, IBR, BVD and on a limited scale of paratuberculosis (Johne's disease) (see p. 857).

Furthermore, increasing attention is paid by the

public to animal welfare. The consumer demands food

Veterinarians must be fully aware of the agents and

from animals that are kept under conditions that allow

their epizootiology. The Achilles heel in the infection

them to function more or less naturally. Reducing

chain must be found for each individual pathogen to

the frequency of microtraumata by comfortable barns

develop effective eradication strategies.

beyond doubt increases the durability of dairy cows. In

the future veterinarians may participate in the supervision of the production of food from cows by paying

attention to their comfort.

FMD (see Chapter 43c)

As long as FMD exists somewhere in the world, the

Knowledge of diseases, and especially knowledge of risk of introduction of the virus into Western Europe their cause and aetiology, the consequent knowledge to by food products from infected animals remains real. prevent problems and the skills to prevent a decline Intensive traffic movements of people over the world of milk production and disasters on dairy farms must increase the risk of accidental introduction of contam- be an essential characteristic of veterinarians in the inated food products. When the FMD virus is next decade. Veterinarians may also contribute to the introduced into a West European country, the risk of achievements of dairy farmers by increasing the quo- an epizootic depends on the time lapse between intro- tient milk production versus manure per surface unit. duction of the virus and the diagnosis, and of the Two aspects for veterinary prevention programmes number of animal movements over the country or to must be considered: prevention of the introduction of other countries during that period (see p. 705).

IBR (see p. 289)

in life. In the last phases of the disease bacteraemia develops and as a consequence milk and meat might be

In the 1980s, voluntary eradication programmes infected with the bacterium.

for IBR were started in the Scandinavian countries.

In man, Crohn's disease has a pathology that is very

*However, there was no thorough economic basis for the similar to paratuberculosis in the bovine. *M. avium* ssp.*

eradication of IBR. IBR is also not a threat for human paratuberculosis was isolated from the intestines of

health, the economic damage is limited and clinical

patients with Crohn's disease, but not from the

disease can be prevented by an appropriate vaccination

intestines of humans suffering from other intestinal

programme. However, as Scandinavian countries

*disorders. The PCR for *M. avium* sp. paratuberculosis became officially free of IBR several other European*

is significantly more often positive in patients suffering

countries were forced to achieve IBR negativity as well.

from Crohn's disease than in those suffering from

It was mainly a political decision to eradicate IBR.

other gastrointestinal disorders. This does not prove,

Eradication programmes were successful in Switzer-

however, that M. avium ssp. paratuberculosis is the land, Denmark, Sweden,
Finland and Austria. The basis

causative organism for Crohn's disease. The agent

for the success of these programmes was that all sero-

might take advantage of the pathological changes in

logically positive cows, which might be latently infected,

the intestines of human patients with Crohn's disease

were eliminated. The percentages of animals found

to colonize the bowel. It demonstrates, however, that

serologically positive for IBR in these countries did not

the agent must be present in the human environment

exceed 20 per cent of the dairy cows.

and probably in human food. Actually, about 6 per cent

However, percentages of serologically positive

of milk for human consumption in the UK was

animals turned out to be as high as 50–60 per cent in

contaminated with M. avium ssp. paratuberculosis, The Netherlands and
Belgium, and therefore the above

although it is unclear whether the bacteria in milk are

mentioned method was not applicable. Vaccination viable.

programmes using gene deleted vaccine virus, but with

*It is, however, important to ensure that the human
a serological response distinguishable from field virus
food chain is free from this bacterium and veterinari-
infections, are applied to all cattle herds that are not
ans are obliged to pay attention to the eradication
proven negative for BHV1. These programmes started
of the agent from the cattle population. On the other
recently (1998), so there is no evidence that these
hand, efforts to eradicate paratuberculosis (Johne's
schedules will turn out to be completely successful.
disease) in all countries are limited.*

BVD (see p. 853)

BSE (see p. 909)

*Bovine virus diarrhoea virus (BVDV) has a real
Bovine spongiform encephalitis prions are suspected
economic impact on infected farms and the disease
to be the cause of the [new] variant Creutzfeldt Jacob
deserves full attention for eradication. Although some*

Disease ([n]vCJD). These prions are present in tissues
Scandinavian countries have voluntary eradication pro-
from the central nervous system and jejunum of
grammes, the efforts to eradicate this virus are far less
cattle with overt clinical signs of the disease. Cows are
intensive than the efforts to eradicate IBR. In Sweden
believed to contract the disease by consuming ruminant
and in Denmark voluntary programmes for the eradi-
proteins produced from infected ruminants. The major
cation of BVDV have been in progress since 1993. The
breakthrough was the total elimination of animal
backbone for the eradication programme is the detec-
proteins (except fish and poultry proteins) from the
tion and destruction of all persistently infected animals.
ruminant food chain. The consumer requires a guar-
A herd has to start with a virulological search for the
antee that animals for human consumption have never
presence of persistently infected animals. After the
consumed animal protein and puts emphasis on the
elimination of these animals a regular serological
source of the meat and its control chain.

survey of the animals between 6 and 12 months is performed, and later on by testing for the absence of antibodies against BVDV in bulk milk samples.

Metabolic diseases (see Chapter 46)

The increased capacity for milk production necessitated

Paratuberculosis (Johne's disease) (see p. 857)

improved feeding regimes. The quality of grass silage

Johne's disease is caused by *Mycobacterium avium*

and corn silage increased and facilitated higher pro-

paratuberculosis. After infection by the oral route

duction levels. Next to roughage a substantial amount

during the first months of life, clinical disease charac-

of concentrates are fed. Concentrates are added in the

terized by diarrhoea and emaciation may develop later

milking parlour or in concentrate stations, where each

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Table 68.2

Prevalence of disease problems in 8272 cow years

(roughage). In experimental farms in The Netherlands

in nine dairy farms in The Netherlands during a 10-year period.

with a management above average the incidence of this

disease was limited to 1 per cent of the cows.

Fertility problems

34.9%

A disease biochemically very similar to acetonaemia

including 19.0% irregular heat periods

may develop within two weeks after parturition. In the

Udder problems

24.6%

peripheral blood high levels of non-esterified fatty acids

including 22.5% mastitis cases

(NEFAs), high levels of b-hydroxybutyrate and low

Locomotory

36.7%

levels of glucose are found, indicating severe energy

problems

including 6.8% laminitis

including 17.7% solar ulcers

deficit, but with very high levels of triacylglycerol in the

Metabolic problems

19.7%

liver. This phenomenon is known as fatty liver, a

including 5.5% acetonaemia

very peculiar phenomenon in that the peripheral body

including 14.4% milk fever

is deficient in energy supply, while in the liver high

levels of energy (up to 25 per cent of triacylglycerol of

wet liver tissue) are available (see p. 801). The cow is ill

and may die from energy depletion while abundant

individual cow can obtain an amount relative to milk

energy is present but is not made available for the

production, or a total mixed ration is provided.

metabolism. The origin of the disease is considered to

Indications for a causal relationship between food,

be a sequel of excess of energy supplementation in the

feeding and diseases are increasing and substantiated

last part of the lactation period and during the dry

by experimental work. Metabolic disturbances also

period. About 10 to 12 weeks after parturition, the

significantly influence the health of claws and may

initial decrease in body weight due to the physiological

exacerbate the consequences of external forces (mini-

negative energy balance turns and becomes positive.

trauma) on claws, causing lameness (see Table 68.2).

The body weight increases until drying off.

Veterinarians should be fully aware of the relation-

*During the dry period the body weight must be kept
ships between food, metabolism and disease, and must*

constant. However, the increasing energy value of the

be able to translate food changes into possible devia-

roughage produces an energy surplus during the dry

tions of the health of the animals in a herd. Thus the

period and as a result many cows increase their body

veterinarian involved in cattle herd health management

weight and are fat at calving. Although exact mecha-

must be familiar with food constituents and compo-

nisms are poorly understood, many cows in a fat body

nents and their digestion and metabolism.

condition at calving (BCS 4 or higher on a 5 point scale)

Large quantities of rapidly fermentable starches in

suffer from a decreased appetite after calving. This

the diet may cause disturbances of rumen fermentation,

reduced appetite may be related to or caused by leptins,

resulting in low milk fat percentages, off feed syndrome,

by high levels of NEFAs, by a combination of these laminitis and ruminal acidosis consecutively, depending factors or by other not yet described causes. In these on the grade and the period of acidosis. These phe-fat animals with often high milk production the energy nomena are mainly observed in herds with high milk intake is short in comparison to the energy demand. production levels being fed high amounts of concen- Increased lipolysis, in order to compensate for energy trates in the diet. Although there are no exact data demand, results in the accumulation of triacylglycerol on the incidence of these diseases in Europe, in The in the liver, i.e. fatty liver. In the short term this Netherlands and Belgium these disturbances have metabolic disease may result in emaciation and death; occurred rather frequently in the past, but are now in the long term it may cause reduced reactivity of the decreasing due to the lower percentage of easily immune system, making the cow more vulnerable to fermentable starches in the concentrates. common bacterial infections. There may be a high

If the energy in food is maintained in accordance prevalence of mastitis in the post partum period in with the milk production level, cows lose body weight animals suffering from fatty liver. Ova mature during moderately during the first few weeks of lactation. The an eight-week period and the first ovulation after negative energy balance during the first weeks after parturition in these cows produces an ovum matured parturition is physiological (energy intake by food and during the period with high levels of NEFAs in the energy from reserve fat equals energy demand). If this blood. These ova are less fertile and may result in equilibrium is disturbed by low energy content of the lowered conception rates. This metabolic disturbance food acetonaemia some three weeks after parturition causes decreased ovarian activity with prolonged develops, characterized by high levels of b-hydroxybutyrate intervals from calving to heat, resulting in prolonged tyrate and low glucose levels in the peripheral blood. intervals from calving to conception. The incidence of This problem is still known but occurs less frequently

fatty liver is not known, but it is suspected to occur than previously due to increased quality of the diet more frequently than anticipated.

Diseases Related to Management in Europe • 1155

Dietary deficiencies are rarely diagnosed in dairy in management which result in reduced fertility, herds in areas with a high milk production. The diet reduced immunoreactivity or in lameness .

receives adequate levels of minerals and vitamins from the addition of concentrates, the composition of which

Further reading

is monitored by the manufacturer. However, as many farms start to feed home-produced diets composed

FAOSTAT Database Collections; <http://apps.fao.org/>

from basic substances there is a risk of miscalculation

Smolders, G. In Annual Reports of the Research Station for and thus excesses or deficiencies may occur. The

Cattle-, Sheep-, and Horsehusbandry 1990-1999, Institute veterinarian will be asked to support management

edn. Research Institute for Animal Husbandry, Lelystad,

decisions taken by the owner and to diagnose failures

The Netherlands.

Chapter 69

Cattle Disease in Africa

R.S. Windsor

Introduction

1156

868), foot-and-mouth disease (p. 700), rabies (pp.

Epizootic diseases

1156

908, 1164), tuberculosis (p. 862) and brucellosis (p.

Rinderpest

1156

280).

Foot-and-mouth disease

1157

- *Parasitic diseases caused by either by helminths, e.g.*

Tuberculosis and brucellosis

1157

Haemonchus contortus (p. 275), or *Fasciola gigan-*

Parasitic diseases

1157

tica (p. 276), or by tick-borne protozoa, e.g. *Theile-Trypanosomosis*

1159

ria parva (p. 750) or *Anaplasma marginale* (p. 761), East Coast fever

1160

Plant poisonings

1161

or by the biting flies (p. 747) (Glossinia spp.), or the various species of Trypanosoma (p. 756).

- *Plant poisonings caused by the varied and extensive **Introduction***

flora of Africa. Among such a profusion only a few examples can be given.

The purpose of this chapter is to indicate some of the differences between cattle problems in Africa and the rest of the world and to highlight some of the condi-

Epizootic diseases

tions that are of major importance to livestock production. North Africa is part of the Mediterranean area and

Rinderpest (see p. 707)

it is separated from the rest of Africa by the Sahara

First among cattle diseases is rinderpest, the greatest

Desert, where there are few cattle. SubSaharan Africa

scourge of cattle, and indeed their owners, that the world

has a variety of climatic conditions, from almost no rain-
has ever seen. When rinderpest was imported into
fall to heavy, regular and seasonal rainfall areas, and in
Britain in the mid nineteenth century it ruined farmers,
consequence there is a variety of environmental types
bankrupted insurance companies and was directly
and eco-systems. This in turn has led to many different
responsible for the foundation of what is today the State
lifestyles from the sedentary agriculturist to the
Veterinary Service. The Royal Agricultural Society
nomadic pastoralist who moves his herds as conditions
considered that rinderpest was responsible for the death
dictate, e.g. the Fulani in west Africa would prefer to
of more than 400 000 head of cattle in Britain in six
graze their animals in the south where there is more
months of 1866 at a cost in 1866 figures of £8 million. It
rain and the grass is better for the cattle. However,
was introduced into Egypt in 1890 and in five years it had
during the rainy season the tsetse flies are active, so the
travelled to South Africa, killing not just cattle but also
cattle are moved north to escape their bites. When the

huge numbers of game animals as it passed.

dry weather returns and the pastures in the north

Following Macmillan's famous 'winds of change'

become depleted of grass they return their animals

speech to the South African parliament in 1960, African

south. In large measure the distribution of the human

countries raced towards independence. The cold war

population has been governed by this fly (Glossinia

was beginning to push America and Russia: both wished

spp.). To many African people, the well-being of their

to develop spheres of influence in Africa and so they

animals governs their lifestyle. The differences in the

poured money into the continent. Joint Project 15 was

ecology of the continent govern the behaviour of

set up, with almost limitless funding, to eradicate rinder-

disease agents, parasites and vectors: therefore, cattle

pest from Africa. A vaccination campaign, using the

diseases to be considered here fall into three main

Muguga tissue culture vaccine, almost succeeded in

categories:

eradicating the infection. Unfortunately pockets of

• *Epizootic diseases that have mostly been introduced into the continent from Europe – rinderpest disease ‘recolonized’ Africa. The Pan African Rinderpest Campaign (PARC) was set up with the aim of eradicating rinderpest within a period of 10 years. PARC has parasites. Unlike the Amazon Jungle the tropical forests of Africa are not so large as to make huge tracts uninhabitable. Africa has probably more species of tick than any other continent and because of the extensive savannah areas these creatures have had plenty of hosts and time, so that a reasonable equilibrium may become a reality.*

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Cattle Disease in Africa • 1157

indicating rinderpest within a period of 10 years. PARC has parasites. Unlike the Amazon Jungle the tropical forests of Africa are not so large as to make huge tracts uninhabitable. Africa has probably more species of tick than any other continent and because of the extensive savannah areas these creatures have had plenty of hosts and time, so that a reasonable equilibrium may become a reality.

rium has built up between animals (including indigenous domestic cattle), ticks and the parasites that they carry. This equilibrium was destroyed when the

Foot-and-mouth disease (see p. 700)

Europeans imported exotic cattle in an attempt to

In Africa, foot-and-mouth disease (FMD) is more increase productivity of the native cattle. For some important for its political impact than for the disease unknown reason cattle, and indeed humans, have failed that it actually causes. It is endemic throughout much to build up resistance to the parasites carried by the of the continent and the African buffalo (*Syncerus*

tsetse flies (*Glossinia* species) (see p. 747) – the try-caffer) has been shown to be a carrier of the SAT

panosome (*Trypanosoma* species) (see p. 756). In large strains. This makes the eradication of the virus from the

measure it has been the trypanosome that has deter-

continent problematical. Control measures follow

mined the distribution of cattle in Africa and conse-

the normal international pattern, with the exception of

quently it has played a part in the distribution of the

Botswana, which has developed its own technique of

human population. Tsetse fly are recorded in 37 African control. The country is divided by cordon fences (some countries and more than 10 million square kilometres hundreds of kilometres in length) into different areas of land are infested. There are few breeds of cattle that of 'infected', 'clean but vaccinated' (buffer) and 'unvaccinated' areas. Movement of cattle from one area to mostly confined to west Africa.

another is strictly controlled, including physical examination of every animal by the Veterinary Department.

The reason for the difference in resistance may well be associated with the feeding habits of the parasites.

At each biannual vaccination the animal is branded and Hard ticks (and these are the only ones of importance only branded animals are allowed into the abattoirs. As to cattle) are sedentary and remain on the host, or at a result of this control, Botswana is permitted to export least each stage of the parasite remains on the hosts and meat into the European Union from the 'unvaccinated' feeds by sucking blood. There are three types of hard

areas.

tick: one-, two- and three-host ticks, depending upon

For many years Kenya was allowed to export meat

whether or not at the completion of each stage of life

into the European Economic Community. Movement

the tick remains on the host to moult or whether it

controls broke down, the disease spread and Kenya is

drops off. A one-host tick spends its life on one animal:

no longer able to export its meat to Europe. Other coun-

the larvae moult to nymphs, the nymphs to adults on

tries with no export trade outside the continent make

the same animal. The gravid female drops off to lay her

little or no official attempts to control the infection.

eggs on the ground. The two-host tick usually moults

from larva to nymph on one animal but the nymph

drops and moults to an adult on the pasture. The three-

Tuberculosis and brucellosis (see pp. 280, 862) host tick drops off at each moult. It is obvious that

Bovine tuberculosis in Africa is complicated by the

three-host ticks are more efficient at spreading infec-

presence of a large number of 'cold blooded' or atypi-

tions than the one-host variety. By contrast, the tsetse
cal strains of *Mycobacterium*. These make the interprefly takes each meal where
it finds it and moves from

tation of the tuberculin test more difficult and today
animal to animal: there is no time for a host–parasite
outside South Africa there is little done to control the
interaction and the parasite moving from host to
disease at the official level. In most countries with a
host has accustomed itself to varying host immune
commercial dairying industry (e.g. Kenya, Ivory Coast)
mechanisms.

the industry itself tries to ensure freedom from both
Each species and genus of tick has a different distri-
tuberculosis and brucellosis, thus enabling producers to
bution, a different life cycle and differing host prefer-
market ‘attested’ milk.

ences and site predilections. Specific ticks transmit
specific infections: e.g. *Rhipicephalus appendiculatus*
(the brown ear tick) transmits East Coast fever (ECF)

Parasitic diseases

(theileriosis) (p. 750) and other theilerial parasites,

whereas *Boophilus decoloratus* (the African blue tick)

The African continent has the climate, soils and hence

transmits *Babesia bigemina* (the cause of bovine

ecology to support a great diversity of animals and their

babesiosis) (p. 748) and *Anaplasma marginale* (the

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cause of anaplasmosis) (p. 761). By the same token,

tions in order to reduce the dipping and work towards

there are different species of *Glossinia* that inhabit dif-endemic stability; the second is to try and control the

ferent ecological niches and that transmit different

ticks by intensive dipping.

species of *Trypanosoma*. The demonstration by Bruce,

Over the years there has been a different approach

at the end of the 19th century, that it was the tsetse fly

to the control of tsetse flies. Perhaps the greatest

that transmitted pathogenic trypanosomes to livestock

natural control of the fly was the epidemic of rinderpest

led the way to understanding the role of arthropod

that started in Egypt and moved south at the end of

vectors in animal disease. The tsetse fly has been a

the nineteenth century. Not only were cattle killed, but major impediment to the development of a large part there was a massive destruction of game animals and of the continent and environmentalists would say that hence a lack of food for the tsetse flies: e.g. rinderpest this has been a good thing! With the rapidly increasing eliminated the tsetse fly from the Limpopo valley population in Africa and the need for more food it is between Botswana and South Africa and in almost 100 essential that tsetse flies and ticks be controlled.

years the valley has not been recolonized. Human activ-

It has been known for many years that some animals ity has an effect:

in a herd are more resistant to a specific disease than

- Clearing forests for timber or settlement destroys*

others, and some more susceptible. Breeders have used

the habitat of the fusca group of flies (G. brevipalpis these characteristics and, as mentioned above, trypan-in particular).

otolerant breeds of cattle are found in west Africa. It

- Clearing the savanna has made the greatest impact*

has been demonstrated recently that some cattle

on the morsitans group (*G. morsitans* and *G. pallidipes*). These primarily feed on game animals increased resistance is associated with immune mechanisms. It has been said that the trypanotolerant Ndama wildlife. At levels of human habitation in excess of 40 per square kilometre, the morsitans group dies out completely.

nism by which this occurs has not yet been demonstrated. Resistance to *Babesia bigemina* (p. 748) has human activity, as these are the flies of the river-bank, and it is rare for human activity to clear the tick, than to the parasite itself. Resistance to tick infestation was first demonstrated in Australia. It was also cleared land but pass through the riverine bush to

- The palpalis group may be beneficially affected by

*noted in Africa that indigenous breeds kept with exotic
get to the river. The establishment of game parks
animals often showed much lower (down to 1/6) tick
has provided breeding grounds for tsetse flies and
burdens. It has been possible to breed cattle with
food for them to eat!*

*increased resistance to the one-host tick, but so far not
to the multi-host ticks.*

*Pesticides have been used to control the flies, by knap-
It is unlikely that tick resistance can be developed to
sack spraying the area of the flies' resting sites.*

*the point where there is no transmission of the proto-
Organochlorine compounds such as DDT and dieldrin
zoal diseases. Consequently a combination of resistance
were widely used and effective, with synthetic
with acaricide treatment will probably always be
pyrethroids taking over when the organochlorines were
required. Before deciding upon a programme of tick
banned. A more efficient form of spraying was from the
control by acaricides it is essential to know which ticks
air. Today with the rotary atomizers and night flying*

are to be controlled, and when. For many years Malawi equipment, the spraying is much more efficient and in had a policy of strategic dipping of cattle: in the north the Okavango Swamp in Botswana, the use of endo- of the country it was confined to the wet season when sulphamonomethoxime has reduced the environmental contamination the number of ticks increased dramatically. In the dry – only 8 g per square kilometre are used and the drug winter the tick burden was at a minimum and so the targets the tsetse fly rather than other insects; so far as gain was less than the cost. In areas of Africa where is known, the only detrimental environmental effect ECF is not a problem it is often not necessary to that it has is to inhibit fish reproduction for a season. attempt acaricide dipping or spraying of cattle. In areas Tsetse flies can be caught in traps and although this of Africa where ECF has been introduced, there may was not successful at eliminating flies from an area, be a mortality rate approaching 100 per cent, and then work with traps led to the discovery that the flies are it may be necessary to use chemicals to keep the tick

attracted by the colour blue. The use of odour to attract burden to a minimum. In the absence of ECF, there are the flies only had limited success, but the development two options: the first is to control the remaining tick-of targets impregnated with insecticides has had a striking success. As the fly lands on the blue cloth it receives ensure that the young animals are exposed to the infection a dose of insecticide.

Cattle Disease in Africa • 1159

Trypanosomosis

as the nutritional state of the host. Tissue lesions vary with the species: *T. vivax* and *T. congolense* are mainly This disease occurs in humans, cattle (where it is known intravascular parasites inducing changes in the as nagana), camels, horses and pigs, but the infection endothelium of the capillaries, whereas *T. brucei* has occurs in most wild mammals. Trypanosomes are pro-an affinity for tissues. The anaemia seen in trypanoso-tozoa that possess a kinetoplast and a flagellum and mosis is caused by a multiplicity of factors including require two hosts to complete their life cycle: a mammal

haemolysins and enzymes such as proteinases or phospholipases. The trypanosome attaching to the erythrocyte makes it a target for the body defence mechanisms. Trypanosomes multiply in the mammalian host and, with the exception of *T. equiperdum* which is venereally transmitted, they are ingested by blood sucking insects: more severe disease.

In Africa normally by tsetse flies. Multiplication within the fly varies with the *Trypanosoma* species, but the infective stage ends up either in the salivary gland or in

T. vivax and *T. congolense*; *T. brucei* causes much less the faeces. Other biting flies are able mechanically to

transmit the parasite to a new host. However, there are no specific signs for

trypanosomosis and they are dependent upon the

There are three major species that infect cattle: T. degree of damage to specific organs and the severity of brucei (p. 759), T. congolense (p. 758) and T. vivax (p. the anaemia. Acute disease with death within two to six 758). The last has become established in South America weeks is seen in animals introduced into an endemic where there are no tsetse flies and so transmission is by area, but chronic disease with the animal surviving means of other haematogenous flies, and it is assumed months or even years is more common. Acutely that no development of the parasite occurs in these flies. affected animals rapidly lose weight and all the classic Trypanosomes multiply by longitudinal binary fission signs of fever may be seen. In the chronic form the in the fly and the mammalian host, although sexual animals show periods of weakness and lethargy fol- processes occur only in the fly. Mature trypanosomes lowed by a recovery; the animal becomes pale with are ingested by feeding flies and they undergo devel- raised pulse and respiratory rates. Good nutrition may opment in the salivary gland or gut. Development and

help an animal to throw off this chronic infection, but maturation occur in the vector, after which the parasite the more normal outcome is for the animal to become is transferred to another mammalian host. Transmission thinner and thinner. The infection induces immunosuppression, which can allow other infections such as saliva or by contamination of the host's skin by trypanosomiasis to take over.

panosomes in the faeces of the fly.

It can be very difficult to diagnose this disease

A critical factor in the epidemiology of trypanosomiasis is that, once infected, a tsetse fly remains infected blood and there are no specific clinical signs. Moreover for life. The development of the parasite in the fly varies there is no relationship between the number of parasites

from a few days in the case of *T. vivax* up to 45 days in and the severity of the disease. Trypanosomiasis must be

the case of *T. brucei*. The role of game animals cannot considered to be a herd problem and so the presence of

be overemphasized; the present policy of game ranch-

parasites in the blood of a 'normal' animal should be
ing in conjunction with rearing cattle and the increas-
taken as significant. In many endemic areas there are
ing numbers of game parks has increased the risk of
inadequate laboratory services to assist in making the
disease in cattle. Another significant factor in the epi-
diagnosis and so many veterinarians consider that
demiology of the disease is the use of trypanocidal
response to treatment is a good way to confirm the diag-
drugs, which may in turn lead to the development of
nosis. Serological tests have until recently suffered from
resistant trypanosomes.

an inability to produce specific antigens. However, the
The pathogenesis is not completely understood: the
indirect fluorescent antibody test and the enzyme-
trypanosome multiplies at the site of infection and local
linked immunosorbent assay have recently been intro-
lymph nodes become enlarged, before the parasite is
duced into the arsenal of tests used for making a herd
found in the blood. Then the classic intermittent para-
diagnosis, and monoclonal antibodies have been pro-

sitaemia ensues. As the parasites increase in number, so
duced to identify the three bovine species of try-
does the febrile response: as they decrease, so does the
panosome. The main problems in the differential
fever. The body mounts an immunological defence
diagnosis are with the other protozoan infections, i.e.
against the parasite and the trypanosome changes its
anaplasmosis (p. 761), babesiosis (p. 748) and East Coast
external antigens to defeat the host. The outcome of
fever (p. 750), however, in these diseases it is usually a
this battle often depends upon extraneous factors such
simple procedure to demonstrate the parasite.

1160 • Chapter 69

The control of the infection by the use of drugs pres-
As with trypanosomes, so *T. parva* alternates
ents a continuing problem and the Veterinary Depart-
between its mammalian and invertebrate hosts. The
ments in many African countries control the use of
piroplasms in the erythrocytes of an infected bovine are
them to prevent the development of resistant strains of
ingested by the tick in its blood meal and these develop

trypanosomes. There is more to treatment than drugs via a sexual stage to a stage that invades the gut cells of and a good plan of nutrition is essential; all animals, and the tick. Further development results in a motile stage particularly work oxen, must be rested during convalescence. One of the major problems facing trypanosomosis control is that today there are only four drugs produced; these are the infective stage for the effective in treating the condition, the most recent of bovine, being injected when the tick feeds. Because which (isometamidium) was introduced in 1961; moreover, the vector is a three-host tick and because there is no over, no pharmaceutical company is working on this transovarial transmission the infection is transmitted by problem. Care must be exercised in using a combination of drugs because some of the drugs have a combined toxicity greater than that of the individual drugs.

there will be no transmission from the larva to the adult.

Because of the problems of drug resistance it is essen-

The disease affects only cattle, the African buffalo

tial that optimum use must be made of them, that is to

(Syncerus caffer) and the Asian water buffalo (Bubalis say they should be used in a strategic way whenever

bubalis). No other ungulates are infected and the par-possible to coincide with the local rainfall and hence

asite depends upon Rhipicephalus appendiculatus for

fly patterns. However, the most important factor is to

transmission. This tick is widespread in the warm humid

reduce the fly population and hence the parasite chal-

areas of Africa, and were the parasite to be introduced

lence. The Zimbabwe Veterinary Department has made

to these areas the infection would be rapidly dissemi-

great strides in vector control going back over a period

nated. The peak incidence of the disease is just after the

of more than 50 years. This is well described by Connor

rainy season. Zebu cattle which carry low burdens of

(1994). Many countries in Africa have units within their

ticks rarely show clinical disease: they are infected as

Veterinary Departments (e.g. Botswana, Kenya, Ghana

calves and may show a febrile reaction with complete and Zimbabwe) devoted to tsetse fly control. However, recovery; mortality is low. However, if susceptible Basil Parsons, the Kenyan parasitologist, was of the animals are introduced into this endemic area and opinion that 'when the last man leaves the African continent, he will be bitten on the backside by a tsetse fly'.

The disease can be introduced into a clean area if an infected animal is brought in or if conditions are such that an area can be colonized by the tick. In the **East Coast fever** (see p. 750)

absence of control measures, the mortality can reach 90 In 1965, the owner of a 'high grade' (mostly Jersey cross per cent.

indigenous Kenyan cattle) herd of cattle went on

The disease is primarily one of the lymphatic tissue.

holiday leaving his manager in charge. Because they

The sporozoite enters lymph cells at the place of entry

were 'high grade' they had to be dipped in acaricide

and within a few days schizonts can be seen in the cyto-

every five days to prevent ticks introducing *Theileria* plasm of the cells. Lymphocyte proliferation occurs at parva parva (*T. parva*). Unfortunately the manager the site and within five days infected cells can be found allowed the fences to be cut and the neighbour's indige- in the drainage lymph node. Massive proliferation of nous cattle were able to mix. The farmer returned to lymphoblasts ensues and more distant lymph nodes, find 75 of his best heifers dead from East Coast fever spleen and thymus become affected. Finally all the (ECF). The infection is transmitted by the three-host parenchymatous organs become involved. Within a few tick *Rhipicephalus appendiculatus* and the disease is days lymphocyte proliferation is replaced by lympho- characterized by the multiplication of the lymphoblasts cyte destruction, resulting in immunosuppression. The infected with theilerial schizonts throughout the body. outcome of the disease depends upon the survival of The disease was first recognized in 1897 by Robert sufficient lymphocytes to effect an adequate immune Koch, who called it African Coast fever. Following the

response. Release of piroplasms which then invade the
ravages of rinderpest and the introduction of European
erythrocytes appears to have no specific pathogenic
stock in an effort to replace the dead animals, ECF
effects.

spread south from East Africa down to Cape Province
East Coast fever is characterized by fever with all its
in South Africa, killing more than one and a quarter
complications – lethargy, constipation or diarrhoea, res-
million cattle. The disease was eradicated by a process
piratory signs and abortion, together with enlargement
of dipping in acaricide, movement controls and destock-
of the superficial lymph nodes. Auscultation of the lungs
ing of infected pastures.

indicates severe pulmonary oedema. Because the ticks

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live in the ears it is often the parotid lymph nodes that
in different places and so the famous ‘Muguga cocktail’
first show enlargement. Death occurs from five days to
was developed. This in turn has its problems, in that
three weeks after the onset. Exotic animals that recover

Directors of Veterinary Services are not happy to use often become emaciated and can remain unproductive live, virulent strains of Theileria parva from foreign for months.

countries and this means that it is not possible to

On post-mortem examination the three classic find-

produce the vaccine on a large, regional scale. The

ings are severe lymph node enlargement, a massive

vaccine is expensive and not without its problems and

pulmonary oedema and splenomegaly. Congestion

so it is only used by commercial farmers. Work to

and oedema can be found in any of the internal organs.

improve the vaccine using molecular techniques is in

Numerous small white foci may be present in the

progress.

kidneys, indicating lymphoid hyperplasia in the cortex.

Because of the behaviour of the tick and the parasite

There may be emaciation and subcutaneous oedema.

it is possible to control the infection by isolation of sus-

Diagnosis is confirmed by the examination of smears

ceptible cattle, but they must not be fed purchased hay

stained with Giemsa stain, of lymph node, spleen, blood

which might contain ticks. Tick control can keep ECF or almost any organ. The presence of large lymphocytes at bay providing that mistakes are not made in dipping phoblasts is strongly suspicious: the presence of strength or dipping interval. Leaving the pasture free schizonts is conclusive.

from cattle for 18 months or more will 'sterilize' the pasture because the parasite cannot survive that long: In some cases it is possible to observe lymphocytes containing schizonts in blood smears. It may not be possible to demonstrate schizonts in zebu cattle and so the ticks will feed on other animals, but there will be no transmission of the parasite and so it will die out. The demonstration of rising titres of antibodies should be made, using the immunofluorescence techniques. control of cattle movement is also a good way to halt the spread of the parasite. Because ECF control Among the conditions to be considered in the differential diagnosis are rinderpest (p. 707), bovine virus tries have employed communal dipping as a means of

diarrhoea (p. 853), contagious bovine pleuropneumonia bringing the cost within the grasp of the smallholder. To (Chapter 49(b)) and trypanosomosis (p. 750); the pre-be effective, control measures for ECF need to be long ence of the schizonts is conclusive. At the Veterinary term, take into account the whole management of the Research Laboratory, Kabete, Kenya, in the mid 1960s, herd and the farmer must be eternally vigilant.

each morning the staff were confronted by a line of men

This account has concentrated on the disease caused with bicycles, in the baskets of which were a selection

by *Theileria parva parva*; however, this is not the only of viscera from a dead animal. The farmers were only

theilerial disease. Corridor disease caused by *Theileria*

interested to know whether or not the animal had died

parva lawrencei was first seen in Zimbabwe and is an

from anthrax (if not, then they could eat the meat!);

infection transmitted by ticks from the African buffalo.

most animals had died from ECF.

It is indistinguishable from ECF. Zimbabwe theileriosis,

Although tetracyclines have some effect against *T.*

caused by *Theileria parva bovis*, is also clinically indis-*parva* they are only consistently effective during the tinguishable from ECF, but is treatable with tetracy-incubation period. The development of parvaquone,

clines and shows differences in its epidemiology. The

napthoquinone and febrifugene has resulted in success

cause of 'turning sickness' (p. 752) is a theilerial para-

rates for treatment of over 90 per cent. However, some

site but its identity is in doubt. The disease is manifested

animals may remain carriers following treatment.

by nervous signs and infected lymphocytes can be found

Because immunity to the natural disease persists for

in the brain. None of these conditions has the economic

more than five years, there has been a massive search

importance of ECF.

for a vaccine and millions of pounds have been invested

in this work at the Veterinary Research Laboratory,

Kabete, the Kenya Agricultural Research Institute (for-

Plant poisonings

merly East African Veterinary Research Organisation),

Muguga, and latterly at the International Laboratory

The foreword to *Plant Poisonings and Mycotoxicoses of*

for Research on Animal Diseases (now ILRE), all in

Livestock of Southern Africa (Kellerman et al., 1990) Kenya. The vaccine consists of injecting a controlled

says it all!

number of sporozoites obtained from schizonts, originally from infected ticks but now from tissue culture,

‘Southern Africa is well known for the diversity and and then following this with treatment; initially a tetracycline was used but today the newer drugs have taken unexpected that the sub-continent should be “blessed” its place. Because the vaccine is only effective against with a possibly unequalled variety of poisonous plants homologous strains different preparations must be used and toxic fungi.’

1162 • Chapter 69

‘About 600 indigenous poisonous plants are known to Massive losses occur from this plant; in the Maun occur.’

District of Botswana in 1984 one farmer lost 34 animals

‘In this part of the world, where livestock are traditionally (10 per cent of his herd) and more than 700 cattle

tionally kept under extensive conditions on veld that is deaths were reported in the District. At the same time frequently denuded by droughts, overstocking and in the Chobe Game Park more than 70 buffalo were uncontrolled fires, the animals are often forced to eat found dead by the side of the Chobe River. If the poisonous plants which they would normally avoid. farmer sees his animals eating mogau he can save Devastating outbreaks have been reported under such them by restricting their access to water for 24 hours. conditions, for instance, during 1926–27 about 600,000 Because of its nature *Dichapetulum cymosum* is even sheep died of plant-induced photo-sensitization in the more difficult to eliminate from the pasture than north western Cape Province and during 1929–30 over *Pavetta*.

one million were killed by *Geigeria* spp., in Griqualand A plant that is found throughout Africa and beyond

West.’

is the castor oil shrub *Ricinus communis* (*Euphorbiaceae*). It has been grown as both a crop plant and as It is only possible to give a few examples with a view

an ornamental plant in gardens because of the striking
to stimulating the reader to carry out his own
leaves and its red stems. The toxic principle is ricin, a
researches. Among many strange poisonings, perhaps
toxalbumen, that is immunogenic and so repeated
the most odd is that caused by *Pavetta harbori*
ingestion of small doses produces immunity. Ricin is
(Rubiaceae) (gousiekte). The original report concerned
found in all parts of the plant, but particularly in the
a mob of fat cattle waiting in the holding pens at the
seeds. Pure ricin is one of the deadliest substances
rainside of Mahalapye Station, Botswana. The train
known to man (Georgi Markov, the Bulgarian refugee,
came into the station, sounded its horn and 18 beasts
was murdered by this poison being injected from an
dropped dead. The author found it hard to believe until
umbrella on London Bridge!). The toxin is not so
he had a similar experience (on a much smaller scale):
potent when ingested and the author remembers seeing
when he was investigating an outbreak of this disease,
an outbreak in Kenya in which 25 yearling dairy heifers

he drove into a kraal, shut the door of his truck and a
were involved. All recovered, but the moment they
fat bullock in excellent condition dropped dead. A post-
were turned back into the field they went straight back
mortem examination revealed no gross abnormalities.
to the plant and started eating it: they were removed
There had been a severe drought and the only green
from the field. The classic signs of the disease are a
plant on the range was *Pavetta harborii* and the animals profuse watery,
haemorrhagic diarrhoea, accompanied
had been eating it for several months. The lesions pro-
by inappetence, pain and bellowing. The normal post-
duced by this plant are caused by one or more cardiac
mortem picture is of a severe haemorrhagic gastroen-
glycosides and produce a diffuse hyaline degeneration
teritis. Since there is no way to demonstrate ricin, it is
and necrosis of heart muscle, with the muscles being
essential that a careful examination of the rumen con-
replaced by fibrotic tissue. There is no treatment for this
tents be made for the seed or leaves. The best means of
condition and the only control is by preventing access

control is to keep the animal away from the plant.
to the plant. Since there are few farm fences in
This is just a small sample of the 'unequalled variety
Botswana the only remedy is to dig up the plants – a
of poisonous plants' that are found in Africa. For those
forlorn hope.

whose interest has been stimulated, the books by
A second plant which kills by means of a cardiac
Kellermann et al. (1990) and Watt and Breyer-Brand-
poison is *Dichapetalum cymosum* (*Dichapetalaceae*)
wijk (1968) make excellent and entertaining reading.
(Mogau [Tswana] or gibflaar [Afrikaans]),
the

toxic principle of which is monofluoroacetate. In
the rumen this is converted into fluorocitrate which

Conclusion

interferes with the Krebs cycle. The plant has been
likened to an underground tree: the tips of the branches
In conclusion it must be said that only a small number
protrude above the soil while the vast majority of
of diseases have been discussed from a continent of infi-

*the plant is underground, making it very difficult to
nite varieties. There are virus diseases such as lumpy
eliminate. Unlike Pavetta, mogau is an acute poison
skin disease (p. 887) or bovine malignant catarrh (p.
which acts within four to 24 hours of ingestion, but as
935); there are protozoal diseases such as elephant skin
with Pavetta the animal drops dead without any pre-
disease or Ondiri disease (p. 763), rickettsial diseases
monitory signs. There are no post-mortem lesions and
such as heartwater (p. 765). There are many, many prob-
there are often no histological lesions. The only way to
lems and the governments have no money to control
confirm the diagnosis is to demonstrate the leaves in the
the major diseases, how much less these minor infec-
rumen.*

tions. Africa is in need of our help.

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Further reading

*Koch, R. (1903) Interim report on Rhodesian redwater or
'African coast fever'. Journal of Comparative Pathology and*

Parasitic diseases

Therapeutics, **16**, 273–80.

Theiler, A. (1904) East Coast fever. *Transvaal Agricultural Journal*, **2**, 421–38.

good diagrams and maps is:

Modern papers on various topics are given below:

Coetzer, J.A.W., Thomson, G.R. & Tustin, R.C. (ed.) (1994) Brown, C.G.D., Malmquist, W.A., Cunningham, M.P., Radley,

Infectious Diseases of Livestock with Special Reference

D.E. & Burridge, M.J. (1971) *Immunization against East*

to Southern Africa. Vol. 1, Section 1, 3–163, Oxford University Press, Cape Town. various authors and topics on this

schizonts grown in cell culture. Journal of Parasitology, **57**, 59–60.

Chema, S., Chuomo, R.S., Dolan, T.T., Gathuma, J.M., Irvin,

Trypanosomiasis

A.D., James, A.D. & Young, A.S. (1987) *Clinical trial of*

halofuginone lactate for treatment of East Coast fever in

Connor R.J. (1994) *African animal trypanosomiasis . In Infec-Kenya. Veterinary*

Record, **120**, 575–7.

tious Diseases of Livestock with Special Reference to South-Africa (ed. by J.A.W. Coetzer, G.R. Thomson & R.C.

(1985) *Immunization against theileriosis in*

ern Africa (ed. by J.A.W. Coetzer, G.R. Thomson & R.C.

*Africa. International Laboratory for Research on Animal
Tustin), Vol. 1, pp. 493–552. Oxford University Press, Cape
Diseases, Nairobi, Kenya.
Town.*

*Murray, M., Morrison, W.L., Murray, P.K., Clifford, D.J. & Trail, J.C.M. (1979)
Trypanotolerance – a review. World Animal*

Plant poisonings

Review, 31, 2–12.

Murray, M. and Dexter, T.M. (1988) Anaemia in bovine

The classic modern text, which is brilliantly written and

African trypanosomiasis . Acta Tropica, 45, 389–432.

superbly illustrated, is:

Nantulya, V.M. (1990) Trypanosomiasis in domestic animals.

*Kellerman, T.S., Coetzer, J.A.W. & Naudé, T.W. (1990) Plant The problems of
diagnosis. Revue Scientifique et Technique Poisonings and Mycotoxicoses of
Livestock of Southern*

de l'Office International des Epizooties, 9, 357–67.

Africa. Oxford University Press, Cape Town.

Apart from this the only book required is a good flora and the

East Coast fever

finest, although old, is:

*Watt, J.M. & Breyer-Brandwijk, M.G. (1968) The Medicinal For an early
description of the disease see the reports of Koch and Poisonous Plants of*

Southern and Eastern Africa, 2nd or Theiler:

edn., E. & E. Livingstone, Edinburgh.

Chapter 70

Rabies

R.S. Windsor

Introduction

1164

Aetiology (see p. 908)

Aetiology

1164

Epidemiology

1165

Europe

1165

Rabies virus belongs to the family Rhabdoviridae and

Africa

1165

is classified as a lyssavirus, from the Greek word for

North America

1165

rage. In recent years the rabies-related viruses have

The Caribbean, Central and South America

1166

been added to this genus. Today the rabies virus is

Asia

1166

Transmission

1166

referred to as lyssavirus serogroup 1: the Lagos bat,

Pathogenesis

1167

Mokola and Duvenhage viruses are serogroups 2, 3 and

Clinical signs

1167

4, respectively. What previously was thought to be a

Necropsy and pathological findings

1168

single, simple agent has been shown to belong to a

Diagnosis

1168

complex grouping. Studies using monoclonal antibod-

Differential diagnosis

1169

ies have shown that within a given geographical area

Vaccination

1169

the virus adapts to its environment, resulting in distinct

Control

1170

biotypes. Despite this, the antigenic structure of rabies virus has remained stable, which is a boon for diagnostic workers.

This is not the place to discuss the structure of

Introduction

the virus: suffice it to say that it is bullet shaped, and contains a single-stranded genome of RNA and five Rabies is the most feared of all diseases. Once humans structural proteins. Following attachment to a cell the show signs of disease, they die. This virus disease affects ribonucleoproteins are released and act as a template all warm-blooded vertebrates and is usually transmitted for replication in the cytoplasm. These accumulations ted by the bite of a carnivore. The virus enters the

of viral proteins in the cytoplasm give rise to the bodies peripheral nerves and spreads to the spinal cord and described by Negri.

brain, a process that might take several months. The The rabies virus is delicate and is destroyed by nervous disease that ensues may last from a few days sun and ultraviolet light, heat and many of the common to several weeks before the animal dies. In humans one disinfectants, e.g. 0.2 per cent quaternary ammonium of the characteristic signs is a fear of water – hence its compounds. However, it remains viable in nerve tissue, alternative name of hydrophobia.

the length of viability depending upon temperature The disease appears to have had a wide distribution and storage conditions.

since time immemorial, but it was Louis Pasteur Because of the work of Pasteur, rabbits were used as who demonstrated the association between the causal laboratory hosts for experimental work up to the agent and nervous tissue. By serial passage in Second World War, since which time the mouse has

*laboratory animals he transformed the field ('street')
been the principal experimental animal. At the same
agent into a 'fixed' agent with a shortened reproducible
time the virus was adapted to grow in embryonated
incubation period. Not until the twentieth century
eggs; these were later used to produce very successful
was it shown that the agent of rabies was a virus which
vaccines for use in animals. In 1958 the virus was first
could pass filters that retained bacteria. And in the
grown in tissue culture and there is a multiplicity of cells
same year, 1903, Negri demonstrated the intracytoplas-
in which the virus will grow. The rabies-related viruses
mic inclusions in the nerve cells of infected animals.
will also grow in tissue culture. Recently the human
These Negri bodies form the basis of diagnosis even
diploid cell line has been used for the production of a
today.
very successful human and animal vaccine.*

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Rabies • 1165

Epidemiology

(1)

The urban dog cycle: in the towns dogs spread the disease which can spill over into other species, It is mostly island countries that are reported to be free most commonly man. Cattle are not involved in from rabies infection – Great Britain, Ireland, Malta, this cycle.

Cyprus, New Zealand, Australia, Japan, Fiji and certain

(2)

The jackal cycle: this is the rural cycle with the Caribbean islands. Some mainland countries are also disease spreading between the black backed free from the infection – Denmark Portugal, Spain, jackal (Canis mesomelas) and being transmitted to Sweden, United Arab Emirates and South Korea. These cattle, small ruminants, wild ungulates and man. It are not exhaustive lists but the majority of the countries is jackals that infect most cattle. in the world have endemic rabies.

(3)

The mongoose cycle: this is seen in the yellow

mongoose in south eastern Botswana. The disease mostly cycles within the burrows of these animals

Europe

but can affect wild and domestic cat populations.

Rabies has been known and feared here since time

The domestic cat is responsible for most human

immemorial. Dogs, foxes and wolves have all played

infections in this part of Botswana. Cattle are not

their part in the spread of the disease. From the end of involved in this cycle.

the second world war rabies in foxes has become a

(4)

The kudu cycle: this is really a Namibian problem

serious problem with the disease moving westwards and

but may involve Botswana. The kudu (*Tragela-*

eastwards from Poland, reaching France in 1968. *Vacci-*

phus strepsisteros) outbreak was responsible for

nation and rigorous control of urban dogs has reduced

the death of more than 30 000 kudu between 1975

the disease dramatically in the past 30 years. The

and 1985, but still continues. The disease spreads

upsurge of rabies in foxes has resulted in an increase in from kudu to kudu: herbivores are normally the disease in cattle; until today cattle rabies is more thought to be 'dead end' hosts because they do not common than dog rabies. However, human rabies has spread the disease. Kudu groom each other, which been on the decline for many years and today it is a may account for this atypical spread.

rarity in Western Europe. The move to reintroduce the In East Africa the disease was first confirmed in 1912, wolf into Scotland has been halted for the time being with intermittent outbreaks until the 1960s. The war by the fear of rabies.

with Somali insurgents after Kenya's independence resulted in wholesale destruction of the herbivores in

Africa

the northern districts of Kenya. The jackals and hyenas, being deprived of their prey, moved into the villages It is likely that far more rabies occurs in Africa than is and there was an upsurge in human and dog cases. reported. In the 1960s rabies was mostly controlled

Jackal and urban dog cycles remain a problem to this throughout the continent. War, strife and the breakdown of veterinary services have resulted in an upsurge of the disease, but less disease is being reported from rabies is a problem in cattle when the jackal population many countries. There is some debate about whether is allowed to increase.

rabies existed in subSaharan Africa or whether it Non-fatal rabies-like disease is seen in west Africa and was introduced by the European colonists. The answer classical rabies does occur. The author will never forget is probably both: as will be seen below there is some walking into a house in northern Nigeria to find a mother evidence of virus modification in west Africa.

protecting her children from a rabid dog that had The first outbreak of rabies confirmed on the African entered the house. After dogs, cattle are the animals continent was in the Cape Province of South Africa most commonly seen with rabies in West Africa.

in 1892 in a dog imported from England that spread
In the countries bordering the Mediterranean, rabies
the infection to dogs, cats and cattle. The outbreak was
has been known since time immemorial: it is primarily
controlled and the disease was not confirmed again
a disease of urban dogs, but it spills over into herbi-
for more than 30 years. A rabies-like disease affecting
vores, including cattle, and humans. The Saharan coun-
mongeese and humans on the western side of the
tries are sparsely populated by animals and man and in
country was known and, in 1928, rabies was confirmed
consequence rabies is not a major problem in countries
in two children bitten by a yellow mongoose (*Cynictis*
such as Mali, Niger or Somalia.

penicillata). Since then rabies has been regularly
diagnosed throughout South Africa.

North America

Although suspected for many years, rabies was not
confirmed in Botswana until 1938. Workers in Botswana
Was rabies endemic in America or was it brought by the
believe that there are four manifestations of rabies:

Europeans? The latter may hold good because the

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disease was not seen until the middle of the eighteenth century. It is also possible that the disease came across for huge losses in the cattle population: some authorities quote annual losses of half a million for South America alone. It has been thought for many years that is well reported in Eskimo folklore. It was not common until the civil war, after which it increased they can be infected without being affected and yet in dogs on a regular basis until the end of the second world war. With increasing vaccination of dogs and consequent decrease in the number of cases there has been a variable length of incubation period before they a concomitant increase in the amount of sylvatic rabies.

succumb. The urban dog cycle is seen in the large South

Today there are approximately 4000 cases of rabies a

American cities, but it is not important to cattle rabies.

year in the USA, of which almost 90 per cent is in wild

animals (bats, foxes, raccoons and skunks being the

Asia

animals principally affected); domestic cats and herbi-

vores (including cattle) make up the remaining 10 per

There are many Asian countries with huge cities as in

cent of cases. The number of cases of rabies in cattle has

South America, and again urban dog rabies is a serious

remained static over the past 30 years, during which

problem. As a result, India has an enormous human

time the disease in dogs and cats has been decreasing,

death toll: some authorities put it as high as 25 000 a

thus demonstrating that it is not the urban dog cycle

year, although this figure is disputed. There is little hard

that involves cattle. Cattle rabies occurs most fre-

evidence of the level of infection in the dog population

quently in those states that have the highest levels of

in any of these poor Asian countries; there is even less

skunk rabies (Iowa, Texas and Minnesota), indicating information on the incidence of the disease in the cattle that it is this sylvatic cycle that involves cattle. The populations. More is known about the disease in India decrease in urban dog rabies with an increase in the than in other Asian countries and jackal and mongoose sylvatic cycle has also been seen in Canada.

cycles are known to occur.

It has been said that some wild species of mammal are natural or 'reservoir' hosts for the virus and that the rabies virus persists in these animals without inflicting

Transmission

harm until the virus is transferred to a different mammalian host. The extension of this theory is that the Rabies is normally transmitted by a bite, but not all infection will not be controlled in domestic animals animals bitten contract the infection. Although the until it is controlled or eliminated from these hosts.

virus is present in the salivary glands of infected carnivores for several days before the animal shows signs

of vampire bats in the spread of rabies (an important of disease, it is not normally secreted in the saliva cause of the disease in cattle), 30 of the insectivorous until clinical signs are apparent. There is no convincing species of bats have been shown in the Americas to be evidence of a species' susceptibility to rabies other infected with the true rabies virus (lyssavirus 1). It was than epidemiological data. Despite this animals have originally thought that all true rabies viruses belonged had their susceptibilities rated, as in Table 70.1.

to a single strain. However, recent work has suggested The site and severity of the bite is also important: the that there are species-specific biotypes of the virus. nearer the brain, the more rapid the onset. Non-bite transmission is not common. Transplacental transmission has been demonstrated in cattle, dogs and humans.

The Caribbean, Central and South America

There are reports of oral transmission from dam to The introduction, from India, of the grey mongoose offspring in humans and sheep via the milk. The only (Herpestes auropunctatus) in an attempt to control rats and snakes in the sugar

plantations has not been an unequivocal blessing. It soon became the principal car-

Table 70.1

Susceptibility to rabies.

nivore on the islands. Today almost 50 per cent of the mongoose population has antibody to rabies virus in its

Very high

High

Moderate

Poor

blood. Rabies has been eradicated from many of the islands but the mongoose population presents a serious

Foxes

Cattle

Humans

Opossum

Jackal

Cats

Primates

threat should the virus be reintroduced. In Mexico the

Coyotes

Skunks

Dogs

major problems are urban dog rabies and vampire bat

Rodents

Sheep/goats

rabies. The vampire bats are of several different genera

Mongoose

Horses

*but the most important is *Desmodus rotundus*. These*

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species in which this is a significant route of transmission has commenced there is transport of particles of sion is the kudu, which transmits the infection by the genome along the axon to the central nervous licking. There has been a report of aerosol transmission system. Viraemia can occur but it is thought to be of of rabies virus to two humans in a bat-infested cave in importance only in animals with damaged immune Texas, which was confirmed by placing animals in systems or pregnant animals. The mechanism of spread insect-proof cages in the cave. Transplantation of

from neuron to neuron is not properly understood, but corneas from cadavers to patients has resulted in the the genomic material may complete its maturation or transmission of the disease.

there could be direct transmission of the genomic mate-

The study of the outbreak in foxes in Western Europe
rial at the synaptic junctions. Once it reaches the brain
over the past 30 years has shown that normally the
the spread there is rapid, although adjacent structures
infection spreads from one family group to its neigh-
are thought to be the first affected. The accumulation
bour, with the occasional jumping of a group. Because
of virus in particular parts of the brain is thought to be
fox ranges are much larger in Canada than in Europe,
responsible for the clinical signs. Once the virus reaches
the disease spreads more rapidly there – up to 100 km
a particular site in the cord the virus can travel in a cen-
a year compared with 20–60 km here. Within a wild
trifugal direction out to the tissues. Thus the virus can
animal species it is thought that the disease spreads at
be found in a range of tissues and organs. However, it

a rate related to the number of animals available. As is thought that the spread to the salivary glands occurs the disease kills them so there is less opportunity for once the virus spreads within the brain.

spread and the disease becomes quiescent. The number Emaciation or failure to thrive is often seen and this of cases in cattle can indicate the weight of infection in is caused by damage to the hypothalamus and interfer- the predator.

ence with the production of growth hormone. Cell The level of urban rabies in a population is controlled mediated immunity is suppressed. Post exposure vacci- by the size of the city and the numbers of dog, the socio- nation is known to be effective, but the mechanism is economic status of the people and the efforts of gov- still not definitely understood. However, the production ernment to control the disease. Rabies was a serious of virus neutralizing antibody correlates with resistance. problem in Lima until the Veterinary Department sup- Passive antibody has been shown to be effective in ported by the World Health Organization instituted

animals that have been infected, if given in large effective vaccination campaigns.

enough doses. While it is in the nervous system it

The understanding of the pathogenicity and spread

seems that the virus does not stimulate the immune

of rabies-related viruses is as yet incomplete and any-

mechanisms but once it has passed into the non-

thing that muddies the waters of rabies control needs

nervous tissues, antibody production is stimulated. It is

investigation. One of the problems is that rabies-related

then too late to help the animal.

viruses have been isolated from domestic animals vac-

Recent workers have suggested that the pathogenic-

inated against rabies. There have also been cases of

ity of rabies is eminently suitable for the persistence of

rabies-related viruses in humans that have no history of

the virus and the spread of the disease. The virus is

a bite by a wild animal. Rabies-related viruses have

hidden from the immune system until it is too late. The

been demonstrated in bats in Australia, where rabies

distribution in the brain spares the neocortex and

has never occurred. Recently, bats infected with these results in behavioural changes that promote confrontation between rabid and healthy animals and these have come from Europe.

changes coincide with the arrival of the virus in the salivary glands. The high mortality ensures that there is a minimal accumulation of immune individuals in the

Pathogenesis

population and the occasional long incubation period ensures that the virus survives until there are sufficient A skin wound following a bite from an infected animal susceptible individuals present in the population!

is the common route of entry of the infection, hence the common route of entry of the virus is the sensory nerve endings in the affected area. With deep bites the virus

Clinical signs

enters the stretch proprioceptors of the muscle or the motor nerve endings in the muscle. The virus is thought

Man

not to enter through the cut surface of damaged nerves.

and this is followed by hydrophobia. Death may supervene in about 10 days or the patient may slowly develop relieve the choke at your own risk! Salivation occurs in paralysis and pass into a coma before death. A small less than half the bovine cases in the USA. As the percentage of patients develop signs similar to the disease continues, paralysis of the tail is seen, posterior dumb form of dog rabies. There is a gradual onset of paresis and a swaying gait are noted and the animal paralysis; once the respiratory muscles are affected the bellows. Loss of condition ensues and the animals go off patient dies. There are only three recorded cases of their legs, develop paralysis and between bouts of humans recovering from clinical rabies, all from the convulsions go into a coma and die. In north west New World.

Argentina a completely dumb form of the disease is seen in cattle, with the animal showing progressive paralysis. Other domestic animals show variations on

Dog

the pictures described above.

In the dog the incubation period is commonly two to

The classic sign of rabies in wild animals is the loss of

eight weeks, although there is a record of a dog

fear of humans. This was very dramatically seen in the

imported into Britain that developed rabies eight

kudu outbreak in Namibia in which the animals would

months after it arrived. It is likely that the prodromal

walk into houses. Jackals become very friendly but have

period seen in humans occurs in dogs but is recognized

sudden mood swings. Wild cats become more aggres-

by few owners, after which the dog passes into the

sive with rabies, although even some of these animals

classic 'furious' phase, showing nervousness, restless-

become docile. Most humans bitten by rabid mongeese

ness and exaggerated response to stimuli. Self-infliction

are bitten while handling these animals, thinking that

of injury at the site of bite is not uncommon. They may

they are tame.

attack anything – human, animal or inanimate objects

– and some will eat anything, including stones and

lumps of wood. There is no forgetting the look in the

Necropsy and

dog's eyes: a fixed stare or a far-away look which is dif-

pathological findings

ficult to describe. They may howl between the convul-

sive seizures and they often die during a seizure, or

*No consistent macroscopic lesions are seen. There may
paralysis ensues, the animal passes into a coma and dies.*

be emaciation and self-mutilation. The histological find-

Dogs that develop the dumb form of the disease may

ings are, however, consistent in the central nervous

show periods of furious activity. The muscles of the jaw

system. There is perivascular cuffing, focal and diffuse

are often the first to be affected and so the dog lies with

gliosis, neuronal degeneration and intracytoplasmic

its mouth open. The dog is unable to eat and many

inclusion bodies from 2 to 8 mm in diameter: these are

owners suspect that the dog has a bone in its throat.

the acidophilic Negri bodies. There is a marked species

Argentine veterinarians believe that the disease is

difference in the presence and distribution of these

caused by two different strains of the virus: the furious bodies. In the Ammon's horn (the usual place to look for the strain involved in the urban dog cycle and for such bodies) the number of bodies in humans and the dumb form by animals involved in the fox cycle.

dogs is variable. In herbivores, particularly goats and donkeys and to a lesser extent cattle, large numbers of Cattle

Negri bodies of variable size are found. In cats, Ammon's horn is a poor source of Negri bodies and Much less is known about rabies in cattle than in either so it is more common to make the smears from the humans or dogs. The incubation period is said to be cerebellum.

between two and 12 weeks (three to 21 weeks in the USA). Where the disease is transmitted by jackals it is not uncommon to find several animals affected at

Diagnosis

one time (although this was not the experience when the infection was introduced into Ghanzi District in The history and origin of the animal will suggest the

Botswana, where the single animal was the norm). The possibility of rabies and abnormal behaviour of any sort affected animal separates itself from the herd, shows should lead the clinician to suspect rabies. In most

Rabies • 1169

countries rabies has to be reported to the state or carried out on samples that were negative to the FAT. national veterinary authorities. If they suspect rabies as Histological examination was abandoned. Recently an the cause of the problem the normal action is to keep ELISA test has been developed which apparently gives the animal under observation for up to 10 days and to similar results to the FAT. The results can be read with kill it for laboratory diagnosis if the disease is suspected. With the advent of the fluorescent antibody test achieved without the need for expensive equipment. it is no longer necessary to wait for clinical disease to Viral tissue culture, the detection of viral nucleic acid progress before the animal is killed for examination. and serological tests are not used in diagnostic labora-

*This is particularly so with wild animals or animals of
tories but are restricted to research and investigation
unknown ownership.*

laboratories. Monoclonal antibodies have been used in

*The animal should be killed in such a manner that the
the identification of the rabies-related viruses.*

*brain is not damaged; it should be divided sagittally into
two halves, one half should be immersed in a large
quantity of 10 per cent formol saline (histological exam-*

Differential diagnosis

*ination) and the other half should be put into a large
jar of glycerol-saline (viral examination). Both jars are*

*There are many diseases that can be confused with
placed in a special container with sufficient absorbent
rabies in different animals. Distemper and canine
material to soak up the liquids should the bottle break;*

*hepatitis are the most common infections in dogs,
the parcel should be sealed and all should be sent to
heartwater (p. 765), cerebral babesiosis (p. 748),
the laboratory. In Botswana, where there were daily
cerebral theileriosis (p. 750), meningoencephalitis*

temperatures of up to 40°C, samples that were delayed caused by *Haemophilus somnus* (p. 907) and BSE in for a week en route could still be examined with a good cattle and parasites and toxic plants and chemicals in all chance of success.

species. One should first eliminate rabies and BSE and The tissue in formalin is used for making histological then proceed to the differential diagnosis.

sections and, in negative cases, may often help to confirm a differential diagnosis, e.g. babesiosis in cattle and distemper in dogs. Smears are made from the

Vaccination (see p. 1018)

Ammon's horn and can be stained by Seller's stain (a Romanovsky stain) in which the eosinophilic bodies Pasteur produced the first vaccine by suspending spinal stain pink. This technique is rarely used today because cords from infected rabbits in an atmosphere of sodium it has been replaced by the use of the fluorescent hydroxide for various periods of time. The drawback to stained antibody test in which the smears along with this vaccine (used for many years) was that the patient

control positive and negative smears are stained and had to receive 14 injections on a daily basis. There was examined under the ultraviolet light microscope. A also the possibility of the patient developing a neuropathy (paralytic neuritis) following vaccination. The definitive test is to make a suspension of the brain and inject this into the cranium of sucking mice. If rabies virus is present, the mice will die within 30 days and these immature creatures have less myelin, so reducing Negri bodies can be demonstrated in their brains. the risks of auto-antigenicity. Hen (for animals) and Unpublished results from Botswana show the relative duck (for human) eggs were the next vaccines produced value of these tests (Table 70.2). and both were used extensively for upwards of 20 years. Complete comparability between the fluorescent In some African countries the Flury strains are used to antibody test (FAT) and the biological tests was demonstrated. this day and they work well – high passage for cats and strated. Following this work, biological tests were only

cattle and low passage for dogs. Today the vaccine most

Table 70.2

Results of all brains examined over a period of three years.

Positive

Negative

FAT

Biological

Histological

FAT

Biological

Histological

207

207

156

235

235

286

FAT, fluorescent antibody test.

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commonly used in man and animals is the human

lation such slaughter may be counterproductive: rapid

diploid vaccine. Post exposure treatment of humans in reproduction will soon increase the numbers and all the Botswana is given on days 0, 4, 7, 11, 17 and 84. increase will be susceptible. Cats are not usually vaccinated because they play less part in the disease cycle. Oral vaccines have been developed for use in the control of the disease in wildlife, particularly for foxes. In countries where there is movement of animals then in western Europe. It has been shown that foxes will some form of permanent marking or certification is take chicken head bait laced with the vaccine in a required. Although the modern vaccines produce capsule. This has proved very successful in controlling immunity that lasts for three years it is normal practice the disease in foxes. So far it has not been extended to to mount a vaccine campaign on a yearly basis. Move- other species of wild animal. Future research will ment control from the affected areas is practised and undoubtedly be into the production of recombinant only vaccinated dogs are permitted to leave the area. It vaccines using harmless viruses: in this way it might be

is considered that an area can be considered rabies-free possible to vaccinate all species of wild animals that if there has been no case of the disease in the last two carry the rabies virus.

years.

In Botswana, cattle owners are not permitted to vaccinate their cattle against rabies. A susceptible cattle

Control

population acted as a marker for the infection of jackals. The economics of vaccinating cattle in Africa

In Britain, as in many other islands free from infection, are such that the farmer would not recoup the cost of the perfect control measure was in place, namely the the vaccine as it is cheaper to let the odd bovine die.

prohibition of importation of carnivorous animals

This is not the case in South America where the unless they underwent six months' quarantine. This was vampire bat can wreak havoc in a herd. The author changed in 2000 to a system of movement controls and knows of no country in South America that has a policy vaccination. It is interesting to speculate when the first

of cattle vaccination: it is left to the individual owner.

case of rabies will be diagnosed in Britain. Most coun-

The presence of rabies-related viruses in a country

tries in the world have laws in place to control this

complicates the control of rabies. It is known that with

disease. These vary from the strictest in Australia, where

some viruses the rabies vaccine provides some protec-

carnivores may only be imported from countries free

tion and so this has been used in human patients bitten

from infection and they are still required to be kept in

by bats. In the National Veterinary Laboratory,

quarantine for six months, to poor countries such as

Botswana, it was the policy that staff were not routinely

Tanzania where almost nothing is done (there were 400

vaccinated against rabies. It was thought that confi-

human deaths in the Mwanza region a few years ago

dence would breed carelessness. It was known that

because there was no money to buy vaccine!).

pre-exposure vaccination even with the tissue culture

What is needed to control the disease is an efficient

vaccine would not give adequate protection, if it were

laboratory to diagnose the infection and an efficient not followed up with post exposure vaccination. Over veterinary service to undertake field studies to determine the areas of the country in which the infection course of rabies vaccination.

is most prevalent, the various species of animal that With rabies eternal vigilance is required.

are involved and the populations of these animals. It is necessary to determine whether there are wild-life

Further reading

reservoirs of infection or whether it is purely a problem of urban dogs.

With this information it is possible to plan the cam-

A modern text with a bent towards southern Africa is:

Swanepoel, R. (1994) Rabies. In Infectious Diseases of paign of control and budget for its costs. In most Third Livestock with Special Reference to Southern Africa (ed. World countries rabies vaccination is supplied free to by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin), Vol. 1, dog owners during the campaign, either at predeter-

pp. 493–552. Oxford University Press, Cape Town.

mined vaccination sites or on house to house visits.

Owners who require their animal to be vaccinated

An older text on rabies in the tropics including vaccination:

Kuwert, E., Mérieux, C., Koprowski, H. & Bogel, K. (eds)

outside this time usually have to go to a private veteri-

(1985). *Rabies in the Tropics*. Springer-Verlag, Berlin.

nary surgeon. In Botswana, at the time of vaccination,

the dog is painted on the head. Once the vaccinations

An older text concerning South America:

are completed then control teams go into the area and

Anon (1984) *Septimo Informe*. Comité de expertos de la OMS

capture or shoot all dogs without the paintmark. Other

sobre Rabia. World Health Organization, Geneva. (Control

countries do not have such draconian measures. In

in cities, pp. 39–41; vampire bats, pp. 71–5.)

countries where there is little control of the dog popu-

Rabies in cattle in the USA:

Rabies • 1171

Baer, G.M. (1986) *Rabies in cattle*. In *Current Veterinary* Steck, F., Wandeler, A., Biochel, P., Capt, S., Hafliger, U. & *Therapy 2: Food Animal Practice* (ed. by J.L. Howard),

*Schneider, L. (1982) Oral immunisation of foxes against
pp. 501–504. W.B. Saunders, Philadelphia.*

*rabies: laboratory and field studies. Comparative Immunol-
ogy, Microbiology and Infectious Diseases, 5, 165–71.*

Oral vaccination of wildlife against rabies:

Chapter 71

Dairy Farming in Saudi Arabia

J.C. Fishwick

Introduction

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nies, both privately and state owned. In addition to

Cattle industry

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farming activities most dairy companies have their own

Dairy farming

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processing plants and distribution systems.

Role of the veterinary surgeon

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The full range of pasteurized fresh dairy products

Function of the health team

1176

that would be found in any Western country is readily

Summary

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available. The largest selling product is a traditional

Middle Eastern fresh natural drinking yoghurt called

Introduction

laban. About 60 per cent of fresh milk production is

fermented into laban while approximately 25 per cent

With an area of approximately 2.1 million square kilo-

is processed as fresh milk. The retail price of a litre of

metres and an estimated population of over 19 million

milk has fallen rapidly due to competition pressures

people, the Kingdom of Saudi Arabia dominates the

from the equivalent of 75 pence (US\$1.05) in 2000 to

Arabian Peninsula. Within its boundaries, the country

about 53 pence (US\$0.80) in 2002.

holds the two most sacred sites of the Islamic faith, the

In contrast to Europe, a higher value is placed on fat

Holy Mosques of Mecca and Madinah, and Islam is

than protein in milk and this is reflected in the nutrition

the all-embracing force which is seen in every aspect of of dairy cows and in sire selection. This is primarily Saudi society. The nation was united into its present because of the demand for traditional breakfast cream form by King Abdul Aziz in 1932 and is still ruled as an called ghiste. However the demand for skimmed and absolute monarchy by his descendants.

semi-skimmed milk is increasing and both products are Saudi Arabia consists almost entirely of desert or readily available. Milk quality is generally excellent and semi-desert areas and is one of the driest countries on most companies produce milk with a bacterial count earth, with an annual rainfall in most areas of around 100 and somatic cell count that would qualify for the highest mm. The rainfall that is seen is irregular and unreliable. payment bands offered by UK milk purchasers.

There are two distinct seasons with summer lasting from The beef industry is quite small. The majority of March to early October, when daytime temperatures are locally produced red meat consumed is sheep, goat consistently above 40°C and commonly reach as high

and camel and the best quality beef is imported. Most as 50°C, while at night the temperature will remain local beef is slaughtered at up to 18 months of age and above 30°C. The daytime temperatures during the winter is referred to as veal. Veal cattle are usually castrated months are usually in the range 20–30°C. Coastal regions Holstein bulls sourced from the dairy sector. There is suffer with extremely high humidity, especially in the also a small amount of beef produced by nomadic summer, whilst the central regions are characterized by farmers who keep suckler cows.

a very dry atmosphere.

There are effectively no private veterinary surgeons Saudi Arabia has the largest known oil reserves in working in the cattle industry; veterinary surgeons the world, together with vast amounts of natural gas and are exclusively employed directly by individual farming mineral wealth. The discovery of these natural resources companies.

had a dramatic effect on the nation during the latter half of the twentieth century and the health of the economy is

now closely linked to the price of oil on the world market.

Dairy farming

Milking cattle are Holsteins which are zero grazed

Cattle industry

with a diet based on home-grown alfalfa hay and maize

silage. Saudi Arabia is fortunate in having enormous

The dairy industry is sophisticated and is concentrated

underground reservoirs of water and it is this water

in the hands of relatively few large, integrated compa-

which is used for both dairy farming and arable pro-

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Dairy Farming in Saudi Arabia • 1173

duction. Certain areas of the desert are extremely cooling during the summer months and even during fertile if water is provided and the basis of crop and the winter cooling still plays an important role. A good forage production is an irrigation system known as the illustration of the high temperatures experienced is the centre pivot (Fig. 71.1). A pivot consists of a bore hole fact that mercury clinical thermometers are of little producing water which then passes into a horizontal

use in the summer. The air temperature is considerably overhead pipe mounted on wheels which makes a slow higher than the normal body temperature of a cow and circuit and irrigates a circular area of land around so it is almost impossible to make an accurate measurement of a cow's rectal temperature.

required and pivots may have a diameter of up to The technology for cow cooling was originally 800 m. From the air pivots may be seen as 'green circles developed in the arid regions of Arizona, USA. One in the desert'.

commonly used cooling system involves a large electrical fan mounted in the roof which blasts air downwards such as soya, flaked maize, cottonseed, cottonseed hulls, onto cattle. The air may be cooled by evaporating concentrated sugar beet pulp and fishmeal. Feed is usually fed as a trolled amounts of atomized water into it. A cow house total mixed ration off the floor with the cattle in lock-will need a whole series of fans to provide effective

able head stanchions. Feeding time provides an opportunity for breeding and other management procedures. Cooling and for much of the year these will be operating 24 hours a day (see Chapter 8). Cooling is also essential in parlour collecting yards (Fig. 71.2). The best dairy units have around 4500 to 5000 dairy cows together with a large youngstock rearing operation. Cows are housed on sand and, over time, the original sand bedding accumulates increasing amounts of dung. Cows are milked four times a day in a two hundred stall parallel parlour which operates 24 hours a day, with This provides a peaty type of bedding which is comfortable between shifts for cleaning and routine maintenance. A farm of this size has a total staff of about 145 men, including everyone from the farm manager to cooks and administration staff. Rolling herd average milk yield in the best units is over 12 000 litres per head. Lactating cows are housed in groups of 300 and considerable effort is spent to ensure that this number of animals calves in as short a period as possible to fill the house quickly. The spread of calving dates in the house

must be minimal so that they can be managed and fed as a single group. First lactation cows are housed separately from older cows.

The extremely high temperatures experienced both day and night throughout the summer months make careful control of the cows' environment essential.

Efficient milk production is difficult without effective

Fig. 71.2

Coolers over a collecting yard. Note the udder

Fig. 71.1

A centre pivot irrigation system.

washers on the floor.



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*portable and works extremely well, provided it remains
ment of livestock into the region from the Horn of
dry. To maintain the beds in good condition houses are*

Africa and from more northerly states such as Turkey, groomed four times a day by scraping the top layer of Iraq and Iran. The greatest threat to dairy units comes bedding outside and leaving it in the sun to dry, whilst from the large, mobile population of sheep and goats in previously dried material is scraped back inside. It is the region and these may commonly be infected whilst possible to maintain a house for several years before showing only minor clinical signs.

completely cleaning out and starting with fresh sand.

Prevention of FMD is maintained in two ways:

Rain is uncommon, but when it does fall it can cause physical security of the unit and a stringent vaccination serious problems with the bedding which is associated policy. The principle of physical security is that live- with an increased incidence of mastitis and lameness.

stock, people and vehicles do not enter the unit unless

Mastitis is maintained on well-run units at a level absolutely necessary. When they do, sensible precau- of less than 20 cases per 100 cows per year, and levels tions are taken in terms of disinfection of vehicles and

would be considerably lower in some cases. Meticulous provision of farm overalls and footwear. However, attention to the Five Point Plan for many years has been these precautions are no substitute for preventing entry very successful in reducing contagious mastitis to very low levels and most of the cases are environmental mastitis. There is still a lot to be learned in the management of cows and housing in this type of system to reduce environmental mastitis further.

Farms are fenced with a single point of entry (Fig. 71.3) which is manned 24 hours a day. Cattle sales and disposals are done through a separate gate at the perimeter so that cattle trucks do not enter the farm

Staff are divided into several teams with responsibility for areas such as milking, feeding, health, breeding, (Fig. 71.4). There is a large separation between the calving and youngstock rearing. Because of the large perimeter fence (Fig. 71.5) and the cow houses, typically number of cows being dealt with, members of these

200 m or more. Essential deliveries are kept to a minimum and lorries are disinfected before entry. Visitors are not allowed and all company staff entering the site must leave their vehicles outside the perimeter fence.

mum and lorries are disinfected before entry. Visitors are not allowed and all company staff entering the site must leave their vehicles outside the perimeter fence.

Role of the veterinary surgeon

There are special risks when companies with multiple sites have to move livestock between their own

The veterinary surgeon has an essential, proactive role farms. The risk is reduced by using lorries exclusively

to play in the productivity and health of the herd.

for their own animals, efficient disinfection and

First and foremost he must establish performance

ensuring a member of farm staff always accompanies

targets, monitor them continuously and intervene

the driver to ensure that cattle are not exposed to any

immediately when performance starts to deviate

unnecessary risk during transit. Forage is only sourced

from that target. As well as collecting, handling and

from suppliers known to have effective fencing to keep

interpreting data this involves working closely with farm

their pivots free from livestock.

staff at all levels to investigate problems as they arise.

Examples include the calving live birth rate, incidence of mastitis, conception rates, incidence of ketosis and cow and calf mortality. If any of these parameters fall below the set target investigations must start immediately.

Secondly, he must establish the optimal working practices for each team and produce written manuals to set these out in a clear and methodical manner. It is important to utilize the very best current knowledge available in an area and produce firm guidelines that can be applied simply on a very large scale.

Thirdly, he has the responsibility for the maintenance of herd disease security. There is probably no other situation in the world where herd disease security takes on such an important role. Foot-and-mouth disease (FMD) and rinderpest are a real risk and if either of these were to enter a large unit the results could be catastrophic.

FMD is endemic in the region and attempts to

Fig. 71.3

A farm gatehouse, which is controlled to reduce the control it on a national scale are thwarted by the move-risk of foot-and-mouth entering the farm.



Dairy Farming in Saudi Arabia • 1175

Fig. 71.4

Loading ramp at the perimeter fence for livestock

Fig. 71.5

Farm perimeter fence. Security is important in a movement. Cattle are moved through a separate gate from the region where foot-and-mouth is endemic. The perimeter fence is main entrance to ensure that cattle trucks do not enter the farm.

separated from the cattle housing by several hundred metres.

One feature of farming in this region is that a large proportion of farm staff are expatriates from countries such as the Philippines, Kenya, Sri Lanka, Nepal, Pakistan, India, UK and Ireland. Most staff are resident on site and this reduces the chance of staff coming into contact with other local livestock.

Preventing the movement of cattle and personnel does nothing to prevent the chance of airborne spread between units. This is a particular risk if another dairy unit in the region is suffering a clinical FMD outbreak. The chance of infection by airborne spread appears to be greatest when humidity is above 60 per cent.

The second component of FMD prevention is vaccination. The antigenic components included in the

Fig. 71.6

A 100-stall parallel milking parlour.

vaccine available in Saudi Arabia are authorized by the Saudi Ministry of Agriculture and Water, based on the best scientific advice available from authorities such as the Institute of Animal Health at Pirbright, UK.

FMD vaccination is carried out every 75 days in all stances and the schedule described above would be stock over 6 months old. This routine is carried on a amongst the most vigorous seen in the region.

predetermined day and all cattle are included regard-

Despite the great attention given to FMD preven- less of any other considerations. Calves less than 6 tion, outbreaks of the disease do occur sporadically and months old are given a course of three vaccinations at they will probably remain a feature of dairy farming 4, 5 and 6 months old before joining the whole herd while the disease remains endemic in the region. schedule.

A fourth role is the development of effective control

Vaccination against FMD is a tremendous cost to any and contingency programmes for a number of diseases

dairy organization. Apart from the direct costs of vaccination which may occur. FMD provides the most obvious example but other diseases such as mycoplasmal mastitis, brucellosis and tuberculosis, all of which may significantly but short lived reduction in milk yield. Typically total daily milk production may fall by an amount equivalent to between 0.5 kg and 2.0 kg per cow for occur, need definite policies in place to deal with them. In the case of mycoplasmal mastitis a control programme must ensure that sensible precautions are in place to prevent it, that sampling and laboratory loss associated with the keeping of dairy cattle in the procedures are effective in continuously monitoring for region. Dairy companies do vary in the vaccination the disease and that should an outbreak occur a well-schedule employed depending on their own circumstances organized response can be implemented immediately.

Other roles include the coordination of parlour

Any cows that show problems are removed and given testing and maintenance, which assumes great importance when cows are being milked around the clock in returned to their houses or retained in the hospital for one or two hundred stall parlours (Fig. 71.6); maintenance as necessary.

nance and monitoring of raw milk quality, co-ordination

The most common disease identified in fresh cows is with consultant nutritionists and selection of bulls.

ketosis and the diagnosis of this condition is assisted with the use of a urine dipstick. Any fresh cow that is submitted for clinical examination will have a urine test

Function of the health team

done as a routine. The incidence of diseases such as ketosis and left displaced abomasum is recorded daily

Staff on a large dairy unit are commonly divided into and this information is used by nutritionists, the farm a number of specialist teams, one of which is the health manager and the feeding team to assess the effective-

team. The principle of a health team's activity is that
ness of their feeding management.

problems must be detected early and rapid intervention

Careful observation of cattle at frequent intervals

must ensure that affected cows are returned to full pro-

during the day is a very time consuming activity, but it

ductivity as soon as possible. Any cows that do require

is considered essential to the effective management of

treatment are moved to a separate hospital area which

a dairy unit. A successful health team is judged by an

has its own dedicated milking parlour. All the milk pro-

empty hospital area and not by the amount of sophisti-

duced in this parlour is disposed of as waste to prevent

cated treatments being given to cows in it.

any mastitic or drug-contaminated milk from entering

the main milk output of the farm.

Health teams concentrate most of their efforts on

Summary

the observation of cattle in their houses. All cows are

locked in their stanchions for feeding four times each

The dairy industry in Saudi Arabia has developed

day and it is during this 'lock up' period that health rapidly over a period of about 25 years and has been teams walk the cows and observe carefully for any signs greatly influenced by the experience gained from dairy of abnormality. Particular attention is given to cows farming in arid regions of the USA. It is now probably within the first three weeks after calving. These as efficient and advanced as anywhere in the world. fresh cows are checked four times a day whilst others Prevention and control of foot-and-mouth disease is a are checked twice a day. Although this type of dairy major challenge and is very expensive. farming is carried out on a large scale, great attention Regardless of the size and scale of any operation, a is paid to the health of individual cows as judged by herd is comprised of individual cows. A herd can only careful observation and basic clinical skills. Health men operate efficiently if full attention is given to the individual members of that herd. This applies as much in a properly and looks healthy at the time of each lock up.

unit of five thousand cows as it does in one of fifty.

Chapter 72

***Bovine Medicine in New Zealand
and Australia***

N.D. Sargison and J.J. Vermunt

New Zealand

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Recently, there has been an increased interest in autumn

Dairy farming

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calving, thereby spreading the workload for both the

Beef production

1177

farmer and the dairy factory. Inputs to factory supply

Animal health

1178

herds are low, with little supplementary feeding, no

Reproductive management

1178

winter housing, efficient milking plant and a low labour

Major problems

1179

requirement. Farms are subdivided into small paddocks

The Australian scene

1182

and most lactating cows are mob stocked and shifted daily. The annual production of 3000–3500 l/cow is low

New Zealand

by comparison with grain-based systems. Cows are stocked at an average of 2.7/ha and produce 600–1200 kg milk solids (fat and protein)/ha. Only small amounts

Dairy farming

of hay or silage are conserved for winter feeding and New Zealand's dairy herd of slightly over 3.3 million the use of temporary electric fencing enables a variety cows is small by comparison with Europe and America. of winter grazing systems to ration winter pasture and However, about 90 per cent of the milk production ensure adequate spring growth. A small number of town is processed for export as butter, cheese and milk supply herds have a greater reliance on winter feed and powders, making New Zealand a major contributor to

produce milk to quota all year round.

the world dairy trade. New Zealand's unique climate favours extremely efficient livestock production from

Beef production

clover and perennial ryegrass pastures, top dressed with phosphatic fertilizers, which enables the unsubsidized

The majority of the beef produced in New Zealand dairy industry to compete on the world market.

is exported to the USA as processing beef, some is

Dairy farming is concentrated in areas of reliable

exported to North and South East Asia and about a

high rainfall (Northland, Waikato, Taranaki and

fifth is consumed locally. About 50 per cent of the total

Southland) and where irrigation is possible and cost-

beef production is from suckler cows and 50 per cent is

effective (Canterbury). Most production is from New

derived from the dairy industry, comprising cull cows

Zealand–Friesian and Holstein–Friesian cows (57 per

and Friesian bulls which are reared to between one

cent), although there are still a significant number of

and two years-old on lowland pasture. The original role

Jersey (16 per cent) and crossbred herds (19 per cent).
of suckler cattle in New Zealand was to improve
The average herd size is about 230 cows and managed
pasture quality for more profitable sheep grazing and
by one person, but herds of 800 cows or more are not
to prevent reversion to bush. Whilst recently the price
uncommon. About 30 per cent of the dairy farms are
of beef has improved relative to wool and lamb, most
operated on a share milking basis where, for example,
of the country's 1.4 million suckler cows are still kept
the owner is responsible for the farm maintenance and
on steep and rolling hill country farms in a complement-
the fertilizer costs, whilst the share milker owns and
ary role to sheep. Angus cattle with mature weights of
milks the cows. This system traditionally provided a
about 475 kg are the predominant breed,
although
route of entry to the industry for young farmers, who
there are many herds of Hereford and crossbred cows.
could eventually accumulate enough capital to buy
The numbers of Charolais, Simmental and Limousin

their own property.

cattle are low by comparison with Europe, partly

The majority of dairy farms are seasonal producers, because of their high grazing requirements for maintenance relative to production.

timed with the lush spring growth, and supplying milk

Typical herds of 150–200 suckler cows achieve calving percentages of 80–85 per cent depending on the topog-

raphy of the country. Beef production is wholly depend-

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to three bulls per 100 cows, depending on the topogra-

phy of the farm. Most reproductive problems are asso-

ciated with an extended calving period and veterinary

separate and regularly moved around the farm along

input is, therefore, based on the achievement and main-

tenance relative to production.

with the sheep. Pasture is prioritized depending on the

tenance of a compact calving period.

nutritional requirements of the different classes of live-

- *Heifers or cows which calve late during one season stock at different times of year and care is taken to inevitably calve late during the following season avoid competition with sheep, especially during the late or do not become pregnant before the end of a winter–early spring period. Winter grazing is managed compact mating period.*

by the rotation of mobs around paddocks. Store cattle

- *Calves which are born late in the calving period are are mostly finished on intensive lowland farms, where younger and lighter at weaning than those born weight gains from about 1 kg/day in spring to 0.5 kg/day earlier and, therefore, of lower sale value.*

in autumn are achieved on a pasture-only diet.

- *The labour requirement for supervision is lower when the calving period is compact.*

Animal health

- *Feed budgeting is more accurate for heifers and cows calving over a known short period.*

The unsubsidized New Zealand beef and dairy industries are very sensitive to world production and market trends. The annual performance of most of the country's beef cows and the published results of field studies of reproduction and requirements for veterinary input are, therefore, different when compared to the current situation (Hanly & Mossman, 1977) indicate that the average pregnancy rate is 60 per cent for each oestrous cycle, provided that heifers or cows are cycling and the bulls are sound. After calving, the basis of veterinary input to most herds. The treatment of individual sick animals is often uneconomic, there is a post-partum anoestrus period of at least 40 days, which is related to body condition at calving and especially in extensively grazed beef cattle where early signs of disease may not be seen. However, there is calved heifers than in mature cows with the same body

an important role for veterinary advice on the control condition. Thus, provided that all heifers or cows are and prevention of production limiting diseases. Whilst cycling at the beginning of the mating period, a 5-respiratory disease and calf scour are relatively un-oestrus-cycle (105-day) mating period is required for 99 important diseases in animals which are not housed, per cent of cows to be pregnant. However, in subdependence on a solely pasture diet can predispose to quent years if the bulls are introduced on the same date, specific animal health problems. New Zealand's volnot all cows will be cycling at the time of bull introduction and a 99 per cent pregnancy rate will not be in the absence of supplementary feeding, trace element achieved by 105 days. Therefore, a mean calving interval significantly greater than 365 days is required to Mycotoxicoses such as facial eczema and ryegrass sustain a 99 per cent pregnancy rate. Alternatively, staggers, which are seldom seen in Europe, can also be

a sustainable 365-day calving interval can be important production limiting diseases. Most lameness achieved when bulls are removed after three oestrus in dairy cattle is associated with the unavoidable need cycles, although annually about 6 per cent of the cows to walk long distances from pasture to the milking shed, will not be in calf (Mossman & Hanly, 1977). In the and consequently the lameness-causing lesions differ absence of annual cow subsidy support and provided from those seen in winter-housed animals. Problems that the difference between cull and replacement cows of winter pasture feeding and management, the meta-is low, this not-in-calf rate is more than compensated for bolic demands of late pregnancy and early lactation and by increased weaning weights due to the greater mean stress associated with prolonged wet spring weather age of calves at weaning. often result in a high incidence of metabolic diseases in Good heifer management is the key to achieving satisfactory reproductive performance in the whole herd.

Provided that heifer management is correct, nutrition is adequate and sound bulls, matched for size, with high

Reproductive management

libido and low estimated breeding values for calf birth-

The pattern of pasture growth in New Zealand means weight are used, the achievement of good reproductive that suckler cows must be spring calving and a mean performance in subsequent years is relatively straight-calving interval of 12 months is therefore essential.

forward. Traditional heifer calving at three years of age

Most suckler cows are mated in large groups with two presents few problems. However, about 30 per cent of

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New Zealand beef heifers are first calved as two-

formed by the farmer and 70 per cent by trained

year-olds, which necessitates careful management to technicians. Heat detection is assisted by the use of tail

avoid dystokia and subsequent reproductive problems.

paint. Poor reproductive performance in dairy cows is

Heifers weighing 65 per cent of their mature weight

often associated with extended post-partum anoestrous

at 14–15 months of age should be cycling and achieve periods due to failure of winter/spring pasture to meet target pregnancy rates when mated. For 15-month-old the nutritional requirements of advanced pregnancy Angus, Hereford and Simmental heifers, this critical and early lactation. Intravaginal progesterone releasing minimum weight is in the order of 270 kg, 290 kg and devices (e.g. CIDR, InterAg, NZ; Cue-Mate, Duirs, 310 kg, respectively. Heifers intended for calving as NZ; PRID, Stockguard, NZ) are routinely used in an two-year-olds are preferentially managed and regularly attempt to overcome this problem and to induce monitored to ensure that target weights are achieved oestrus in later calving animals.

through good nutrition and control of growth-limiting diseases, such as parasitism and trace element deficiencies. Heifers which do not reach the critical minimum

Major problems

weight at 15 months of age are usually culled from Copper deficiency (see p. 298)

the breeding herd or mated the following year to avoid

the problems of poor pregnancy rates and failure to breed as second calvers. On many hill farms, or on lowland farms with heavy breeds of cattle, the achievement of critical minimum weights by the age of 15 months is not possible and calving as two-year-olds is associated with high spring levels of molybdenum in peat soils uneconomic.

or the application of molybdenum to pasture as a fertilizer. There are also reports of poor reproductive performance associated with copper deficiency, although the problem of a longer post-partum anoestrous period in calved heifers. When cows are mated over a period of 63 days, this is achieved by mating replacement

ill-thrift.

heifers for only 42 days or by mating heifers for 63 days,

Whilst the copper levels of New Zealand pastures

but introducing the bulls 21 days before the start of

vary little throughout the year, the levels of interfering

mating in the cow herd. Forty-two-day heifer mating is

factors, molybdenum and iron, increase significantly

suited to calving at two years of age, because animals

throughout the winter and spring. Copper availability

are three weeks older at the time of bull introduction,

is therefore lowest in spring, at the time of year when

which can make a significant difference to achieving

animal requirements for late pregnancy, lactation and

critical minimum weights and subsequent herd repro-

early growth are highest.

ductive performance, while 63-day heifer mating is best

Both serum and liver copper concentrations are used

suited to traditional three-year-old heifer calving.

in the diagnosis of poor animal performance, but liver

Poor body condition at calving results in long calv-

is the only useful sample when the reasons for sampling

ing to conception intervals and a high not-in-calf rate are to determine whether reserves are adequate to when a short mating period is employed. Furthermore, last throughout the winter or to monitor a supplementation programme (Ellison, 1992). The correct number conceive late in the breeding season, calve later, wean of samples is essential (minimum of eight sera or four lighter calves and fail to become pregnant as second liver samples), because the laboratory reference values calvers. Under intensive grazing conditions, replacement are based on mean values from these numbers. Liver ment heifers cannot compete with adult cows for nutrient samples are obtained by biopsy or from slaughterhouse ents unless managed as a separate group through to or necropsy material.

their second calving. Having achieved good heifer management, day-to-day management is based on assessment, which depends on many factors including the ing the body condition of cows in accordance with their

severity of the deficiency, production system, stock-reproductive status. If feed inputs are below requirements, cows will lose weight and fail to reach target condition score for mating and eventually fail to rebreed. Short-term supplementation of dairy cattle can be provided by daily drenching with copper sulphate in the milking shed or administration of copper salts through drinking water, where 60 to 70 per cent of the cows conceive to proven sires by artificial insemination. Intermediate-term supplementation is achieved in both dairy and beef cows by the parenteral injection of

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copper glycinate or copper edetate, whilst long-term supplementation can be provided by the oral administration of copper oxide particles (capsules or bullets) or

the mycotoxin sporidesmin, which is present in the pasture top dressing with copper sulphate.

spores of the saprophytic fungus Pithomyces chartarum.

Under the ideal conditions of high humidity (close to 100 per cent) following a prolonged warm period

Selenium deficiency (see p. 302)

(>20°C with a minimum night temperature of 12°C),

About 30 per cent of farmed land in New Zealand is

spore numbers can increase rapidly in the mat of

considered to be selenium deficient. The most important

leaf litter beneath shaded pasture. Sporidesmin is

selenium responsive condition in New Zealand cattle is

rapidly absorbed and becomes concentrated in the liver

ill-thrift in growing calves and growth responses of 5–10

where it causes epithelial necrosis of the bile ducts

per cent can be achieved by supplementation on most

with periductal oedema and inflammation, leading to

deficient pastures. Selenium responsive infertility and

cirrhosis. Most affected animals show no outward

poor milk production have been reported on severely

clinical signs. However, the resulting liver damage

deficient pastures (Grace, 1994). The selenium response causes ill-thrift and a significant reduction in milk production. Photosensitization occurs in about 10 per cent of affected animals and is due to derangement of and widespread supplementation.

the biliary excretion of phylloerythrin (Bruère et al. , The selenium status of cattle is usually confirmed by 1990).

the laboratory analysis of blood glutathione peroxidase (GSH-Px) or selenium concentrations in serum, whole blood or liver, all of which provide useful diagnostic information (Wichtel, 1998). Liver samples collected from finished calves at slaughter are commonly used to assess the risk of disease and expedite preventive measures. Monitoring consists of recording weather conditions and carrying out pasture spore counts using to determine the need to supplement. Within any herd,

spore trapping or pasture washing methods. When there is little between-animal variation in the concentrations rise rapidly, or reach threshold value, the options of selenium in blood, serum or liver, or blood are to move stock to safer pasture, to instigate protective treatment with zinc salts, to apply fungicides to the or four animals usually provide adequate information. pasture or a combination of these practices. Fungicide The New Zealand reference ranges for selenium are spraying can provide safe pasture for six weeks, but based on numerous calf growth response trials can be expensive, especially when it has to be repeated (Ellison, 1992). Overseas reference ranges are of no value to New Zealand conditions and vice versa, administration of large doses of zinc oxide, before exposure to toxic levels of sporidesmin, effectively reduces the severity of liver damage, although the mechanism

pasture nutrition. Economically significant growth is unclear. Milking cows can be dosed daily in the milk-rate responses to supplementary selenium are unlikely ing shed, but other classes of cattle are usually drenched at liver selenium concentrations above 600 nmol/kg weekly at a higher dose rate to overcome the impracticities (wet weight).

calities of yarding and handling. Alternatively, zinc Sodium selenate or selenite administered orally, as sulphate can be administered to dairy herds through a pour-on or by subcutaneous injection can provide a the water supply. Wax-coated, slow-release zinc oxide useful method of short-term supplementation. Long-intraruminal boluses (Time Capsule, AgResearch, NZ) term supplementation is achieved by controlled re-are available for use in calves. The boluses slowly dissolve subcutaneous injections of barium selenate paste, solve in the rumen to provide four weeks' protection intraruminal selenium pellets or pasture top dressing and are useful when animal handling is difficult. Zinc with sodium selenate or sodium-barium selenate prills.

salts have no therapeutic value when administered after

Pasture top dressing is an efficient supplementation

exposure to sporidesmin.

method where stocking rates are high and is the pre-

Outbreaks of facial eczema can be avoided by judi-

ferred supplementation method on about 30 per cent of

cious farm management practices such as feeding of

selenium deficient properties.

hay or other supplements during high risk periods,

avoidance of close grazing and removal of stock from

high risk areas. Affected animals should be provided

Facial eczema

with shade or if possible housed in darkness during day

Facial eczema is an important cause of ill-thrift and

light hours. Milking cows should be dried off and skin

hepatogenous photosensitization during late summer

lesions treated topically.

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Ryegrass staggers

the back of the group. The problem is compounded by

poorly maintained tracks which cause direct trauma

Ryegrass staggers occurs under close grazing conditions to the sole and slow the herd down, causing further tions during summer and autumn in perennial ryegrass impatient handling. Impatient forcing of cows results in (Lolium perenne) dominant pasture-fed livestock cows skidding as animals from the back of the group throughout most areas of New Zealand. The disease is are forced against those in the middle; tussling to caused by tremorgenic mycotoxins (lolitrem A and B) avoid body contact, resulting in unplanned placement which are produced by the perennial ryegrass endo- of limbs; abnormal forces being taken on the abaxial phytic fungus Acremonium loliae and is characterized wall of the lateral claw as cows attempt to resist being by tremors, incoordination and depressed growth rates. forced off the track; shortening of the stride so that the The prevalence of the endophyte in perennial ryegrass hind limbs are no longer placed in the same safe spot is greatest during summer and autumn, in particular as the fore limbs and cows being unable to see their in the plants' lower sheaths, old leaves and seedheads.

lower limbs as their heads are forced up and above the

Many modern ryegrass cultivars have been selected

animals in front (Chesterton, 1989).

*for high levels of the A. Loliae endophyte, because it Most lameness problems
can be managed by patient*

is associated with resistance to attack by the Argentine

movement of cows along the farm track to the milking

stem weevil (Listrionotus bonariensis), an important

shed, proper maintenance of tracks and careful han-

agronomic pest. Such selection has inadvertently re-

dling in the shed. Congestion points and distractions

sulted in an increased incidence of ryegrass staggers

should be removed and the track surface should be non-

(Bruère et al. , 1990).

abrasive, correctly crowned and well drained, especially

Clinical signs develop within one to two weeks after

the parts closest to the milking shed (Vermunt &

introduction to toxic pasture. At rest, the only visible

Greenough, 1997).

signs may be fine tremors of the head and neck, but

when animals are disturbed, clinical signs immediately

become apparent with development of head nod-

Bloat (see p. 832)

ding and jerky movements of the neck and limbs.

Pasture bloat is a common cause of sudden death in

Moderately affected animals have a wide-based, sway-

cattle in New Zealand. It is caused by the production

ing stance and a high-stepping gait. Severely affected

of a stable foam which traps the normal gases of fer-

animals show a stiff-legged gait and usually collapse

mentation in the rumen (frothy bloat) and inhibits eruc-

when moved. Whilst the disease is distressing to ob-

tation. Pasture bloat occurs most commonly when cattle

serve, most losses are associated with misadventure.

graze pastures dominated by lush, immature, rapidly

However, affected animals are difficult to handle,

growing legumes (clovers, lucerne), but there are also

interfering with normal management practices. There is

reports of the disease when cattle are grazed on other

no specific treatment for the disease, but animals re-

materials such as turnips, brassica and cereal crops, and

cover within days of removal from the toxic pasture.

young grass with a high soluble protein content. While
However, the avoidance of close grazing of high endo-
death is the most obvious loss associated with bloat,
phyte ryegrass dominant pastures can be problematic,
subclinical and mild bloat are associated with consider-
especially during dry summer conditions.

able production losses.

Although bloat susceptibility is heritable, progress
in the genetic selection for low bloat susceptibility

Lameness (see Chapter 31)

has been slow (Morris et al. , 1991). Outbreaks of bloat Lameness is a
significant problem in New Zealand dairy

with several cows dead and/or stabbed are devastating,
herds. Unlike the situation in winter housed cattle, the
and nowadays most farmers take precautions by using
main problems are excessive wear and bruising of the
antifoaming agents, which are still the only reliable way
sole, perforation of the sole (septic pododermatitis) and
of controlling or preventing bloat. Historically, farmers
white line disease. Most herd problems are associated
have used detergent based products for controlling

*with poor design, construction or maintenance of farm
bloat. These have included oils, marlophens, pluronics
tracks and impatient animal handling.*

*or turps, and currently the most favoured are the
Observational case-control studies of New Zealand
alcohol ethoxylate derivatives due to their extended
dairy herds have shown that when cows walk at their
persistency of action. Most commercially available
own pace, each limb is accurately placed to avoid injury.*

*products are suitable for daily drenching, trough treat-
The incidence of claw injury is low in herds which are
ment or pasture spraying. The rumen modifier mon-
allowed to drift to and from the milking shed, even on
ensin (Rumensin Liquid and Rumensin Anti-Bloat
rough or poorly maintained tracks. Significant problems
capsule, Elanco, NZ), is marketed as an effective
occur when cows are hurried by pushing the animals at
product in the prevention of milk bloat. Monensin is*

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*not a detergent, but acts to reduce the bacterial/proto-
problems associated with this grain feeding (e.g. abo-*

zoal activity that releases foam producing compounds
masal displacement, ruminal acidosis, salmonellosis and
in the normal rumen.

laminitis/lameness) are therefore relatively common.

Specific diseases common to Australia but exotic
to New Zealand are the insect-borne diseases such

The Australian scene

as anaplasmosis, babesiosis (ticks), ephemeral fever or
'three-day-sickness' and Akabane disease (midges).

Dairy farming in Australia is largely confined to
those climatic regions which are favoured by higher
rainfall and better quality soils, especially in the eastern

References

seaboard states and Tasmania. Flood irrigation is used
extensively in areas where rainfall is very seasonal.

Bruère, A.N., Cooper, B.S. & Dillon, E.A. (1990) Mycotoxi-

Although each of the states in Australia maintains its

coses. In *Veterinary Clinical Toxicology*, pp. 167–84. Foun-own highly regulated
dairy industry, the entire industry

dation for Continuing Education of the New Zealand

is influenced by the dominance of the southern dairy

Veterinary Association, Palmerston North.

industry, particularly that of Victoria, which produces

Chesterton, R.N. (1989) Examination and control of lame-

well over half of the total Australian production.

ness in dairy herds. New Zealand Veterinary Journal, 37, Like New Zealand, the Victorian and Tasmanian dairy

133–4.

industries depend heavily on world markets for their

Ellison, R.S. (1992) A review of copper and selenium refer-

manufactured and export products. The ability of

ence ranges in cattle and sheep. Proceedings of the New

farmers freely to sell market milk contracts (quota) is

Zealand Veterinary Association Sheep and Beef Cattle

limited in most states, as is the ability to establish new

Society, 22, 3–27.

Grace, N. (1994) Selenium. In Managing Trace Element Defi-

dairy enterprises. This is in contrast to New Zealand,

ciencies, pp. 9–23. AgResearch, Palmerston North.

where dairy conversions are occurring on a large scale.

Hanly, G.J. & Mossman, D.H. (1977) Commercial beef pro-

The average milk price to the farmer is slightly higher

duction on hill country. *New Zealand Veterinary Journal*, **25**, than in New Zealand, but is still low when compared to

3–7.

Europe or the USA.

Morris, C.A., Cockrem, F.R.M., Carruthers, V.R., McIntosh,

Similar to New Zealand, dairy farming in Australia

O.T. & Cullen, N.G. (1991) Response to divergent selection is highly efficient in labour and land usage and is prefor bloat susceptibility in dairy cows. *New Zealand Journal dominantly seasonal with the majority of the herds*

of Agricultural Research, **34**, 75–83.

being spring calving. The average Australian dairy herd

Mossman, D.H. & Hanly, G.J. (1977) A theory of beef pro-

is smaller than in New Zealand, but the degree of

duction. *New Zealand Veterinary Journal*, **25**, 96–100.

‘Holsteinization’ of the national dairy herd is further

Vermunt, J.J. & Greenough, P.R. (1997) Management and

control of claw lameness – an overview. In *Lameness in*

advanced. Dairy cows graze pasture all year round

Cattle (ed. by P.R. Greenough & A.D. Weaver), 3rd edn, pp.

and this forms the major portion of the diet. In addition

308–15. W.B. Saunders, Philadelphia.

to this, supplementary feed sources such as grain are

Wakelin, R.L. (1992) Copper supplementation for ruminants.

widely used. Consequently, the average milk produc-

Proceedings of the New Zealand Veterinary Association

tion is higher and the average days in milk longer. Grain

Sheep and Beef Cattle Society, 22, 43–51.

is fed at a much lower cost, because of the proximity to

Wichtel, J.J. (1998) A review of selenium deficiency in grazing the main grain producing areas. Certain animal health

ruminants. New Zealand Veterinary Journal, 46, 47–52.

Chapter 73

North American Dairy Production and

Veterinary Involvement

T.W. Graham

Milk production trends

1183

animal health, give expert advice in milk production,

Veterinary involvement

1183

reproduction and growth, help design and modify

Approaching a herd problem – an example

1184

facilities, design and monitor food safety protocols

Veterinary monitoring on the dairy farm

1185

(pathogen- and chemical-free meat and milk) and

Disease monitors

1185

provide information to producers on-cost-effective pro-

Mastitis monitoring

1185

duction of meat and milk products sold.

Fertility monitoring

1185

Production monitoring

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Veterinary involvement

Milk production trends

The focus of this discussion is to outline those aspects

of dairy production (meat and milk) about which vet-

Production of high quality nutrient-dense milk and

erinarians can provide information to dairy owners to

meat is a primary goal of dairy operators throughout

enhance performance of their enterprise. Clearly our North America. As our populations become more training as veterinarians is focused on animal health aware of human health concerns regarding food safety, aspects of dairy cattle production. Ironically, it is often they have required that we provide them with products poorly planned and maintained housing, milking without pathogens or chemical contaminants. As they parlour/equipment design and inadequate or unbalanced, consumers expect their food to be safe, whole-anced rations that will curtail production and lead to some and highly nutritious. With urbanization, con-disease. Understanding the requirements for clean sumers have become less aware of how their food is comfortable housing and bedding and then helping pro-produced, both as a raw and manufactured product. ducers implement those changes greatly increases Animal welfare issues have increasingly become a animal welfare and performance. The use of inorganic concern for consumers. Now, more than ever, veteri-bedding (i.e. sand) in properly designed freestall barns

narians are expected to provide leadership in animal has a positive effect on feet and legs, mastitis and milk husbandry and care, ensuring that all animals are production. Reducing the time that cows spend stand-treated humanely throughout their lives. Veterinarians ing on concrete reduces the incidence of laminitis and are in a key position to provide leadership in animal sole abcess formation. Increasing the time cows spend welfare, health and food safety.

laying down chewing their cud increases mammary As an industry North American dairies are increas- blood flow, with the subsequent effect of increasing ing in cow numbers, but the number of facilities con- milk production. Laying down a manure-free environ- tinues to decline. Because of increasing animal density ment greatly reduces exposure to environmental strep- it is likely that future dairy owners will face more envi- tococci, staphylococci and coliform bacteria, which ronmental constraints. Environmental constraints are helps reduce the incidence of environmental mastitis. increasing the operating costs for existing and newly

Knowledge of nutrient needs from birth to slaughter built dairies. Environmental laws are likely to restrict greatly increases our ability to reduce health problems building and expansion of existing and new dairy facilities and manipulate growth, reproduction and milk production. Cost of production for meat and milk continues to rise. From our classical veterinary training, knowledge of the pathophysiology of disease allows the attending veterinarian to recognize the conditions being diagnosed and guide therapeutic intervention. Taking meat and milk with high efficiency and that animals remain healthy. Veterinarians are now expected to maintain understanding of the epidemiology and dynamics of

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diseases in groups of animals. Understanding the

tion string were collected directly from the feed wagon

aetiopathogenesis of disease allows for intervention on as it delivered feed to cows. Rations, as formulated, a herd basis after identifying the affected groups and were evaluated to determine any deviations from governmental recommendations (Subcommittee on Dairy underlying causes of diseases within a herd. Knowing what factors predispose cows to disease and what Cattle Nutrition, National Research Council, 2001). control methods can be used to remove or attenuate Antepartum cows had elevated NEFA levels, indicating risk for disease in exposed cattle will greatly help dairy cating weight loss in the last weeks before calving. Cows owners to produce milk and meat profitably, often with had low calcium levels before and after calving with less labour.

only rare milk fever, suggesting that the antepartum diet was not well balanced for prevention of hypocalcemia. Urine pH was 8.0 in antepartum cows, indicat-

Approaching a herd problem – an

ing that the anionic diet was not effective. Non-lactating example

and lactating cows had high serum magnesium levels, suggesting higher than normal intake of magnesium. As a case example, an 1100 cow herd had several hundred cows with diarrhoea throughout the lactating period. Diet analysis suggested that antepartum cows did not have adequate protein intake and their daily diet herd (900 lactating cows). The incidence of mastitis intake was low. This suggested that cows should be varied from zero to ten new cases per day. The expected losing weight before calving, predisposing them to peri-incidence was 0–1/day. In the first 10 days after calving, parturient immunosuppression, retained placenta, mastitis, metritis and displaced abomasum. The lactating placenta and/or metritis. The expected incidence of cows also had high Mg levels and diet analysis indicated retained placenta was 5–8% and was principally associated with dystokia and twin birth. The expected incidence of metritis in newly calved cows was 3–5 per cent.

DM). Lactating cow diets also contained 22 per cent and mostly associated with retained placenta. The protein in the DM. *Mycoplasma bovis* was discovered number of cows with displaced abomasum varied from in a few cows with mastitis in the early lactating period three to ten per week across all lactating strings, but and several cows were culture positive for environmental streptococci, staphylococci and coliform incidence of displaced abomasum was 1–3 per cent of bacteria.

cows calving, with none of the cases coming from cows

Ration and feed analysis suggested the milk cows

later than 30 days post calving. Cows dying or being

were oversupplemented with Mg and protein, and

sold as unproductive approached 50 per cent of the

effective fibre may have been limiting. *Salmonella* cul-milking herd over a 5-month period and the expected

tures were negative and results of BVD serology and

rate was 30–35 per cent. The milk production was 30

PCR probes for BVD suggested that neither *Salmonella* nor BVD were the cause of diarrhea. As formulated, dry cows should have been fed a ration adequate in energy and protein, but intake was low, probably by the herd manager as poorly producing or sick were causing weight loss and contributing to periparturient fever, displaced abomasum, ketosis, low milk production, fertility examinations. All calving and mastitic cows had composite milk samples collected aseptically for identification of *Mycoplasma* and aerobic bacteria. Approximately 10 per cent of cows in three groups had blood samples collected: cows before calving, in the first 10

lited, dry cows should have been fed a ration adequate in energy and protein, but intake was low, probably by the herd manager as poorly producing or sick were causing weight loss and contributing to periparturient fever, displaced abomasum, ketosis, low milk production, fertility examinations. All calving and mastitic cows had composite milk samples collected aseptically for identification of *Mycoplasma* and aerobic bacteria. Approximately 10 per cent of cows in three groups had blood samples collected: cows before calving, in the first 10

out the herd, reduce protein in the lactating cow rations days after calving and approximately 100 days after and to increase the effective fibre in the lactating herd. calving. Metabolic profiles (haemograms and serum Close-up and far-off dry cow rations were reformulated chemistries) were run to help determine the underlying to reduce feed sorting and to increase intake for all causes of disease. Faecal cultures for *Salmonella* sp. cows. String cultures (emphasizing newly calved and were collected, as well as blood for determining exo-hospital cows) and retrospective reculture of all cows sure to or active infection with bovine virus diarrhoea having had mastitis within the past two months were (BVD). Antepartum blood samples were collected to initiated to identify mycoplasma-positive cows. From determine serum non-esterified fatty acid (NEFA) interviews with the dairy manager it was noted that levels. Urine pH was determined to evaluate the effec- most cows were administered 40–120 mg of dexam- tiveness of the anionic dry cow diet. Diets by produc- ethasone for udder oedema in the first one to four

*days after calving. Routine use of dexamethasone was
may be needed to reduce disease to expected levels.*

greatly curtailed to alleviate the immunosuppression

*Identifying factors that may contribute to lower than
associated with its use.*

expected levels of disease can advance the science of

After reducing the protein and Mg levels to govern-

veterinary medicine. Communicating new ways to

mental recommendations for level of milk production,

reduce disease incidence can allow those of us in prac-

diarrhoea in the milking cow herd was reduced to one

tice to contribute to our personal growth, advance our

to two cases/900 cows per week. These were mostly

clients' needs and advance the way veterinary practice

attributed to indigestion. Replacement of long-stem oat

is carried out.

hay with corn silage from the dry cow diets and refor-

A traditional and still important area for veterinary

mulation of the close-up mineral package reduced the

intervention is development of cost-effective vaccina-

urine pH to 6–6.5 and increased diet intake and union protocols and evaluation of their success. This formity of consumption of these two rations. Of cows should be done for all stages of life and production. calving, retained placenta and metritis incidence was Evaluating calving and calf facilities and performance reduced to 6–8 per cent and 3–5 per cent, respectively. is a way to begin routine herd health monitoring and The incidence of displaced abomasum declined to 0–3 intervention. Evaluation of the calving facility cleanliness cases/month. Cows at three to five days post calving or ness, calving protocols for ensuring healthy cows and identified with mastitis had milk aseptically collected calves after calving (e.g. hygiene in assisting with from all four quarters and all cows positive for dystokia or the force applied when pulling calves), mycoplasma were removed from the herd. All purcolostrum monitoring (bacteria counts, amount, chased cows were routinely monitored for contagious density) and calfhood disease control (cleanliness of the

mastitis and treated, segregated, culled or returned to calving and newborn facilities, cleanliness of the feeding the supplier as needed. It was thought that periparturient utensils, milk and feed and protocols for routine disease ent immunosuppression, both from poor feed intake and treatment) are good starting points. Focusing veterinary from use of dexamethasone, facilitated the mycoplasma care in these areas has a high return on investment for outbreak. After initial identification of the index producers as they are areas that are often neglected, yet mycoplasma-positive cows it took approximately one have a great effect on profitability when well main-month to bring the incidence down to zero. Evaluation tained.

of the freestall environment and cows in the milking parlour indicated that the freestalls were not well main-

Mastitis monitoring (see Chapters 23, 25)

tained (wet and manure/urine contaminated), but the cows were clean and dry when milked. It was recom-Mastitis control and evaluation of pathogens in milk is mended that the freestalls be dug out and refilled with

still a very important service provided by veterinarians.

and to encourage stall acceptance and reduce exposure

*Evaluation of types of bacteria in individual and bulk
to organic bedding.*

tank samples, individual and herd somatic cell counts

and total bulk tank bacteria counts can direct

where and when intervention is needed to correct a

Veterinary monitoring on

herd/equipment problem. Routine milking equipment

evaluation, quality control for teat dips and milker

the dairy farm

training schools are valuable services to producers to

help ensure the sale of quality milk.

In addition to traditional veterinary tasks of routine

health care (including biosecurity protocols), surgical

and reproductive intervention, and disease monitoring,

Fertility monitoring (see Chapter 41b)

veterinarians should be involved in facility design,

With several strategies of intervention for improving

selecting equipment and monitoring its performance

pregnancy rates within a herd (e.g. timed insemina-

(feed trucks, milking equipment), personnel training and prostaglandin synchronization programmes), and management, ration formulation, monitoring milk assessing the success and cost-effectiveness of these production and heifer growth, forage production and programs is important. Evaluation of personnel skills on a monitoring of stored feed quality.

facility will help determine the likelihood of success and whether these types of programmes should be used in an enterprise. Use of bull breeding may be most appropri-

Disease monitors

ate for some herds, while in others highly successful artificial breeding programmes can greatly aid in creating a high quality and uniform genetic base for a herd. Goals expected levels of disease and whether intervention for reproductive performance have been well described

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over the past 40 years, but with the advent of products count) will help guide decision-making for feeding

such as recombinant bovine somatotropin (rBST) some changes, forage selection, production, harvest, storage of these goals have changed. Some have suggested that and removal for feeding. Examination of foodstuffs can increasing the calving interval in these rBST herds can help identify those practices that alter cow health be economically advantageous. However, maintaining a and milk production. Poor quality forage production, high proportion of the herd pregnant (50 per cent or harvest, storage and removal for feeding can directly more) with good conception rates (2.0–3.0 services/ affect both both feed intake and cow health. Overly conception) still remains a primary goal to ensure mature feed that is too dry when ensiled can become profitability of a dairy enterprise.

mouldy or compost, leading to both an unhealthy feed Evaluation of body condition at calving, one to two and one that is unpalatable and poorly available. Too months after calving, at conception and at cessation of wet a forage or contamination of feed with animal lactation can help maintain reproductive performance.

carcasses can lead to high levels of clostridial growth,
Increasing rates of anoestrous cows in early lactation
predisposing animals to diseases such as botulism.
(60–120 days in milk) suggest weight loss in the dry
Improperly stored corn, barley, canola, cottonseed, soy-
period or early post-parturient period (0–90 days), heat
beans and other protein meals can lead to mould pro-
stress or inadequate nutrition in the current ration.
duction and subsequent mycotoxicosis. Contamination
High levels of endometritis at 30–45 days' lactation
of feed from bird droppings and wild and domestic
suggest problems in the periparturient period and
animal faeces allows for spread of *Salmonella*,
require that all factors contributing to the endometritis
Leptospira
and other shared pathogens.
Cross-
be identified (e.g. nutrition, hygiene practices, dystokia,
contamination of feeds with cattle manure increases the
twinning). Correcting these deficits will help keep the
spread of Johne's disease, BVD, *Salmonella* spp., etc.

herd reproductively sound, reduce treatment costs,
Recommendations for facility design to limit feed trans-
lower culling rates and help maintain profitability.
mission of infectious diseases facilitate disease control
The use of progesterone assays to verify heat
at the population level. This type of activity is analo-
detection accuracy (at the time of insemination) is
gous to the design of vaccine and treatment protocols
another tool the veterinarian can use to help monitor
that veterinarians have engaged in for years. Taking
herd health, although anoestrous cows can also lack
steps to reduce exposure of the animals on farms to
detectable levels of progesterone. Holding breeder
these agents is also becoming a goal for limiting con-
training schools to teach or review heat detection
tamination of food at the consumer level.
techniques, semen handling and proper insemination
Feed sorting, or the selection of high concentrate
techniques can maintain high fertility in a herd.
feedstuffs over forages by cattle, increases the likeli-
As part of any herd monitoring programme, evalua-

hood of lameness, subacute ruminal acidosis, reduction of the incidence of periparturient disease allows in the production of fat in milk and ultimately lower for a retrospective evaluation of the health of non-milk yield. Directing forage production (selecting seed, lactating cows and cows in the first month of lactation. fertilization, planting density), timing (immature or Recording and evaluating the incidence of ketosis, dis-mature) and method of harvest (e.g. kernel processing placed abomasum, milk fever, diarrhoea, pneumonia, or rolling of corn silage as it is harvested, chop length), mastitis, etc., are critical so that action or target levels size and dimensions of storage facilities for forages, for intervention may be set. Certainly diseases should management of forage removal (face management of be monitored at all stages of life (birth to sale) and goals silage) will help producers keep cattle healthier and of performance evaluated for each stage of production. more productive. Veterinarians, by the very nature of Routine monitoring of urine pH and fresh cow meta-our relationship with producers, often are in the best

bolic profiling (Ca, Mg, P, K, Na, Cl, liver function, position to evaluate feeding practices. If we are evaluating serum protein) will help evaluate the success of those practices it makes sense that we are the ones non-lactating and early lactating feeding and housing directing how to feed the cows for health and production programmes. This will help ensure that cows perform well throughout lactation. Monthly or bimonthly evaluation and formulation. Because cow health is appropriate and can be more or less inclusive directly affected by improper nutrition, it is increasingly depending on the frequency of farm visits.

important that veterinarians become directly involved in ration formulation.

As part of the routine evaluation of cattle health,

Production monitoring

veterinarians should monitor why cattle are sold from Routine evaluation of production parameters, such as the herd and the number sold in a given time. Culling feed intake, calf growth and milk production (milk flow,

rates directly affect profitability. While production may butter fat, solids not fat, protein, lactose, somatic cell appear very good for cows in a herd, this can be deceiving if the number of cows entering and leaving the herd tional and financial advice including commodity con- is high. If replacement costs are low and the value of tracts, crop selection and forage production. The oppor- milk high that strategy could be very profitable. Today tunities for veterinarians are ever expanding, allowing in North America, cattle prices are high and the average several avenues of input into animal agricultural price for milk sold has remained relatively steady. If a production.

producer has a high cull rate (45–55 per cent of the herd/year), milk production per cow can be quite high (13 000 litres (£3000) per 305-day lactation). However,

Further reading

because of the high removal rate and high cost of replacements, this strategy can reduce net profitability

Martin, S.W., Meek, A.H. & Willeburg, P. (1987) Veterinary or prevent a dairy

from growing if cows are removed

Epidemiology, Principles and Methods. Iowa State University for disease. If the high cull rate is because cows are

sity Press, Ames.

being sold for dairy production (highly valued cattle) to

Radostits, O.M., Leslie, K.E. & Fetrow, J. (1994) Herd Health, other herds and the producer has an excellent calf

Food Animal Production Medicine, 2nd edn. W.B. Saunders, raising programme, this strategy could prove to be very

Philadelphia.

profitable. Helping producers evaluate their return on

Sniffen, C.J. & Herdt, T.H. (1991) Dairy Nutrition Management, Veterinary Clinics of North America, Food Animal

investment of a given strategy is an appropriate task for

Practice. W.B. Saunders, Philadelphia.

veterinarians to engage in.

Subcommittee on Dairy Cattle Nutrition, Committee on

In summary, the role of veterinarians is rapidly

Animal Nutrition, National Research Council (2001) Nutri-

changing in North America. Today more of the focus of

ent Requirements of Dairy Cattle, 7th edn (revised). National veterinarians will be in operations management, includ-Academy Press, Washington, DC.

ing herd health, personnel training, udder health and

Van Soest, P.J. (1982) Nutritional Ecology of the Ruminant.

milking equipment evaluation, facility design, nutri-

Cornell University Press, Ithaca, NY.

Chapter 74

The North American Beef Industry

D. Sjeklocha and S. Sweiger

Introduction

1188

because of their ability to deal with the extreme heat.

Cow-calf sector

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In the northern United States Bos taurus breeds are

Cattle distribution

1188

used because of their ability to withstand the cold

Cattle feeding sector

1189

temperatures.

Meat packing sector

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Sixty per cent of the United States' cattle herd is

The practice of beef cattle veterinary medicine in North America is located in the south east. Amongst others, states in this

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area would include Florida, Georgia, Alabama, Mississippi, Louisiana, Tennessee, Kentucky, North Carolina

Introduction

and South Carolina. In this area, only 1 to 5 acres (0.4–2 ha) are required to sustain a cow and her calf for one year. There are a few very large herds in this region,

The North American beef industry is very diverse and the vast majority are very small herds. Many of these are complex. It is distinctly separate from the dairy industry. Small herds are either a hobby or a secondary source of income, with the exception of cull dairy cows and castrated males for the producer. Often, these producers simply use male dairy stock (steers), which are eventually added to have a small parcel of grass that would not be utilized for the beef supply. ‘Dual purpose’ breeds are very seldom at all unless the producer owned a few cows to graze it. used for true dual purposes. There are three sectors to

These producers are not necessarily in the cattle business the North American beef industry: the cow-calf, cattle feeding and meat packing sectors. A very short synopsis would say that the cow-calf sector provides cattle for this, genetic control of production is not taken very seriously in many cases. In recent years, producers and meat packing industry. One may wonder why the meat-veterinarians in this area have taken steps to improve packing sector would be included in this discussion. It genetic selection and to market cattle in uniform has become evident in recent years that all three sectors groups.

must work together and share information in order to The remaining 40 per cent of the nation's cattle herd deliver a beef product that provides a good eating experience spread throughout the rest of the United States' grazing for the end consumer. So, in this light, each of able landmass. In some regions of this land mass, only

these sectors will be discussed.

one acre (0.4 ha) is required to support a cow and her calf, while in other areas, as much as 100 acres (25 ha)

Cow-calf sector

is required to support a cow and her calf. Generally, as the carrying capacity of the land decreases, the size of the herd increases. The regions that have a high carry-

Cattle distribution

ing capacity are also regions that produce a great deal This sector is undoubtedly the most diverse. This is due of cereal grains. Cow-calf producers in these areas take to the fact that it uses grazed forage (primarily grass) advantage of the crop residues by allowing their cattle as its primary raw material. Grazable forage is available herds to graze the fields after they have been harvested, in every geographic region. The landscape and climate gleaned the grains that were missed by the harvesting throughout North America differ greatly between geographic regions. As the landscape and climate changes, little cereal grain production, there is more reliance on

so do the types of forages available in that region as harvested forage (grass hay, alfalfa hay, etc.) for winter well as the type of herd management. Cattle herds are feed. Herds in these regions can be as small as one up developed in such a way to best deal with the environment as many as 15 000 head or more.

ment in which they live. For example, in the southern It is generally considered to be good management for United States, Bos indicus breeds are very popular cow-calf producers to adhere to a fairly rigid 'calving 1188

The North American Beef Industry • 1189

season'. Producers will turn bulls out with the cattle for predicted by the use of expected progeny differences 45 to 90 days for breeding. At the end of this time (EPDs). EPDs give the bull buyer an idea of how a particular bull's calves may perform in various traits.

for better use of the producer's time and a more uniformly sized calf crop. Some producers do not adhere to this type of calving season, some of whom allow their

Cattle feeding sector

cows to have calves all through the year. The majority of calves are born in the late winter or spring and are weaned in late summer or in the fall. The cattle feeding sector has become very specialized.

In the 1960s, many cow-calf producers fed their own calves to harvest weight. As more science and technology within the cow-calf sector. Some producers will go through a strict 'preconditioning' programme, in which and cost-effective for cattle feeding operations to get the calves are vaccinated once while still on the cow increasingly larger, and less efficient and cost-effective (typically against IBR, BVD, PI3, BRSV and clostridial for the cow-calf producer to feed his own cattle. Many disease). Then, approximately three weeks later, they of the modern cattle feeding operations (known as are revaccinated against the viruses, implanted with a 'feedlots' or 'feedyards') can hold 100 000 head of cattle growth-promoting hormone and weaned. The calves

or more at one time.

are then fed a grain and roughage ration for anywhere

A feedyard is a collection of pens that are used to

from a couple of weeks to several months, depending

confine cattle while they are being fed a high-energy

on the marketing plans of the producer. Other

ration. One side of the pen is lined with a concrete feed

producers will vaccinate their calves once, pull them off

trough (feed bunks). Trucks, fitted with a special box

the cow and sell them. Still others will simply pull their

that is capable of mixing feed, auger feed into the feed

calves off the cow without vaccinating them and sell the

bunks as they drive alongside them. Feed management

calves. Most bull calves are castrated at some point

is considered to be one of the most important and

prior to marketing, becoming known as 'steers'.

detailed jobs around the feedyard. Calling for the

Some cow-calf producers will retain ownership of

wrong amount of feed can hurt the performance of the

their calves through the feeding period up to harvest.

cattle in that pen for the rest of the feeding period.

These producers most commonly send their calves to a Feedyard cowboys ride through each pen of cattle every 'custom' or 'commercial' feedyard and pay the feedyard day looking for sick cattle. Sick cattle are removed from to feed the calves for them. This will be discussed in the pen and placed in a hospital pen, where treatment more depth in the cattle feeding section. Most cow-calf is administered and the animal is given time to recover producers sell their calves at some point prior to the before being returned to the original pen.

feeding period. The most common form of marketing Cattle are usually hauled into feedyards by semi calves is done through a livestock auction (also contractor-trailers. These large trucks can carry 50 000 to 56 000 lb (22.5–25.4 tonnes). It is not uncommon for livestock auction, sorted for size or other common characteristic and sold to the highest bidder. Disadvantages feedyard. This puts the cattle under a great deal of stress of this type of marketing are that cattle are commin-

and increases the chances that they will become sick. gled, thus allowing disease transfer. Cattle can also be Over the years, feedyards have become very adept at sold via 'satellite auctions' in which video images of dealing with this stress. Many feedyards have pens that calves are transmitted by satellite all over the country are used especially for receiving cattle. These pens are for bidders to view and bid on. Still more cattle are now usually smaller than other pens on the feedyard and have being sold over the internet/world wide web. These excellent drainage so the newly arrived cattle have a dry latter two marketing options offer the advantage of not place to rest. Fresh feed and clean water are provided at commingling different groups of cattle, as the cattle are all times. After the cattle have eaten, drunk and rested, marketed directly from the ranch of origin. they are put through a series of procedures known col- Within the cow-calf sector is a group of producers lectively as 'processing'. Processing may include any or collectively known as 'seedstock producers'. These are all of the following: vaccination (against pathogens such

producers who specialize in producing breeding stock as IBR, BVD, PI3, BRSV, Pasteurella/Mannheimia, spp. to be used by the commercial cow-calf producer. Careful clostridial pathogens, Haemophilus somnus, etc.), attention is paid to fertility, carcass traits, muscling and implantation with a growth hormone, ear tagging, performance characteristics (average daily gain, administration of a parasiticide, administration of a weaning weight, yearling weight, feed conversion, etc.). vitamin/mineral supplement, castration of bulls or pregnancy examination of heifers. If a heifer is found to be pregnant, the pregnancy may be aborted.

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Upon completion of processing, the cattle are moved in the United States, there are three very large ones that to a regular pen on the feedyard. Over the next two to three weeks, the cattle are fed a series of rations per day can be harvested through these meat packing

designed to acclimate the rumen to starch digestion.

plants. Cattle are humanely harvested and the carcasses

The ‘finishing’ ration will contain approximately 90 per

are hung on an overhead rail to cool. The United States

cent concentrate. Feeding concentrates at this level may

Department of Agriculture provides quality inspectors

result in lactic acidosis, thus proper feed management

and veterinary inspectors to ensure a safe, wholesome

is of the utmost importance.

food supply.

Historically, finished cattle were sold to meat packing

Possibly one of the most positive changes that the

companies simply as a commodity. There were no pre-

meat packing industry has made in recent years is the

miums paid for exceptional quality, so there was no

implementation of information sharing. It is currently

reason for feedyards to feed for quality – just perform-

possible for a cow-calf producer to receive information

ance. Beginning in the 1970s, consumption of beef

about how well his cattle performed, in terms of quality

declined steadily, year after year. It became apparent

grade and yield grade. This type of feedback is very important for genetic advancement in the cattle herd. Meat packing companies and feed-yards started to enter into arrangements that would pay premiums for superior beef quality and yield and would

The practice of beef cattle veterinary

penalize for inferior beef quality and yield. These

medicine in North America

pricing arrangements are commonly known as 'grids' or 'formula pricing'. Most grids are developed to reward Beef cattle veterinary practice has evolved a great deal for both beef quality and yield, but there are some that in the last 20 years. This evolution has developed from primarily reward for yield or primarily reward for a primarily ambulatory practice in which individual quality. Feedyards enter into grid agreements that best animals were treated to a much more production-oriented approach of veterinary medicine. As Ameri- there are still a large number of cattle sold on a cash

can food production has become more and more efficient as a commodity.

cient, American society has become less and less aware

This discussion would be incomplete if 'stockers' and of how their food is produced. This has led to many mis- 'backgrounders' were not included. Stockers are cattle- conceptions among the American people about how men who purchase calves at approximately weaning age food animals are handled and treated for disease.

and turn these calves out to pasture. These calves gain

Society is very concerned about animal welfare, anti-

anywhere from 200 to 400 lb (90–180 kg) while on

biotic resistance and food safety. Beef producers are

pasture. The calves are then sold to a cattle feeder or

very concerned about these things as well, but are also

the stocker retains ownership of the cattle through the

concerned about production performance,

cost-

feeding period. Backgrounders (or preconditioners), on

effectiveness and the ever-tightening profit margin.

the other hand, are cattlemen who specialize in feeding

Individual treatment of animals is still a substantial and taking care of high risk calves (calves that have a part of beef cattle practice. Veterinarians are still called high risk of sickness). Many feedyards will send their upon to treat respiratory disease, lameness, reproductive disorders, digestive and nutritional problems, the feedyard does not have enough employees to give among other things. However, veterinarians are also these calves the level of attention they need. Often, taking a more active role in preventing these situations. calves in a backgrounding operation are checked two Beef cattle producers are being economically forced to or more times per day for sickness. After a few weeks, provide cattle with added value. This means that the the health problems subside. The calves will adjust to a cattle must have the genetics and the immune integrity concentrate-based ration and learn to eat out of a feed-to perform well and stay healthy through each stage of bunk. After several more weeks, the calves are removed production.

from the backgrounding operation and sent to their
Many new opportunities in veterinary medicine have
respective feedyard, where they are fed out to harvest.
arisen because of this change from individual treatment
to preventive medicine. In the cow-calf sector, many
new computer software programs have been developed

Meat packing sector

that can help with production parameters, record
keeping and genetic selection. Because of the labour
Once the cattle have reached the appropriate harvest
intensity involved in beef production, many producers
weight and condition, they are sent to the meat packing
simply do not have the time to get comfortable with the
plant. While there are several meat-packing companies
software and its capabilities, although they still want the

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information that the software provides. Many veteri-
veterinarian. Veterinarians play a key role in control
narians have taken this opportunity to help their clients
and/or elimination of pathogens in the food chain. Edu-
by serving as a data analyst. Veterinarians gather the

cating the beef producer and the consumer in areas information needed and record the information in such as how these pathogens develop and how they can the software program. Because the veterinarian has the be controlled is important for human health and for the opportunity to work with several herds, he/she also has survivability of the industry. Another food safety the opportunity to understand the software and its concern is the use of growth-promoting implants. The capabilities at a deeper level. The veterinarian can then European Union has stopped the importation of beef analyse a client's data, work up reports in various areas from the United States that has been exposed to growth of production and consult with the client on topics that promoting hormones. The use of growth promoting may include replacement heifer selection, herd sire hormones has been proven to be a safe practice. selection, culling, preventive health programmes, nutri- However, the EU's ban has caused some Americans to tion, effective treatment schedules and the cost- question its safety (see Chapter 63). Veterinarians are

effectiveness of various production practices. There is playing a key role in quelling rumours and misinformation about the safety of American beef and the use of growth-promoting hormones by applying good, sound science.

of diseased stock, but veterinary consultation has the potential of a wide scope of development in the away from individual animal medicine and move calf sector.

toward population medicine. Veterinarians must be In the cattle-feeding sector, veterinary consultation cost-effective and must be able to demonstrate their already has a tremendous foothold. With the very large cost-effectiveness. Applying good science to decision-making is a major key to being cost-effective. Skills in effective for the feedyard to hire a consulting veteri-

critical evaluation of the scientific literature need to be
narian to design health programmes, evaluate treat-
honed to a high level of precision so science-based deci-
ment protocols, help in the training of new employees
sions can be made. This is very important in an increas-
and advise on the construction of new facilities and the
ingly litigious society, as good science is much easier to
repair of current facilities. This type of practice can be
defend in court than anecdotal decisions.

very satisfying professionally, but there is a great deal of
There are and will be many, many opportunities
travel involved.

available in beef cattle veterinary practice in the
Whether a veterinarian specializes in the cow-calf
future. The beef industry is changing at a rapid pace and
sector, the cattle feeding sector or both, the issues that
the same is true of beef cattle practice. Veterinarians
the beef industry faces will affect his/her practice. One
must be willing and able to adapt to a changing indus-
of those issues is food safety. Food safety has captured
try and be able to recognize an opportunity when it

*the attention of the consumer, the producer and the
arises.*

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